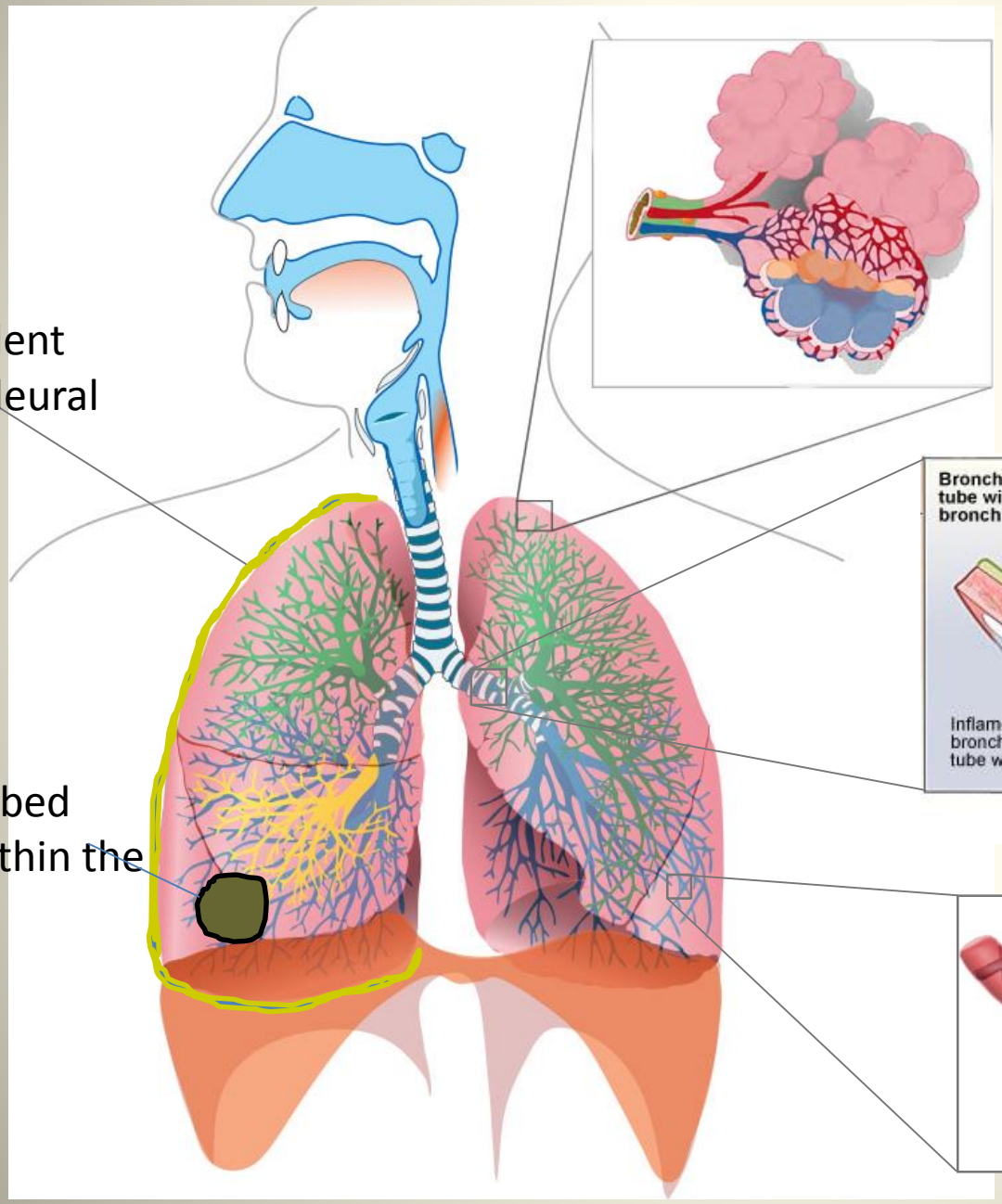

بسم الله الرحمن الرحيم

Paediatric Community Acquired Pneumonia and Complications

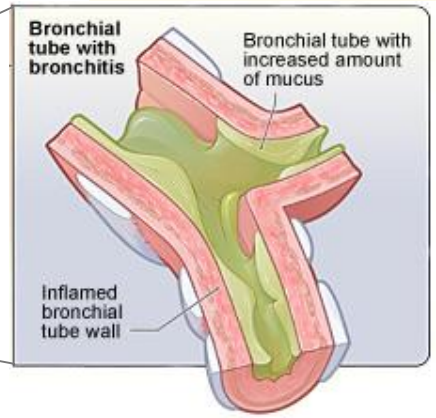
Neat Bilan Peddiatric Pulmonologist

Lower respiratory and pleural disease



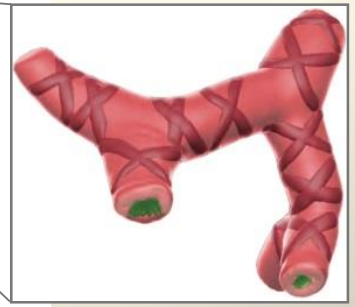
Pneumonia -- infection of alveoli (viral or bacterial)
vs. ***Pneumonitis*** *-- immune-mediated inflammation of alveoli*

Empyema: purulent exudate in the pleural cavity



Bronchitis -- inflammation of bronchi, may be **immune-mediated**, e.g. asthma, COPD, or **infectious** (usually viral but can be bacterial)

Abscess: circumscribed collection of pus within the lung parenchyma



Bronchiolitis: inflammation of bronchioles (often viral but can be bacterial)

PNEUMONIA

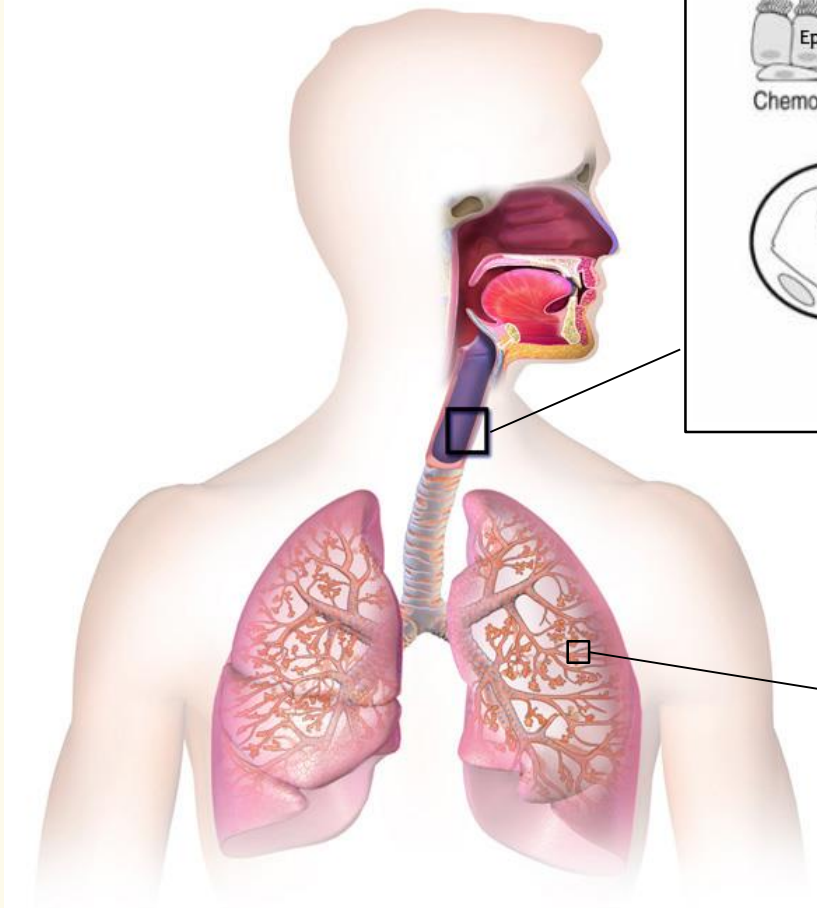
"Inflammation of the lung"

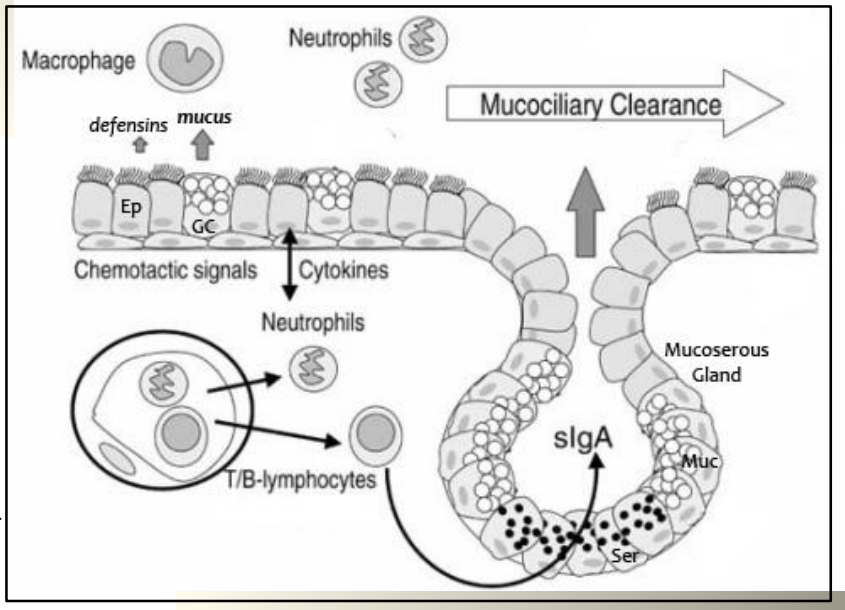
-From Greek pneumōn meaning 'lung'

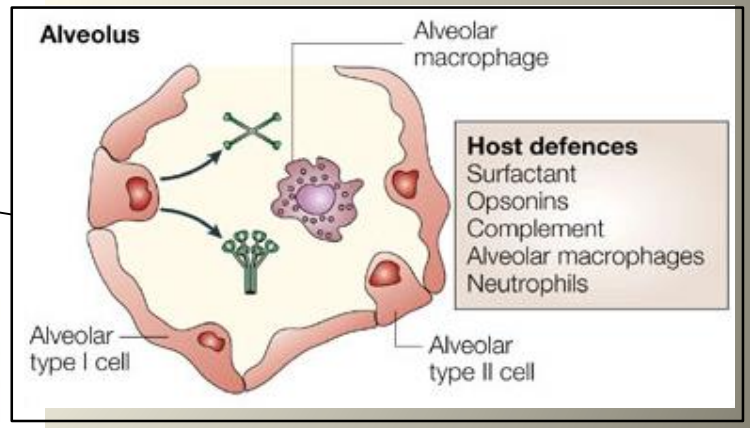
PNEUMONIA:
CLEARANCE vs. COLONIZATION

Microbes constantly enter airways but many factors prevent colonization:

- mucous entrapment
- ciliary clearance
- immune surveillance
- intact epithelial barrier
- secreted factors such as:
  - secretory IgA
  - surfactant proteins (SP-a, SP-d)
  - defensins







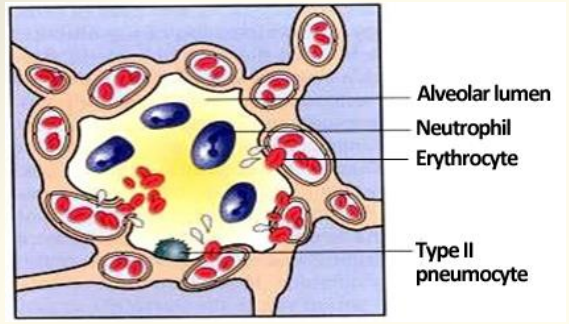
Disrupting or overwhelming these defense mechanisms can allow microbes to colonize the lungs, resulting in PNEUMONIA

Effects and patterns of microbial colonization:

where and how inflammation appears can be informative

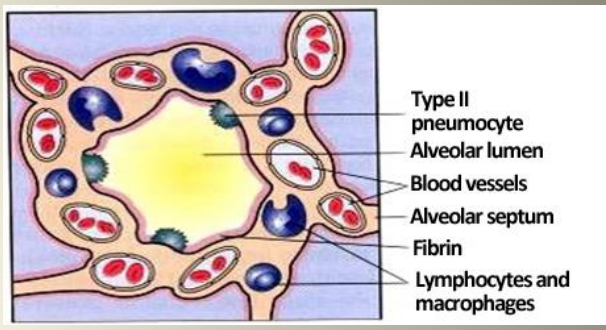
Alveolar

- In alveolar **lumen**
- Purulent exudate of RBCs and PMNs



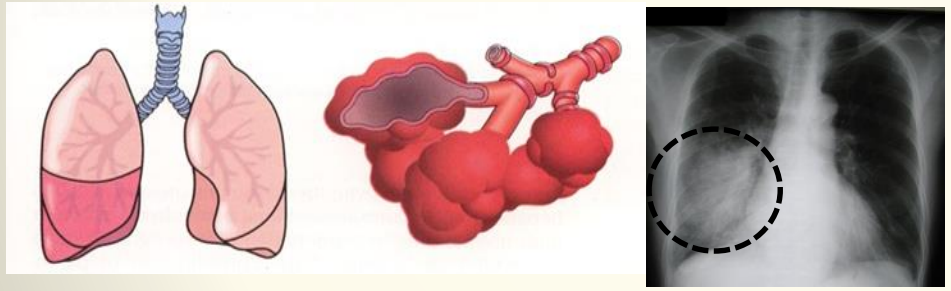
Interstitial

- Mostly in alveolar **wall**
- Mononuclear WBCs
- Fibrinous exudate



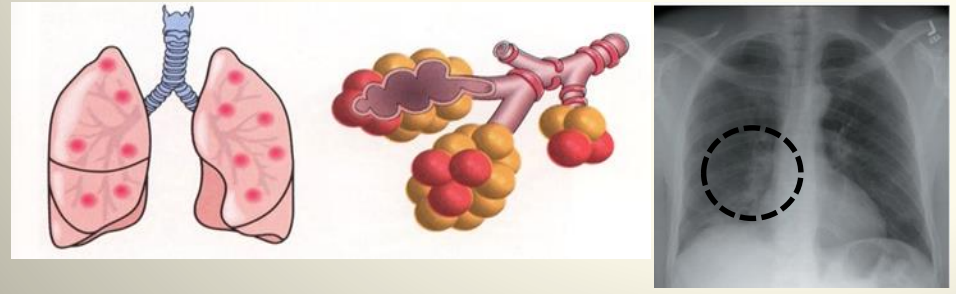
Lobar pneumonia

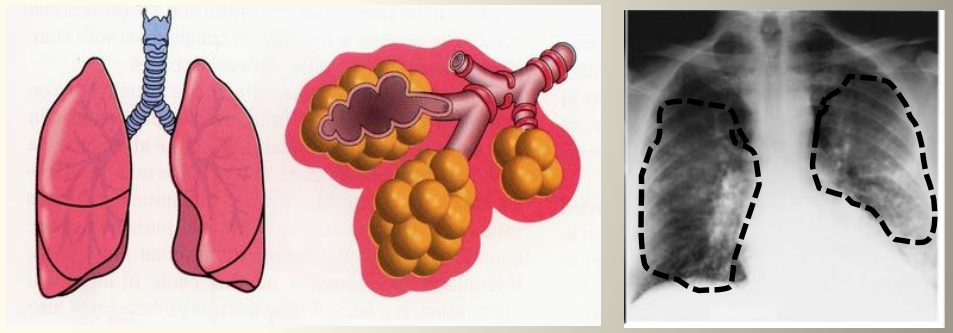
- lobar distribution
- "typical" CAP
- *S. pneumo, H. flu.*



Bronchopneumonia

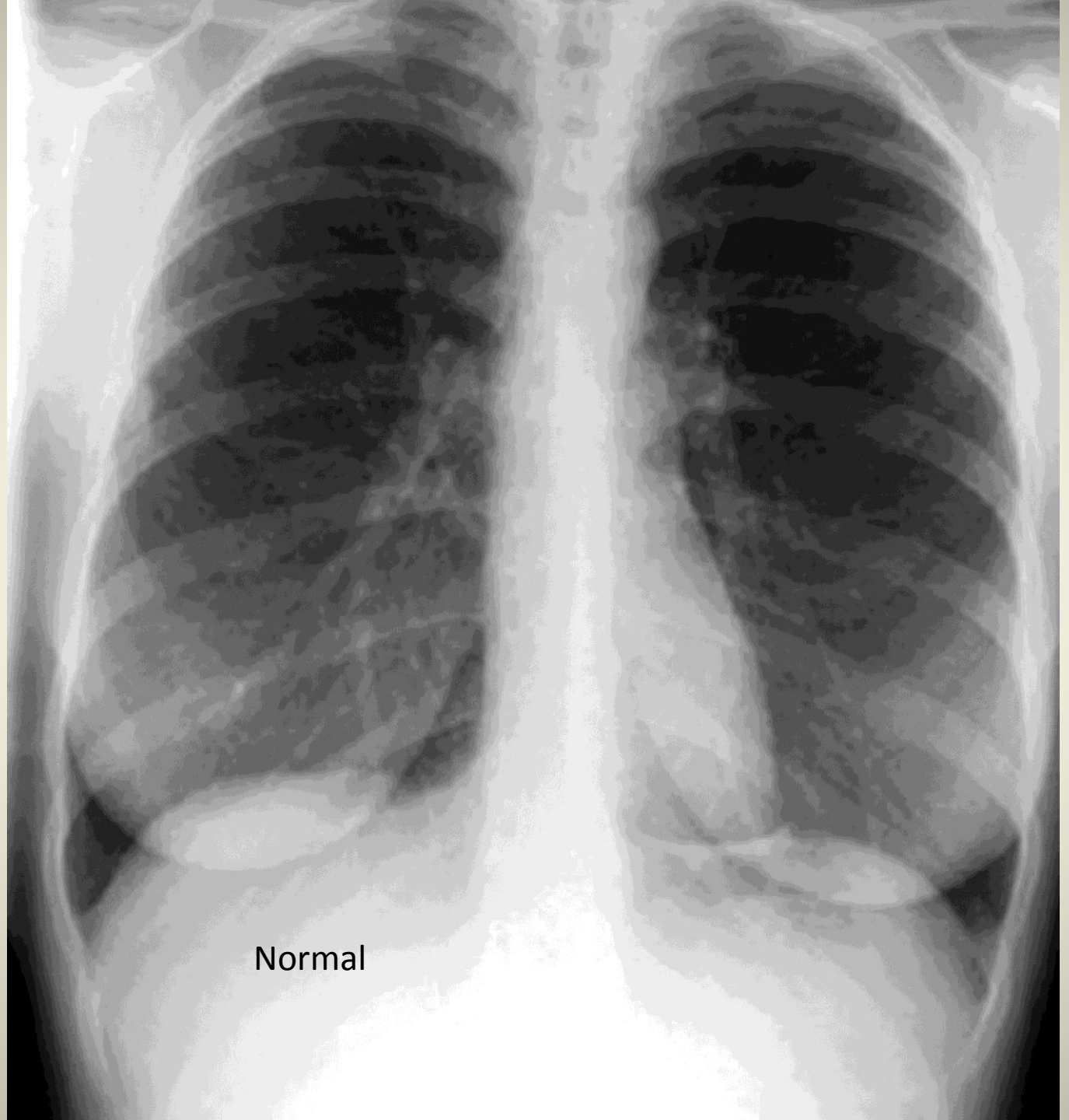
- patchy distribution
- aspiration, intubation, bronchiectasis
- *Staph, enterics, Pseudomonas*





Atypical pneumonia

- diffuse infiltrate w/ perihilar concentration
- *Mycoplasma, Chlamydophila, Legionella*
- Respiratory viruses, e.g. influenza



Normal

Typical CAP presentation

History

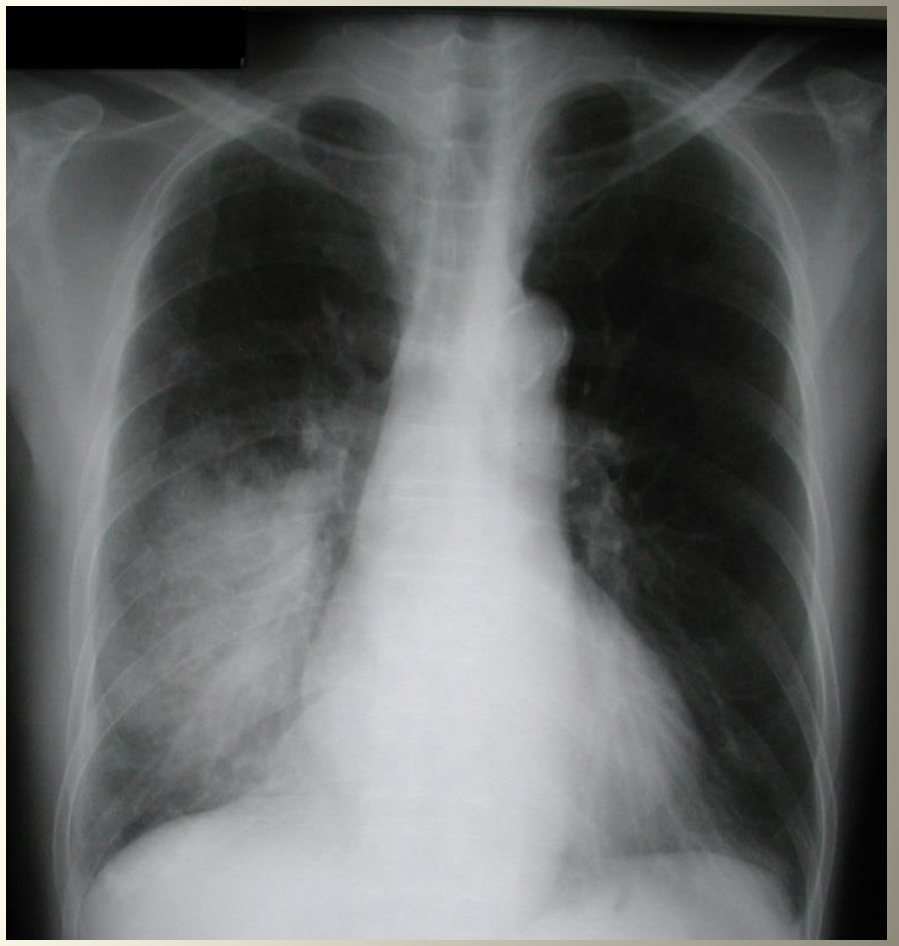
- Previously healthy with sudden onset of fever and shortness of breath

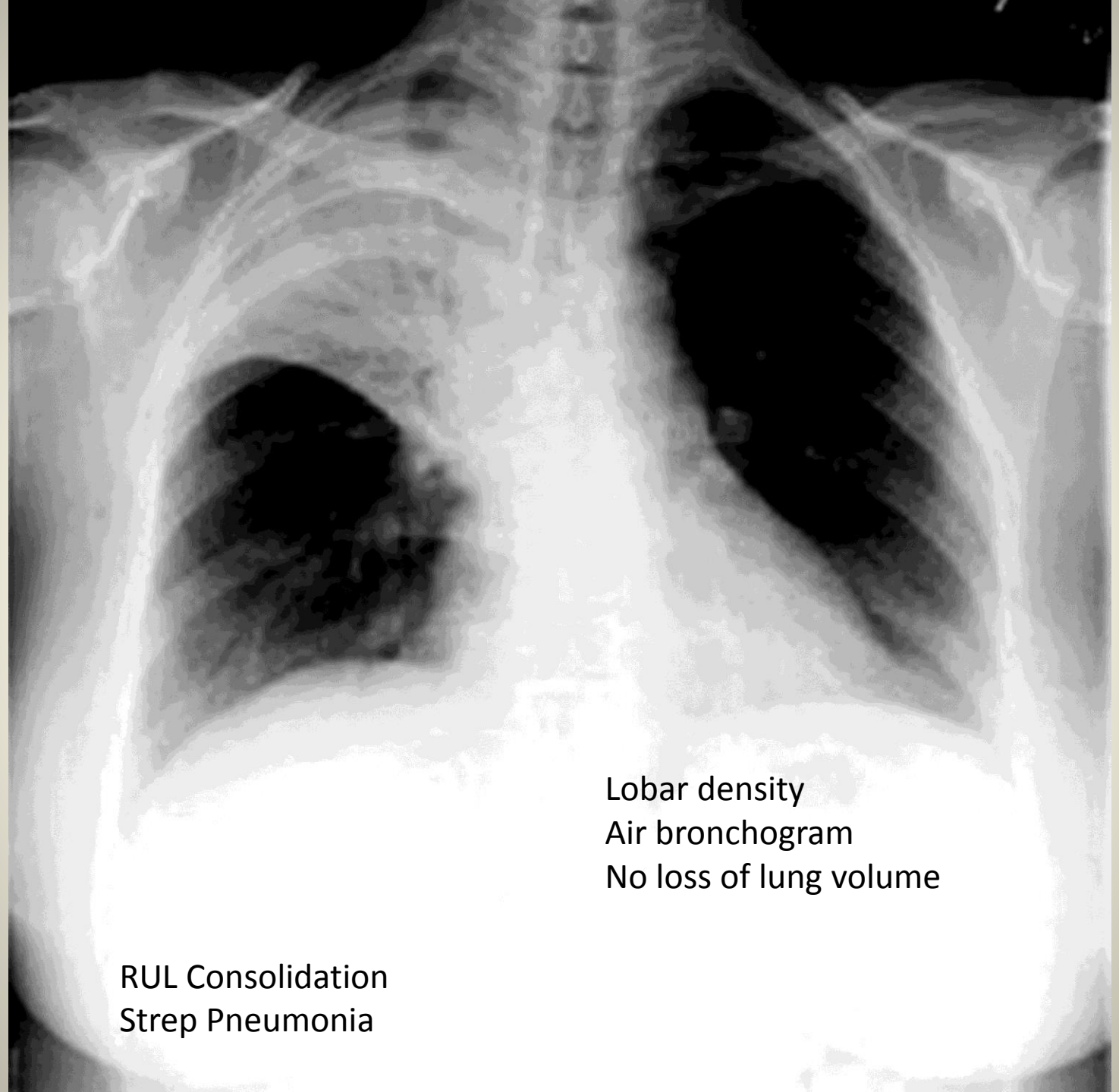
Physical signs and symptoms

- fever
- tachycardia
- tachypnea
- productive cough with purulent sputum and possible hemoptysis
- pallor and cyanosis
- localized:
  - dullness to percussion
  - decreased breath sounds
  - crackles, ronchi, egophony (“E-to-A” change)

Investigations

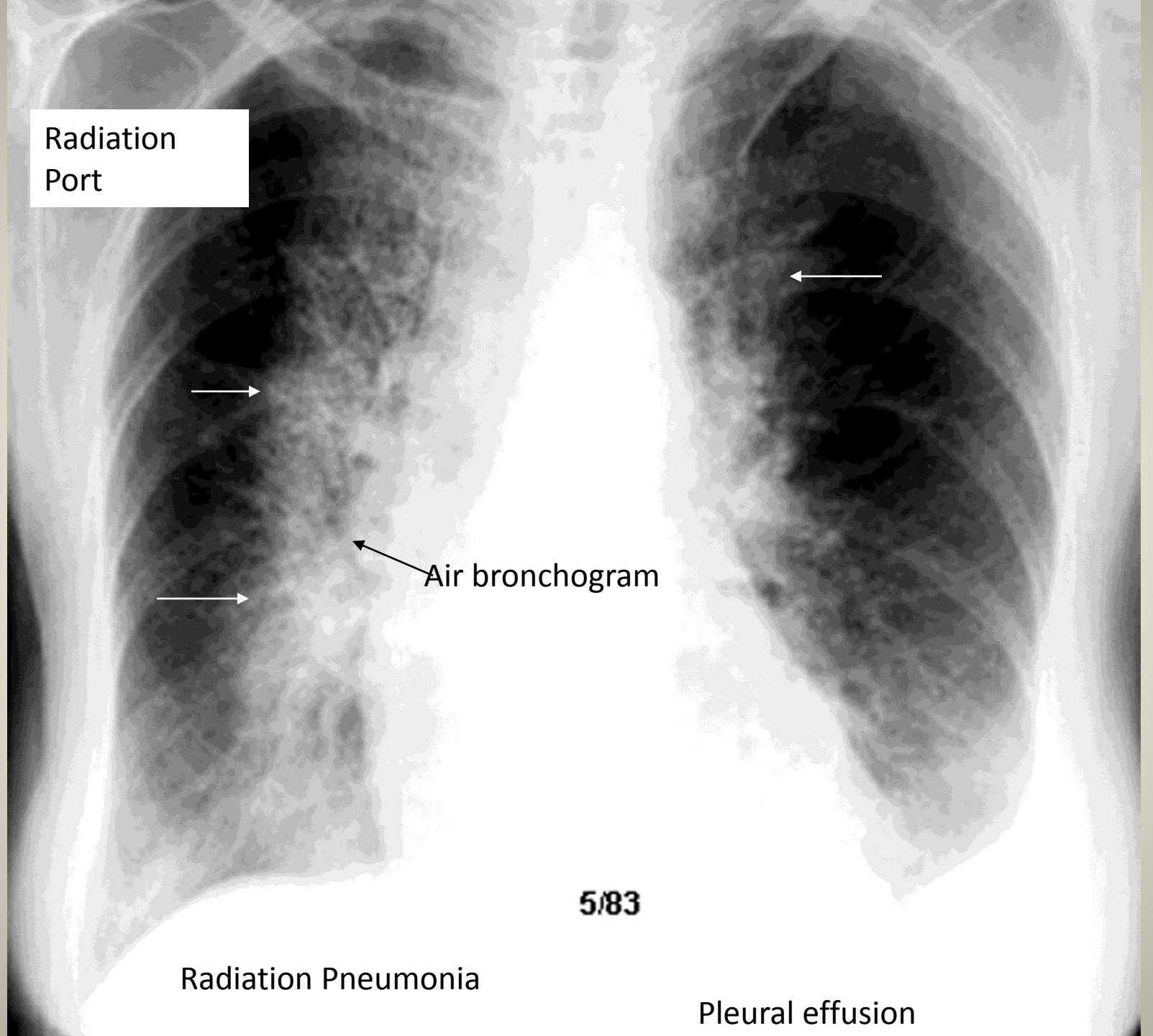
- CXR showing lobar consolidation
- CBC showing leukocytosis w/ left shift
- Sputum sample contains neutrophils, RBCs; Gram stain may be positive depending on organism





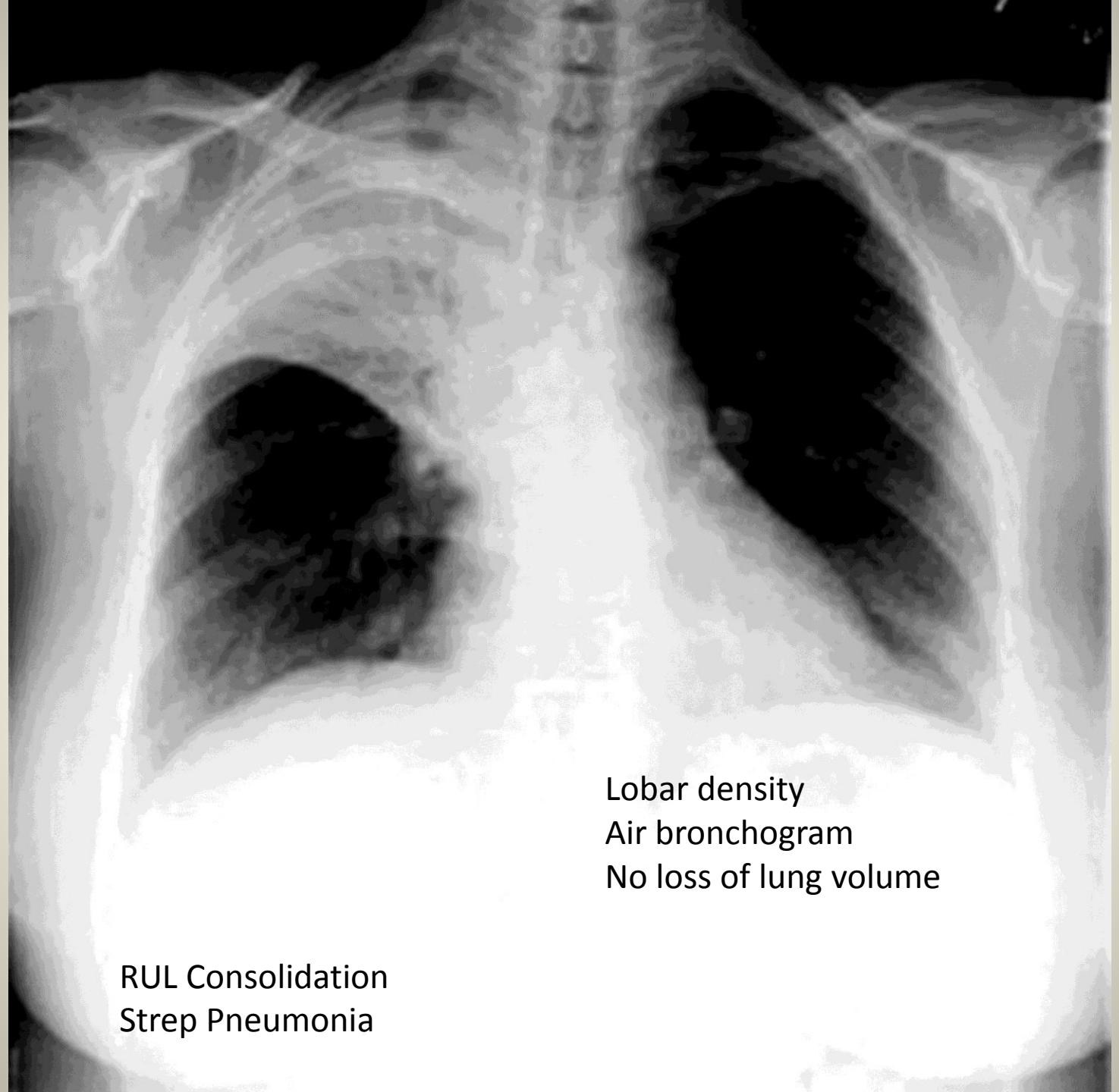
Lobar density
Air bronchogram
No loss of lung volume

RUL Consolidation
Strep Pneumonia



Radiation Pneumonia

Pleural effusion



Lobar density
Air bronchogram
No loss of lung volume

RUL Consolidation
Strep Pneumonia

Community Acquired Pneumonia

- Incidence higher in boys[1]
- Incidence of severe pneumonia higher in children < 5 years of age[1]
- Marked seasonal pattern with winter preponderance for hospital admission due to pneumococcal infection (December and January 3-5x higher than August) [2]
- Winter preponderance also noted for many viral infections including respiratory syncytial virus, influenza and parainfluenza 1 & 2 [3]

[1]JAMA Pediatr. 2017;171:573 [2]Epidemiol Infect 2007;135:262 [3]Journal of Infection 2006;52:37

Aetiology

- Viruses most prevalent cause of CAP in childhood particularly during infancy
- RSV, influenza and human metapneumovirus most common viruses[1]
- *Strep. Pneumoniae* most common bacterial cause of CAP[1]
    - Incidence and severity of disease significantly reduced since introduction of pneumococcal conjugate vaccine[2]

[1] Thorax 2019;74:261 [2]Clin Infect Dis 2014;58:918

Clinical Features

- Tachypnoea
  - relatively specific but not sensitive[1]
  - associated with hypoxaemia in infants (>70 bpm)[2]
- Increased work of breathing
  - signs include grunting, nasal flaring and chest retractions or indrawing
  - highly specific for pneumonia[1]
- Chest examination
  - Crackles on auscultation
  - Wheeze more common with CAP due to atypical bacteria and viruses (also consistent with asthma and bronchiolitis)
  - Signs of consolidated lung including vocal resonance,vocal fremitus, reduced breath sounds, dullness to percussion

[1]JAMA 2017;318:426 [2]Ann Trop Paediatr 1998;18:31

Severity Assessment

Features of Severe Pneumonia:

- Tachypnoea (>70 bpm under 12 months age, >50bpm over 12 months)
- Moderate/severe recession (<12 months)
- Severe difficulty breathing (>12 months)
- Grunting
- Nasal Flaring
- Apnoea (<12 months)
- Cyanosis
- Tachycardia (>170 bpm under 6 months, >160 bpm 6-12 months, >150 bpm 1-3 years, >140 3-5 years, >120 5-12 year, >100 over 12)
- Capillary Refill Time ≥ 2 secs
- Hypoxaemia (sustained oxygen saturation <92% in room air)
- Not feeding (< 12 months)
- Signs of dehydration (>12 months)

Adapted from BTS CAP in children guideline 2011

General Management - Community

- Advise parents and carers about:
  - management of fever
  - preventing dehydration
  - identifying signs of deterioration
  - identifying signs of other serious illness
  - how to access further healthcare (providing a ‘safety net’).
  - the ‘safety net’ should be one or more of the following:
    - provide the parent or carer with verbal and/or written information on warning symptoms and how further healthcare can be accessed
    - arrange a follow-up appointment at a certain time and place
    - liaise with other healthcare professionals, including out-of-hours providers, to ensure the parent/carer has direct access to a further assessment for their child

Complications and Failure to Improve

- Pleural effusion and empyema
  - consider with fever beyond 7 days or not settling after 48 hours antibiotics[1]
  - CXR reveals fluid in pleural space; amount of fluid best estimated with ultrasound
  - if patient persistently febrile the pleural space should be drained
  - further details: BTS guidelines for the management of pleural infection in children[2]
- Necrotising pneumonia
  - characterised by necrosis and liquefaction of lung tissue
  - Usually 2º to pneumococcus (particularly serotypes 3&19), *Staph. aureus* and group A

[1]Clin Infect Dis 2002;34:434 [2]Thorax 2005;60:i1 [3]Eur Resp J 2008;31:1285

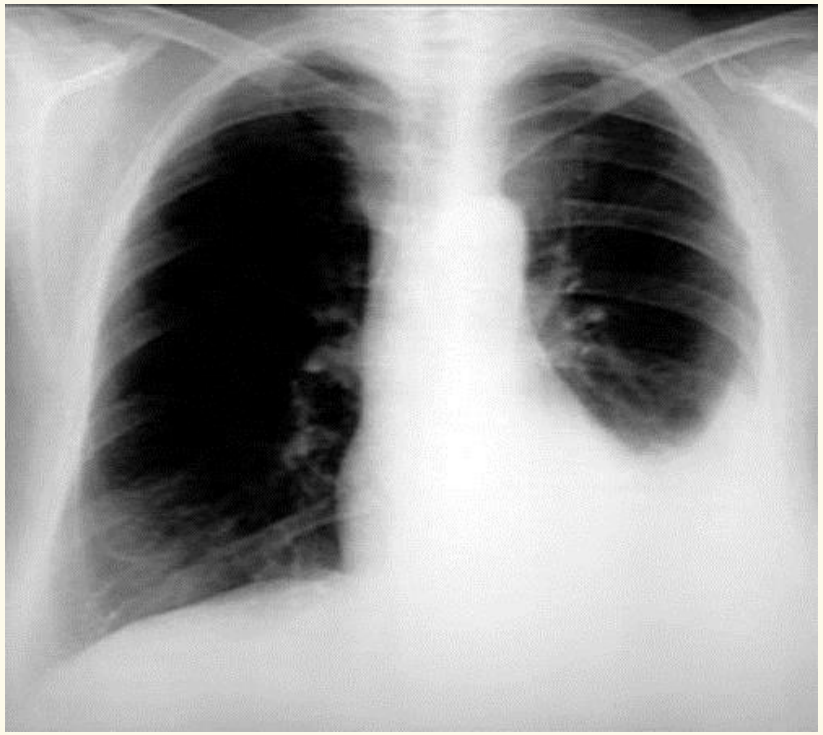
Complications and Failure to Improve

- Lung abscess
  - thick walled cavity within the lung tissue that contains purulent liquid
  - may be secondary to aspiration (especially in children with neurodevelopmental delay), congenital malformations and immunodeficiency
  - may result from inadequate or delayed treatment of lobar pneumonia
  - similar presentation to CAP initially but progresses indolently
  - mainstay of therapy is a prolonged course of parenteral antibiotics

Complications and Failure to Improve

- If a child remains feverish or unwell 48 h after hospital admission with pneumonia, re-evaluation is necessary with consideration given to possible complications

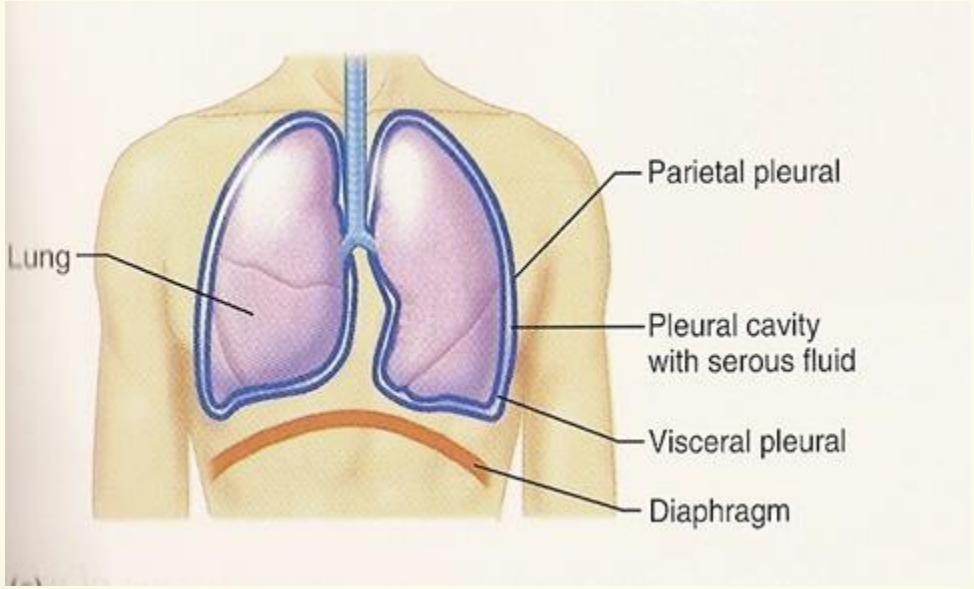
Pleural Effusion



Pleural Effusion

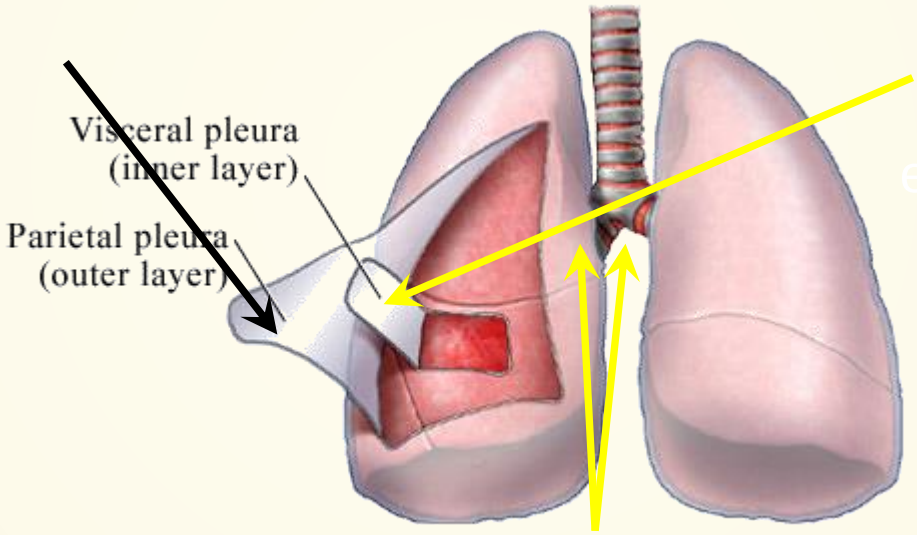
- **Pleural effusion** is an abnormal accumulation of fluid in the pleural space. The 5 major types of pleural effusion are:
  - Transudate,
  - Exudate,
  - Empyema,
  - Hemorrhagic pleural effusion or hemothorax and
  - Chylous or chyliform effusion.

PLEURA


Lung
Parietal pleural
Pleural cavity
with serous fluid
Visceral pleural
Diaphragm

Parietal pleura

, and ribs.



Visceral pleura

envelope all surfaces

of.

Hilum

where pulmonary vessels, bronchi, and nerves enter the lung tissue, the parietal pleura is continuous with the visceral pleura.

PLEURAL EFFUSION

Normally the pleural space contains:

- 3.5 to 7.0 ml of clear liquid
- low protein content
- small number of mononuclear cells

Pleural effusion: presence of large amount of fluid in the pleural space irrespective of the underlying causes

- The normal pleura is a thin translucent membrane consisting of 1-mesothelium, 2-thin layer of subendothelial connective tissue rich in lymphatics, arteries veins and nerves.

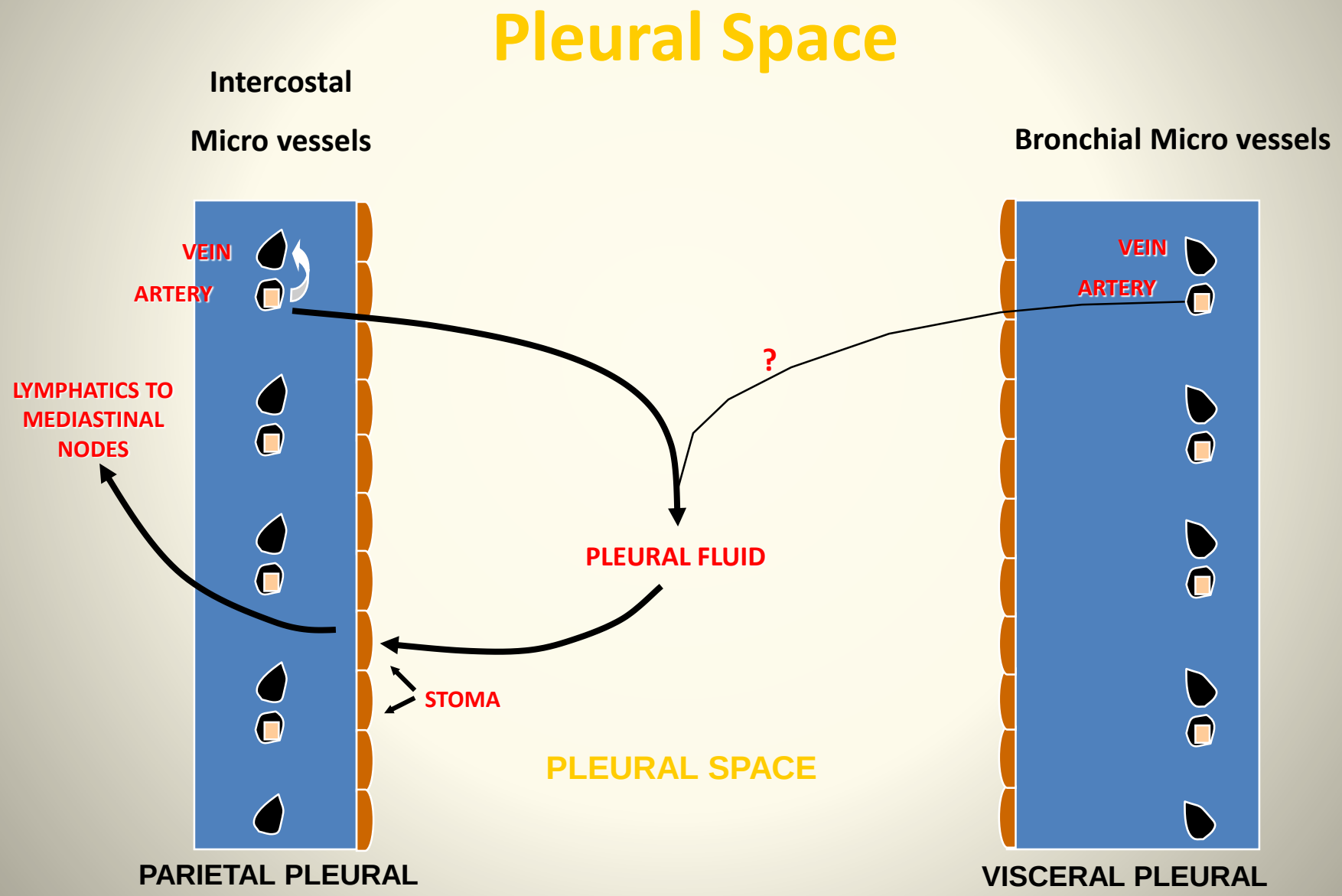
- Functions:
  - Space for movement &
  - protective in volume overload

PLEURAL FLUID FORMATION AND ABSORTION

- The rate of fluid formation is 0.02 ml/kg/hour.
- The rate of fluid clearance is 0.2 ml/kg/hour.

- روزانه 5 تا 10 لیتر مایع به حفره پلور انفیلتره می گردد و از طریق عروق کوچک پلوروپاریتال جذب می شود.
- طبق قانون استارلینگ تبادل مایع بر اساس اختلاف فشار هیدرواستاتیک وانکوتیک می باشد.
- در حالت عادی تنها 15 تا 20 سی سی مایع در حفره پلور باقی می ماند.

Pleural Fluid Formation and Absorption



Development of Pleural Effusion

- ↑ pulmonary capillary pressure (CHF)
- ↑ capillary permeability (Pneumonia)
- ↓ intrapleural pressure (atelectasis)
- ↓ plasma oncotic pressure (hypoalbuminemia)
- ↑ pleural membrane permeability (malignancy)

TABLE 1. LEADING CAUSES OF PLEURAL EFFUSION IN THE UNITED STATES, ACCORDING TO ANALYSIS OF PATIENTS SUBJECTED TO THORACENTESIS.*

CAUSE	ANNUAL INCIDENCE	TRANSUDATE	EXUDATE
Congestive heart failure	500,000	Yes	No
Pneumonia	300,000	No	Yes
Cancer	200,000	No	Yes
Pulmonary embolus	150,000	Sometimes	Sometimes
Viral disease	100,000	No	Yes
Coronary-artery bypass surgery	60,000	No	Yes
Cirrhosis with ascites	50,000	Yes	No

*Adapted from Light.[1]

Other causes of pleural effusion: nephrotic syndrome, TB, collagen vascular disease, urinothorax, SVC syndrome, Meigs syndrome, rheumatoid arthritis, pancreatitis, yellow-nail syndrome, drugs

Evaluation

- History:

dyspnea

pleuritic chest pain

cough

fever

hemoptysis

wt. loss

trauma

hx cancer

cardiac surgery

- Physical:

Dullness to percussion

Decreased breath sounds

Absent tactile fremitus

Causes of Pleural Effusion

Leading Causes of Pleural Effusion in the United States*

Cause	*Annual incidence*	*Transudate*	*Exudate*
Congestive heart failure	500,000	Yes	No
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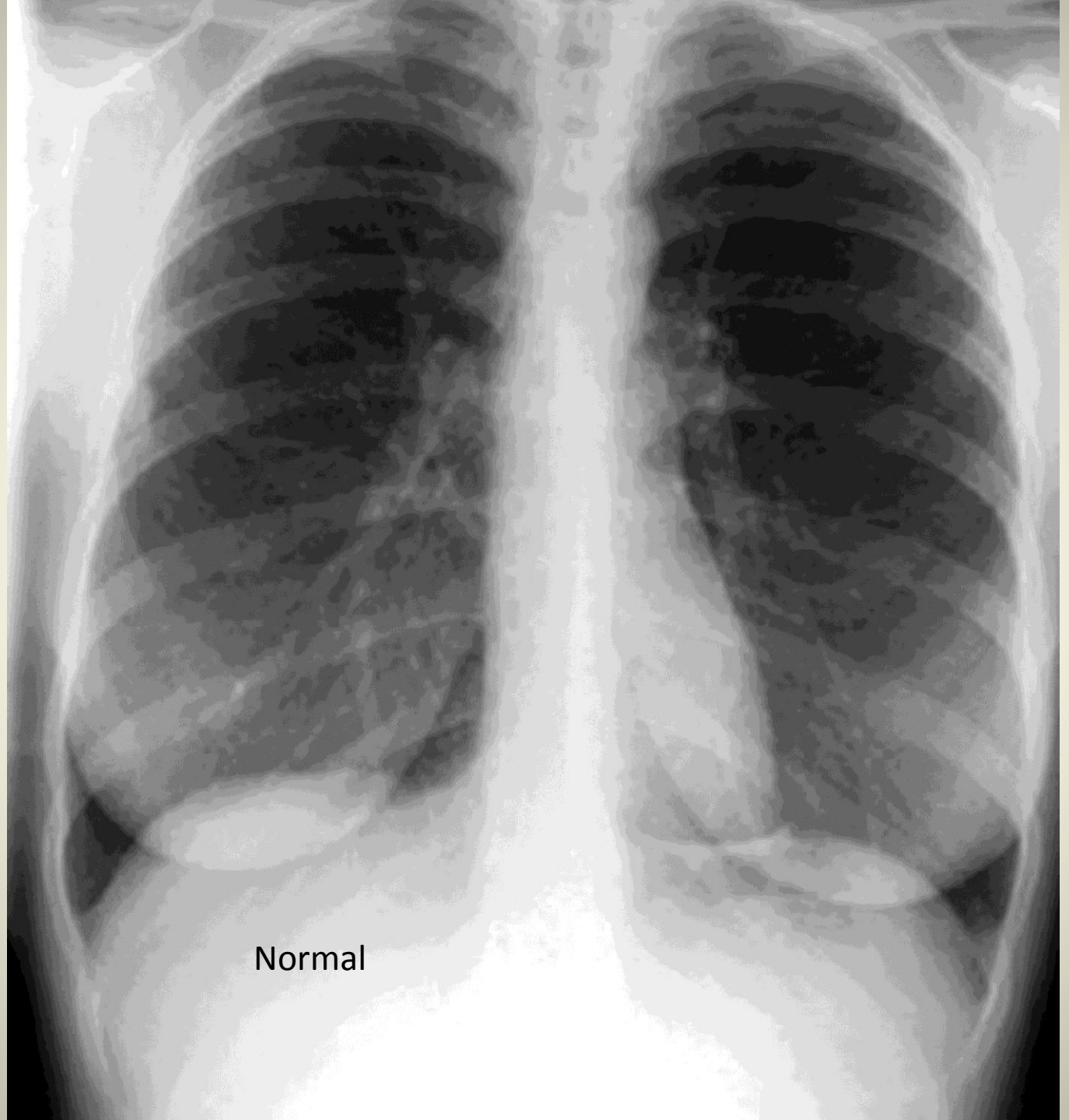
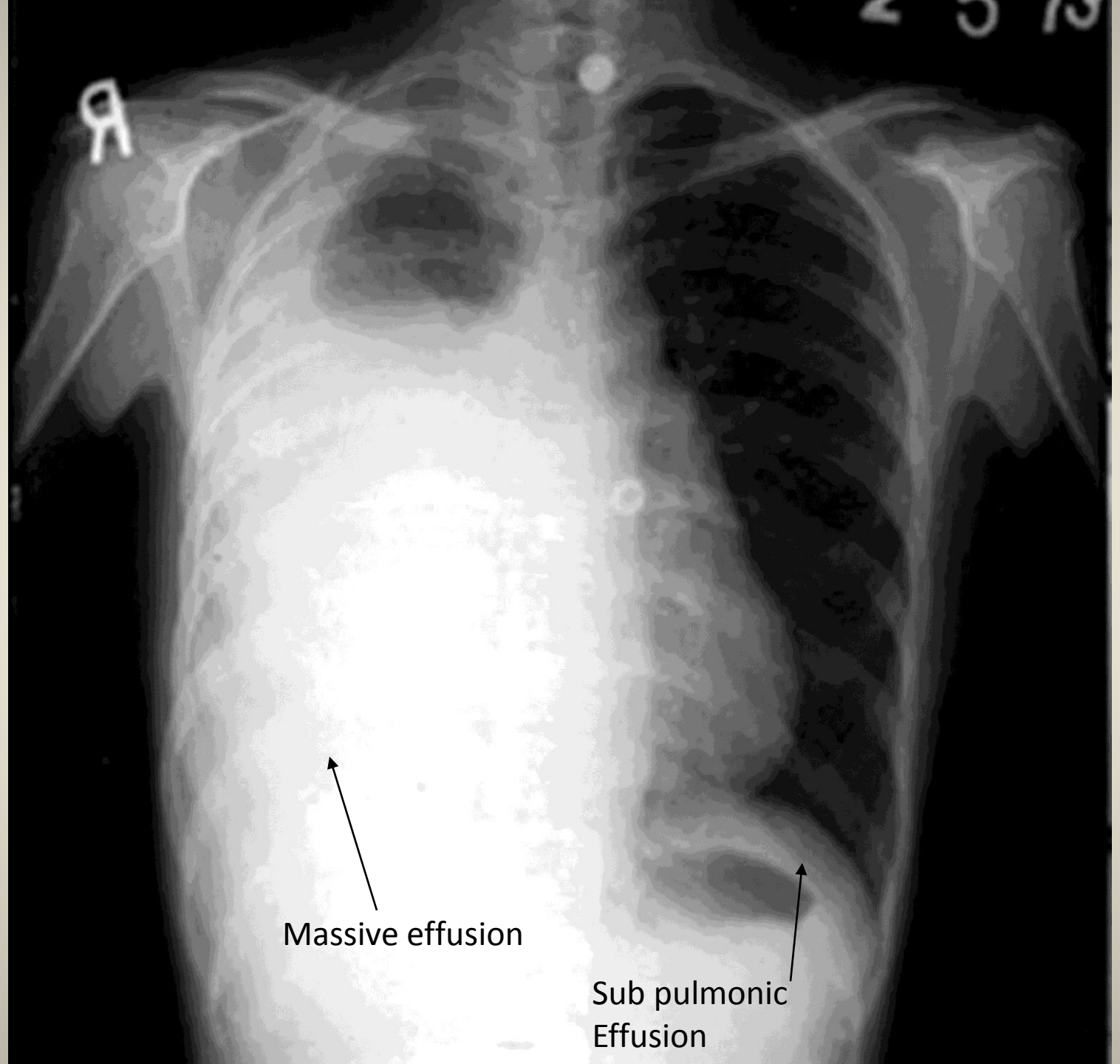
**–Based on analysis of patients subjected to thoracentesis.*

Other causes of pleural effusion: nephrotic syndrome, TB, collagen vascular disease, urinothorax, SVC syndrome, Meigs syndrome, rheumatoid arthritis, pancreatitis, yellow-nail syndrome, drugs

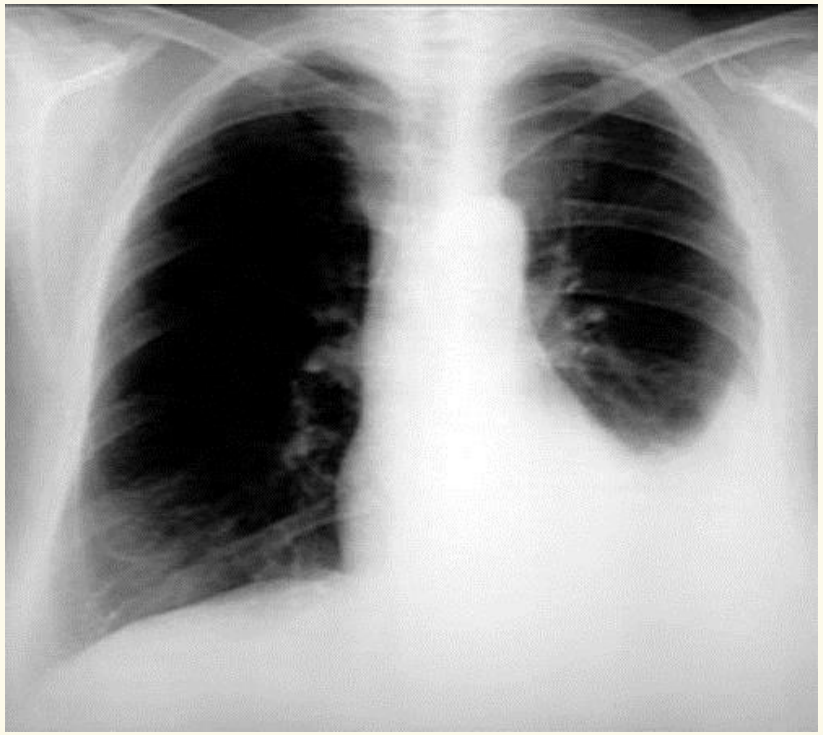
Light. NEJM 2002; 346:1971
Annual incidence in the US



Normal

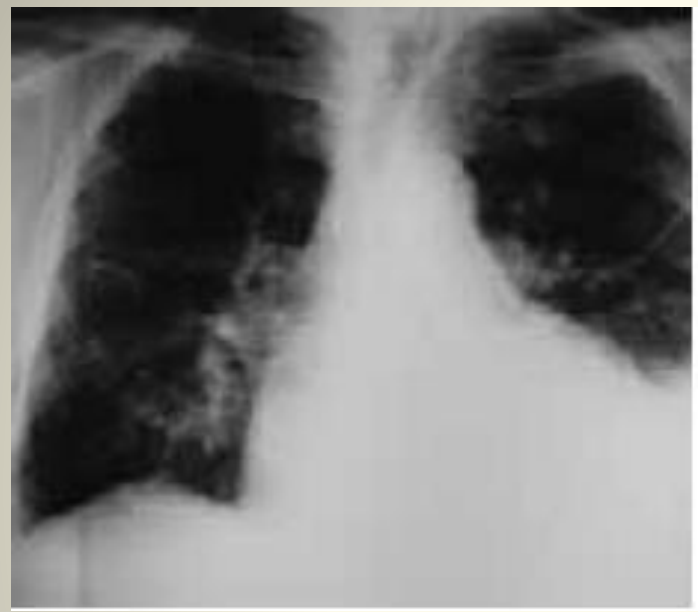

Massive effusion
Sub pulmonic
Effusion

Chest Xray

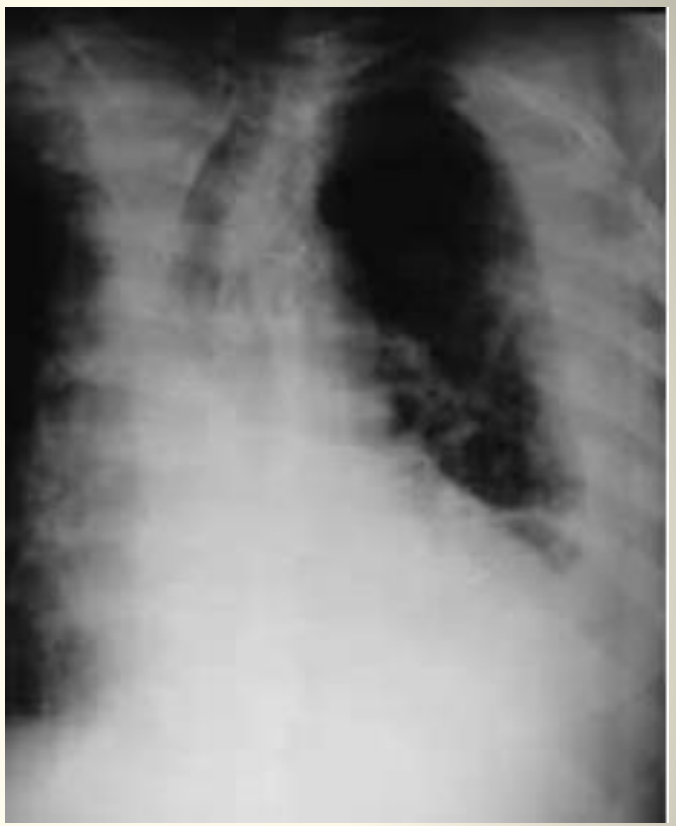


Chest X-Ray

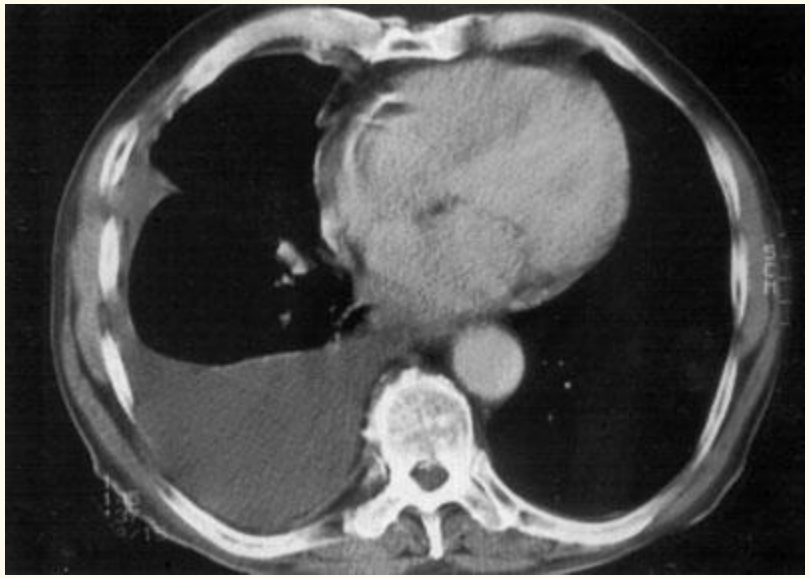
PA



Lateral decubitus



Pleural effusion



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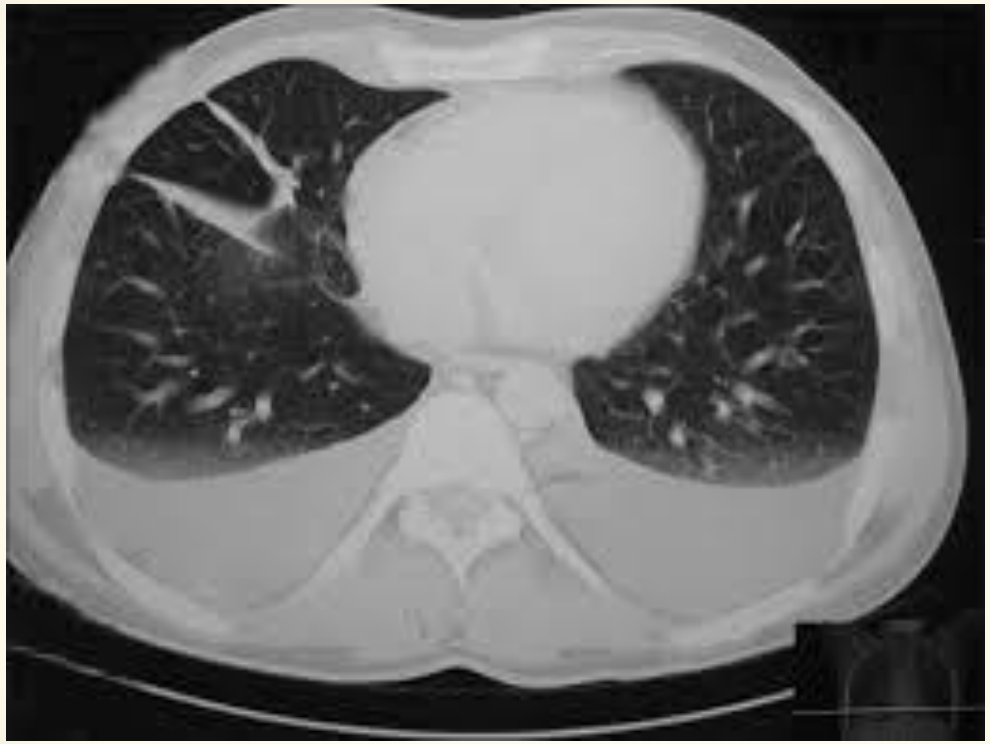
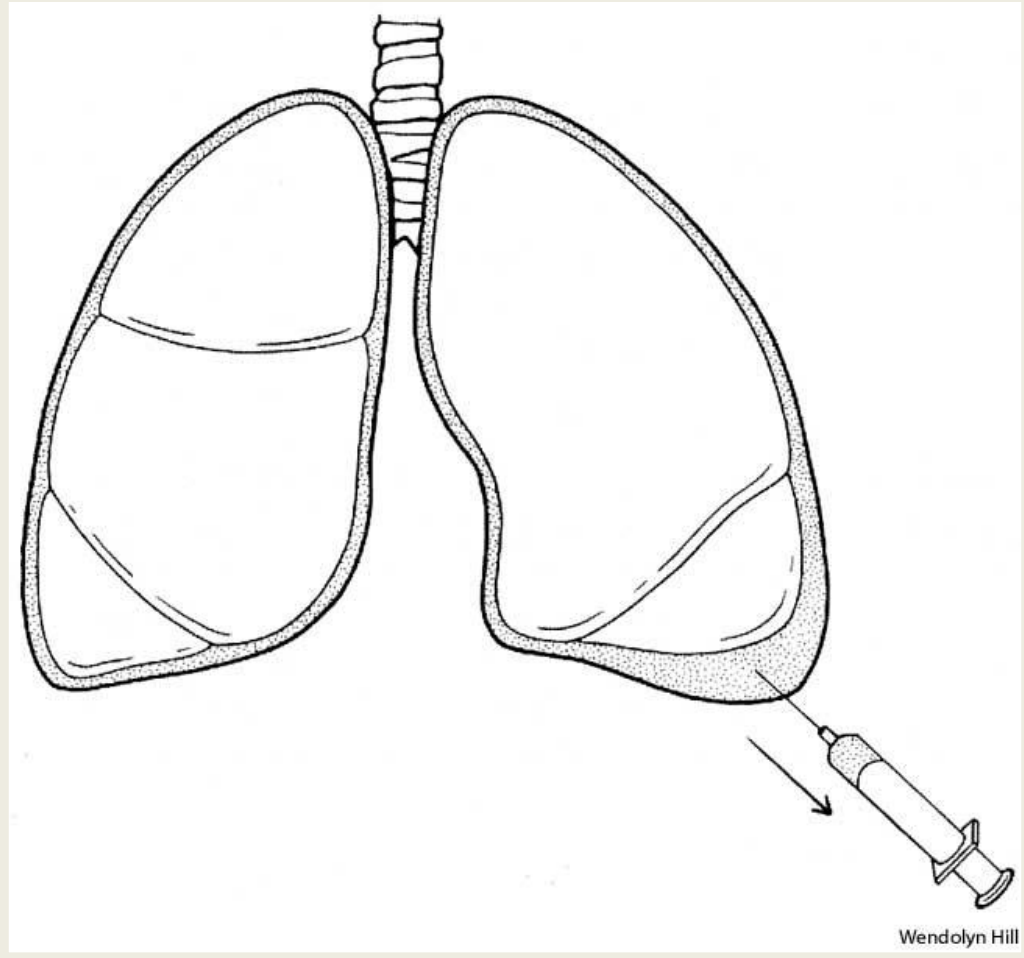


TYPES

- Transudative
- Exudative
- Extrapleural:transmigration

Thoracentesis



LEADING CAUSES OF PLEURAL EFFUSIONS

Causes	Annual Incidence	Transudate	Exudate
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Viral disease	100,000	No	Yes
Coronary artery bypass surgery	60,000	No	Yes
Cirrhosis with ascites	50,000	Yes	No

Light RW. Pleural diseases. 4th ed. Lippincott Williams &Wilkins, 2001

Etiology

- **Transudative**
- **CHF**
- **CLD**
- **Renal Failure**
- **Anaemia/Hypoproteinemia**
- Myxedema
- Gross ascites

- **Exudative**
- **Pneumonia**
- **Tb**
- **CA**
- **Pulmonary Embolism:Both**
- **Trauma**
- **Post surgery**
- **Inflammatory:RA/SLE**

Indications for thoracentesis

- Effusions larger than 1cm height of unknown origin
- No need for thoracentesis for patient with obvious cause (CHF with bilateral effusions). However:
  - In heart failure: febrile/pleuritic pain, unilateral, no cardiomegaly, no response to diuresis

Exudate v Transudate

Patient's serum protein is normal

- Pleural protein is less than 25 g/l =Transudate
- Pleural Protein more than35 g/l.= Exudate

- If not, Light's criteria

Light's Criteria

Pleural fluid is exudate if one or more:

- Pleural fluid protein:serum protein > 0.5
- Pleural fluid LDH:serum LDH > 0.6
- Pleural fluid LDH > 2/3 upper limit nl serum LDH

Transudate

- CHF
- Cirrhosis
- Nephrotic syndrome

Exudate

- Pneumonia
- Malignancy
- Pulmonary Embolism

درمان افیوژن پلور

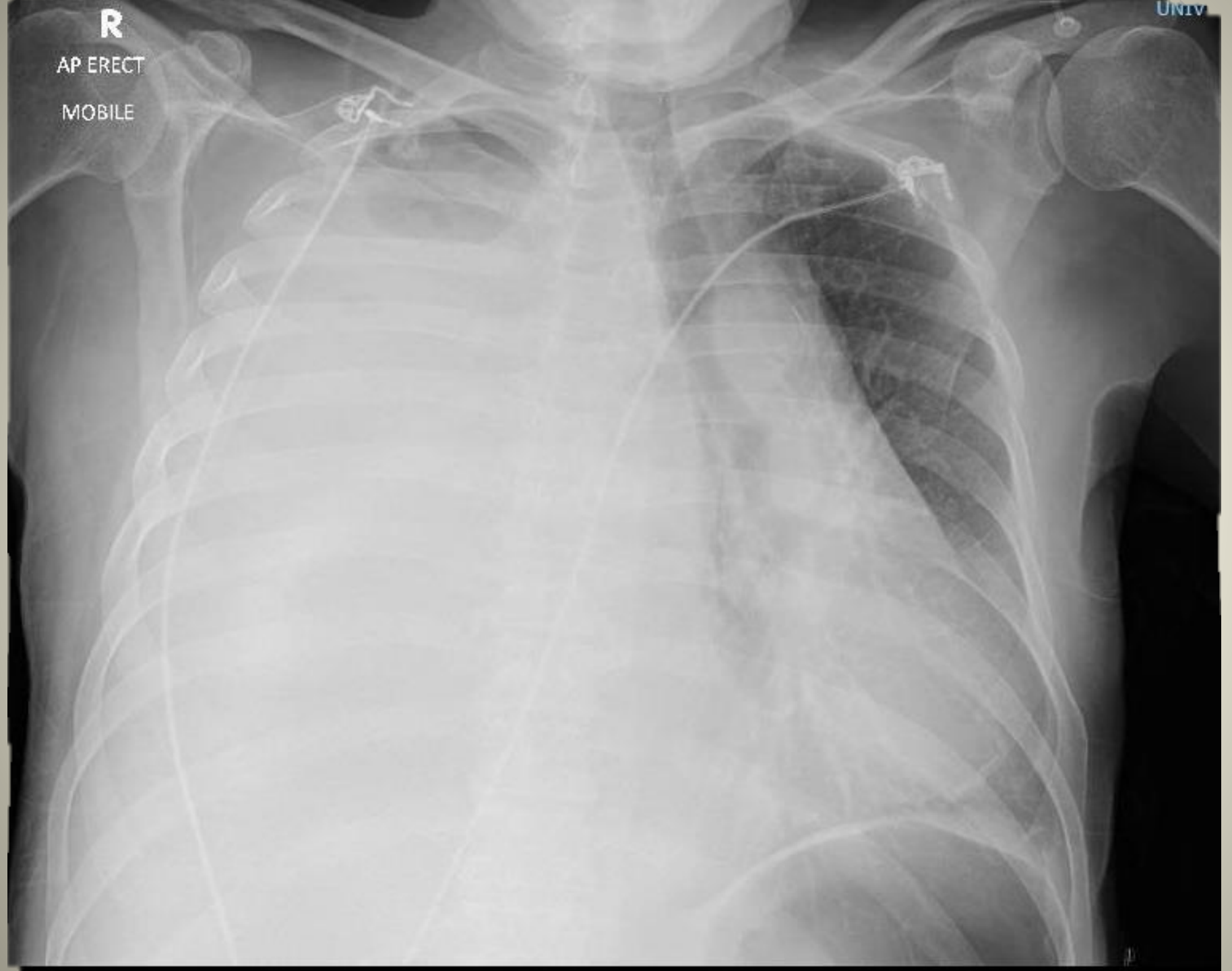
- هدف:
- برطرف کردن علت زمینه ای
- درمان تنگی نفس(توراسنتز)
- درمان جراحی(پلورکتومی)
- گذاشتن شانت پلوروپریتیونئال

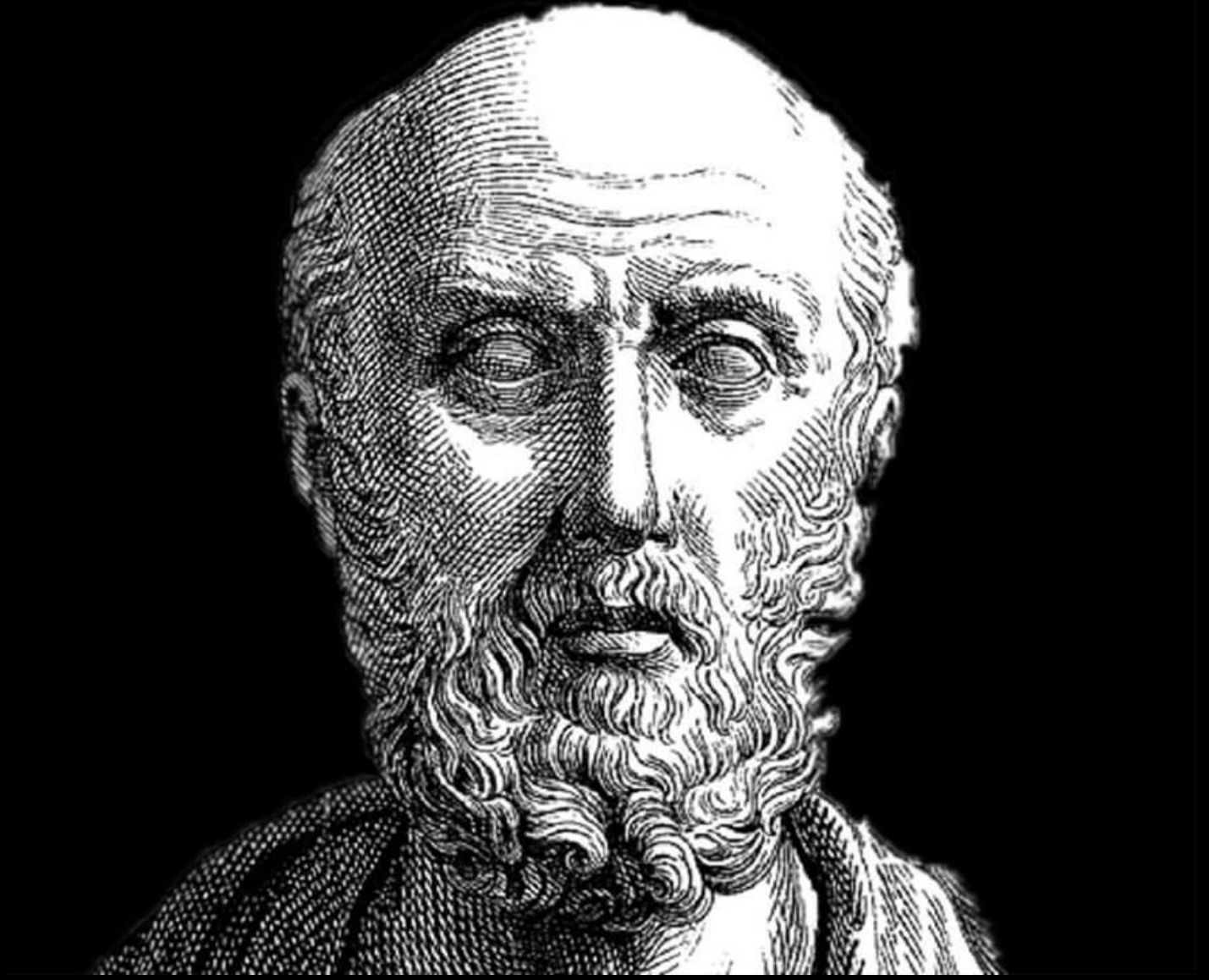
Empyema

تعریف : عبارت از وجود مایع چرکی در حفره پلورال

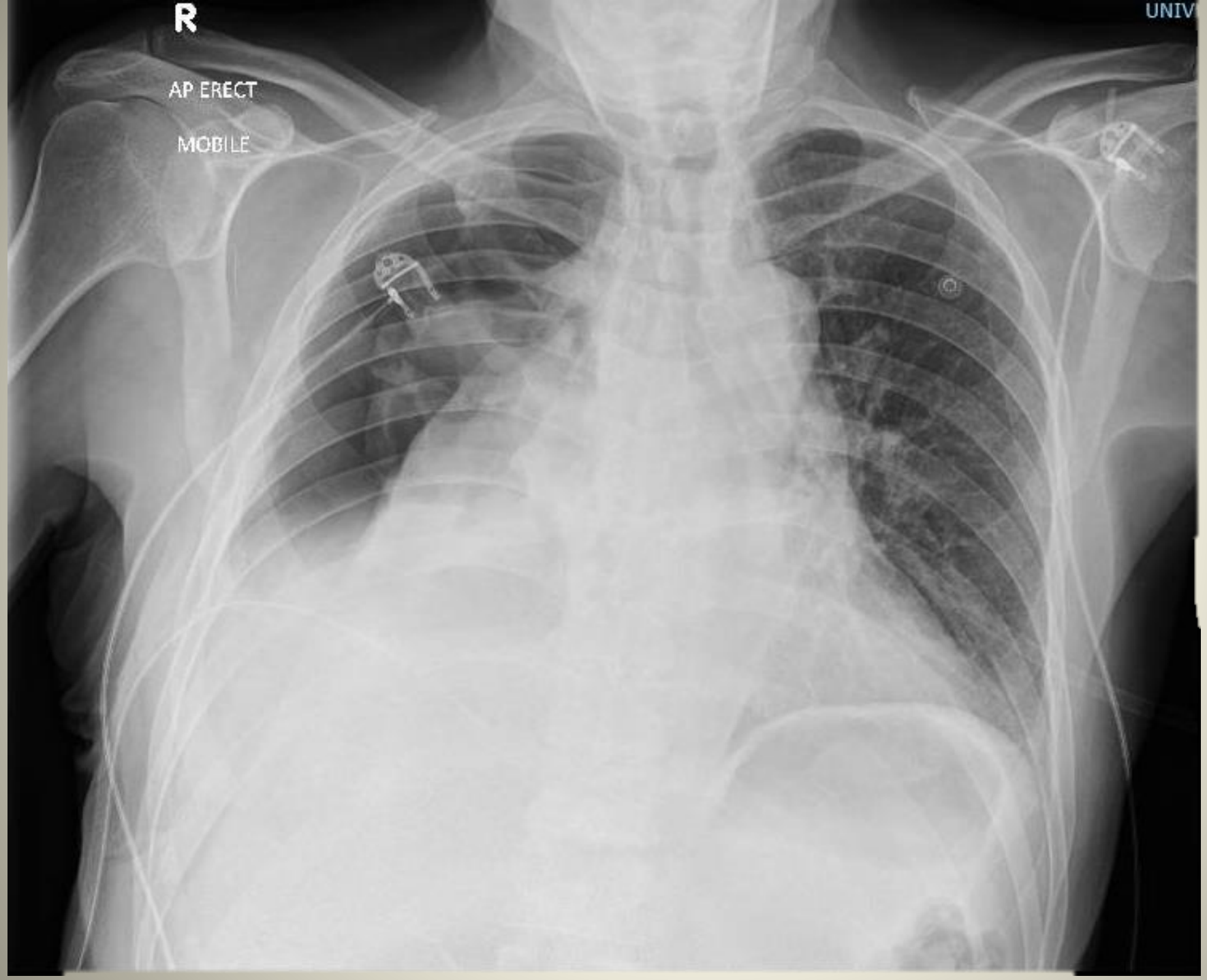
علل : شایعترین علت عبارتست از تجمع مایع بدنبال پنومونی باکتریایی یا آبسه ریه

ولی سایر علل با شیوع کمتر عبارتند از : بعد از جراحی یا بعد از تروما


R
AP ERECT
MOBILE



If an empyema does not rupture, death will occur
- Hippocrates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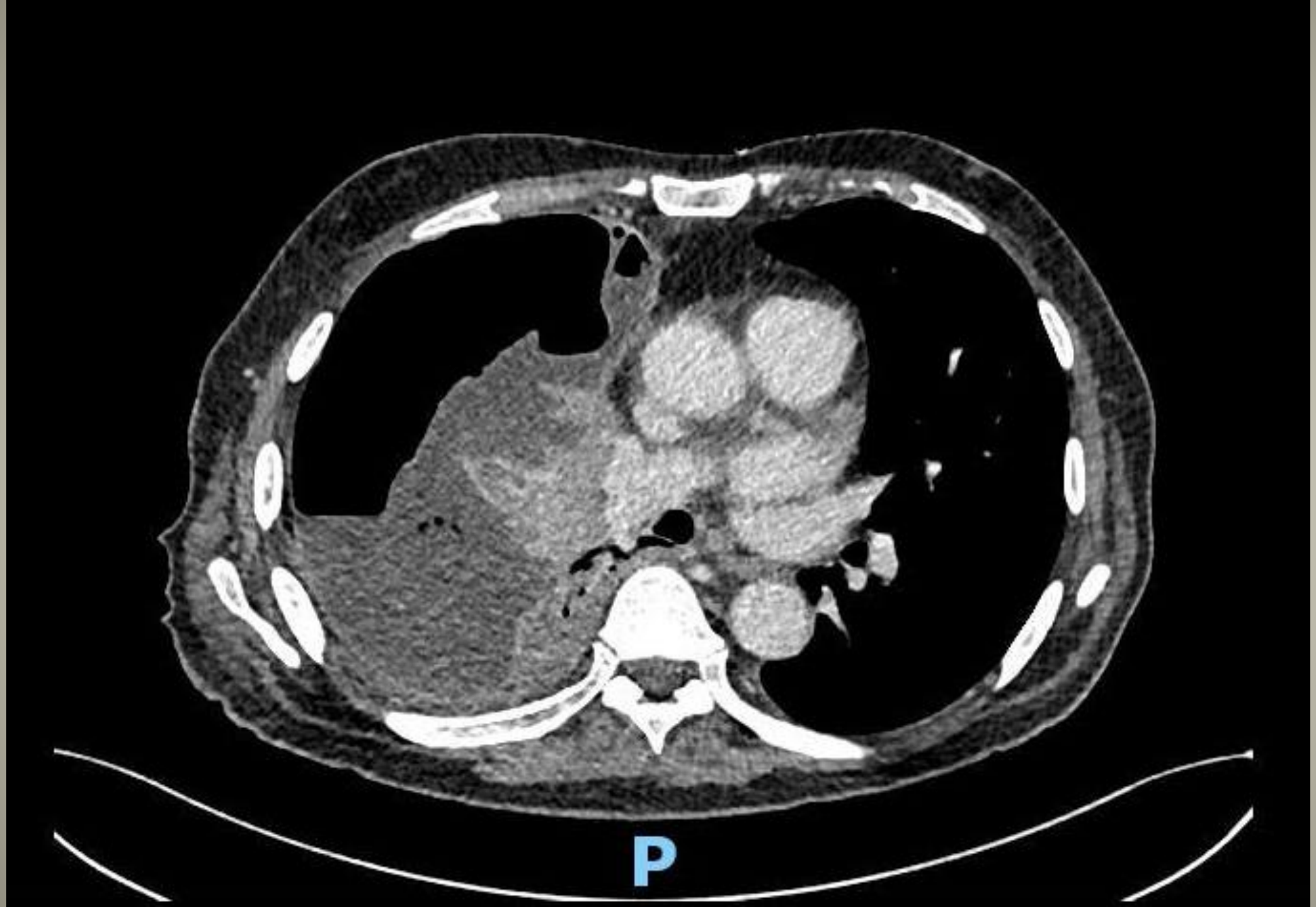
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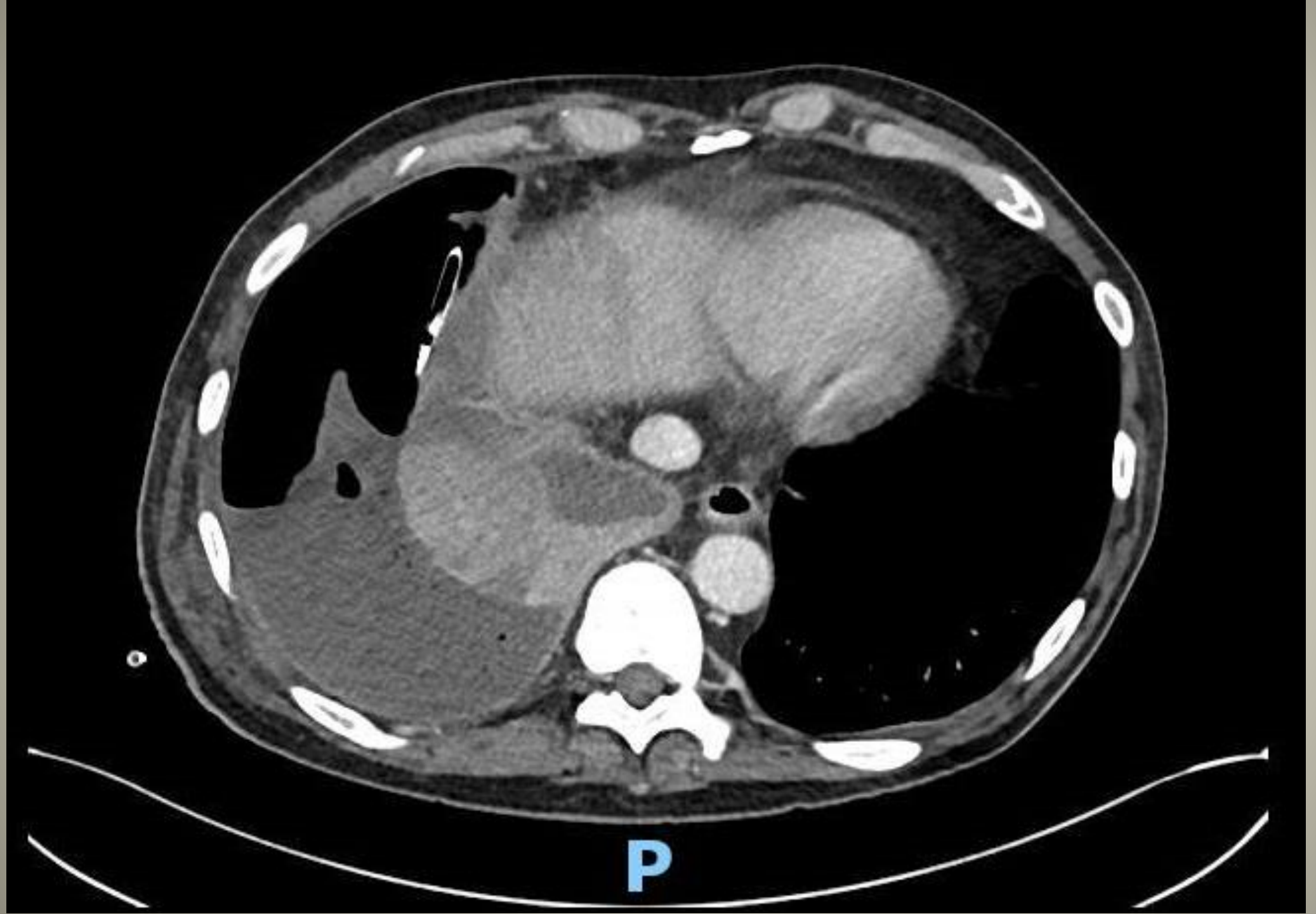
Empyema

- تظاهرات بالینی:
- بیمار به شدت ناخوش است
- علائم شبیه به عفونت حاد تنفسی یا پنومونی(تب، تعریق شبانه، دردهای پلورتیکی، سرفه، تنگی نفس، بی اشتهایی، کاهش وزن

Empyema

- بررسی و یافته های تشخیصی:
- در سمع:کاهش و یا فقدان صداهای تنفسی
- در دق: کاهش صدای لرزش لمسی
- CTS و CXR
- توراسنتز تشخیصی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Results

- Fluid LDH >50000
- pH unable due to viscosity
- Protein 63
- Glucose <0.6
- Fluid grew Streptococcus anginosus

Clinical presentation

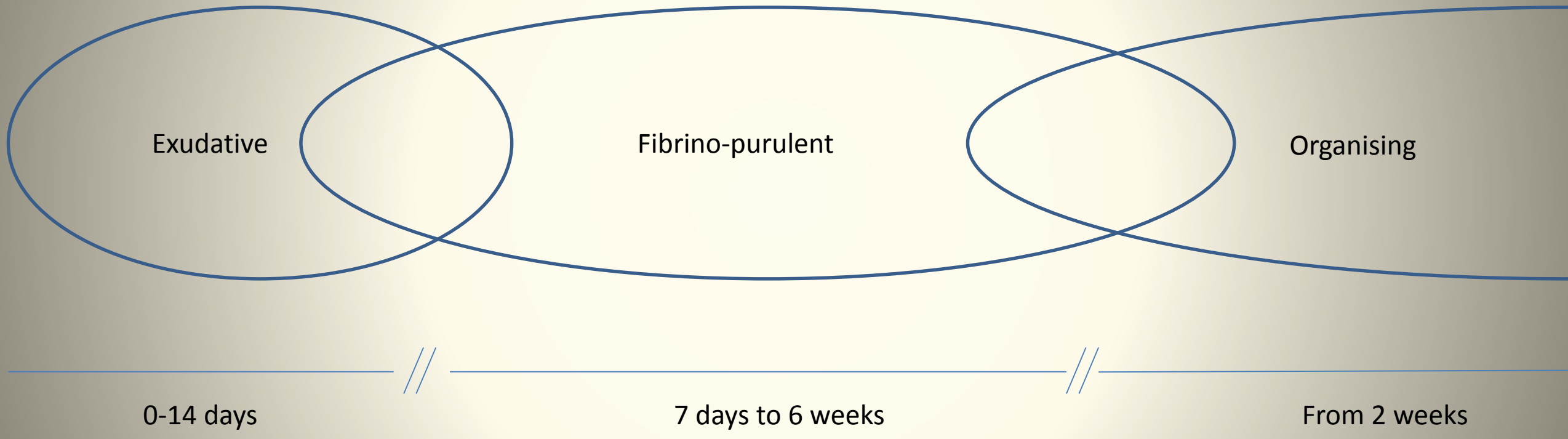
- Can be diverse – fevers with effusion and non-resolving pneumonia
- Another pattern (more elderly) – Malaise, anorexia, weight loss (as in the case) can end up on malignant diagnostic pathways, leading to delays
- Mortality 10-20%

3 stages of Empyema

- Exudative
- Fibrino-purulent
- Organising

- These are not necessarily linear and are dependant on innumerate host-pathogen factors
- Eg: patient with heavily loculated, but serous appearing effusion

Stages of Empyema



Exudative stage (1-7 days) – “parapneumonic effusion”

- Bacterial invasion of lung parenchyma
- Parenchymal inflammation leads to visceral pleural membrane permeability and leakage of interstitial fluid
- Mesothelial lining further disrupted by neutrophil infiltration, leading to mesothelial cells releasing pro-inflammatory cytokines such as IL6, IL8 and TNF-alpha
- High level of endogenous fibrinolytic enzymes
- Anatomical distortion occurs, leading to increased fluid
- Normal pH, glucose and culture negative
- Important to recognise these early and treat

Fibrino-purulent stage (4 days – 6 weeks)

- Bacteria transcend the pleural membrane
- High fibrinolytic levels start to be suppressed by a rise in plasminogen activator inhibitors - PAI-1 and PAI-2
- PAI-1 levels now correlate with residual pleural thickening
- Progressive leukocytosis and fibrin accumulation initially at the pleural surfaces
- Progressive tendency towards loculation
- Bacterial and neutrophil phagocytic activity use glucose and produce lactate
- LDH rises due to release from polymorphs and mononuclear cells

Organising stage (2-6+ weeks)

- Fibroblasts grow onto pleural surfaces
- Formation of inelastic membrane “pleural rind”
- Platelet-derived growth factor and transforming growth factor beta can lead to pleural fibrosis
- Pleural thickness inhibits antibiotic penetration further
- Microbial biofilm occur further reducing antibiotic effectiveness
- Collagen deposition in this phase reduces the effectiveness of fibrinolysis and surgery becomes essentially inevitable

Late complications

- Destruction of lung tissue, bronchopleural fistulae/pyopneumothorax
- Spontaneous entry into chest wall “empyema necessitans”
- Rupture into abdominal cavity

Aims of treatment

- Control of infection
- Expansion of lungs

Antibiotics

- Penicillin and metronidazole most rapidly formed equilibrium of serum and pleural concentrations, flowed by ceftriaxone and clindamycin (rabbit models)
- It remains unclear how effective antibiotics are at preventing bacterial replication in an infected pleural space

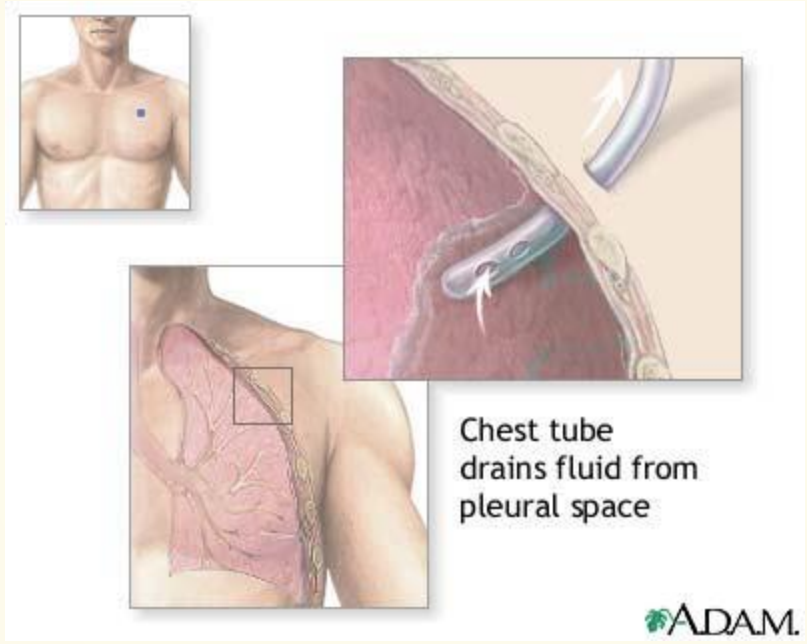
Chest tube

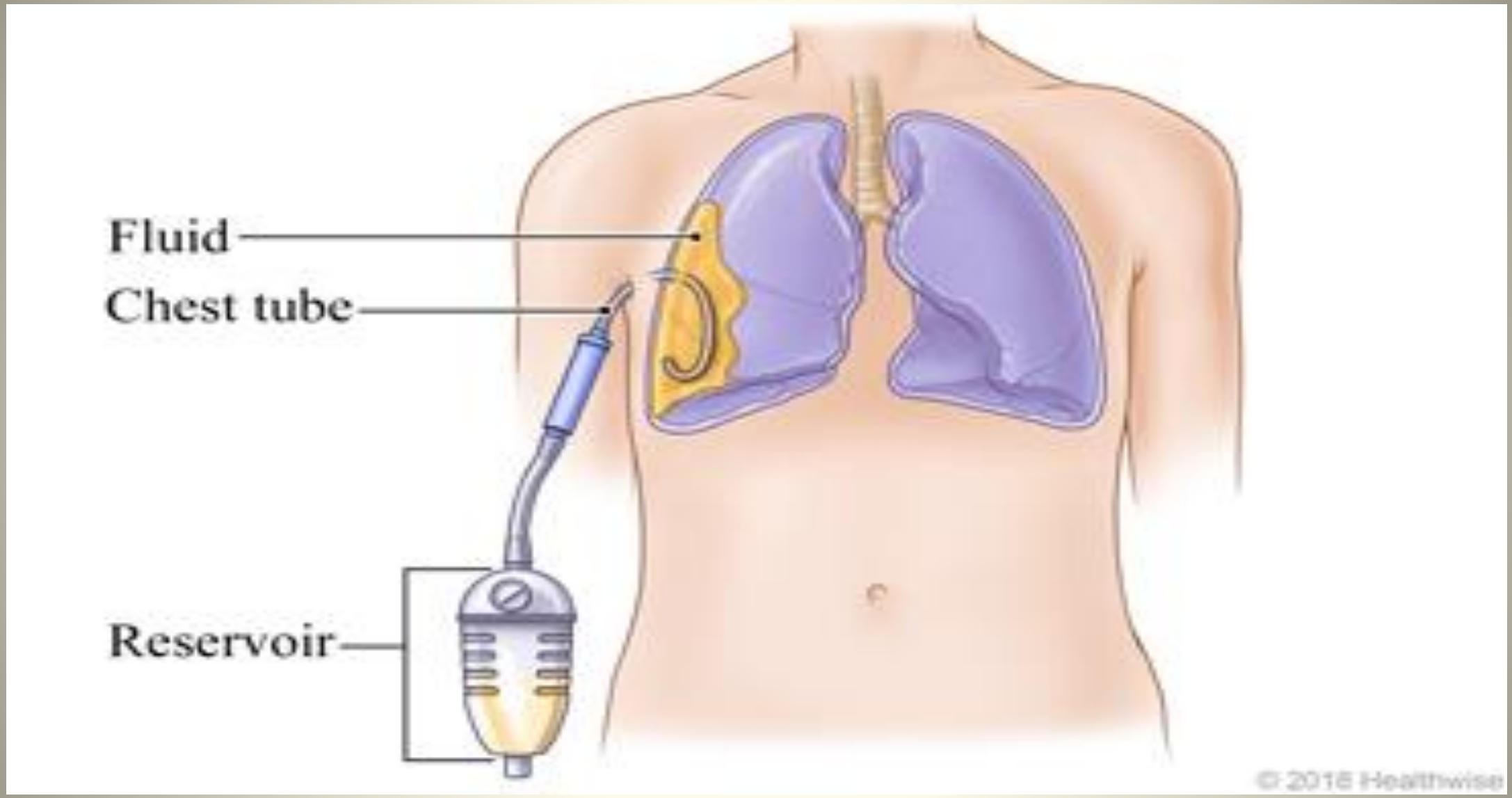
A chest tube is a hallow plastic tube that is inserted into the chest cavity to remove or allow the drainage of fluid, blood or air from that portion of the chest cavity.

- Drainage is marked on the collection chamber and documented every 8 hours.

Indications for Chest Tube

- Empyema
- Complicated parapneumonic effusion PH <7.2
- Hemothorax
- Malignant effusion- chest tube +/- pleurodesis (sclerosants)


Chest tube
drains fluid from
pleural space
A.D.A.M.


Fluid
Chest tube
Reservoir
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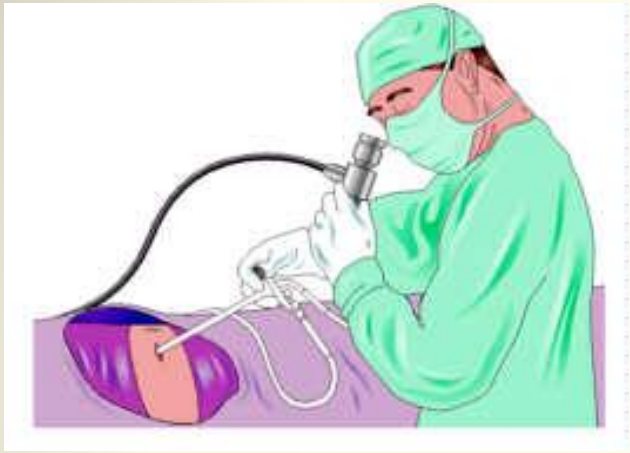
Sites For Chest Tube Insertion

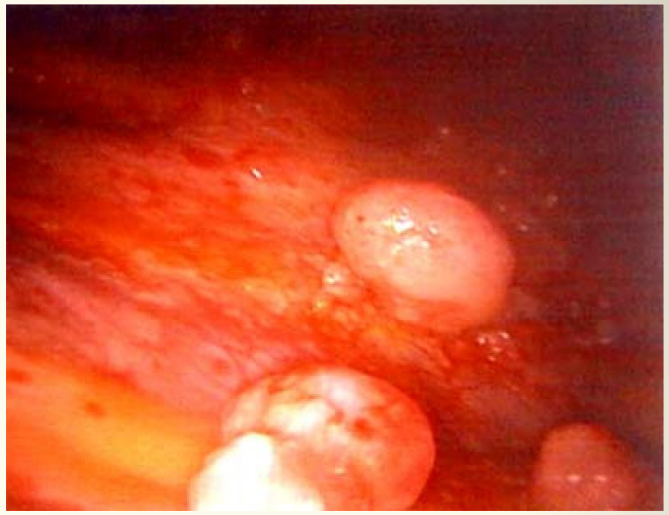
- If removal of air from around a collapsed lung is the goal, the tube is inserted into the upper anterior chest, in the second to fourth intercostal space.
- If removal of fluid is the goal, such as after an injury, the tube is inserted in the lower lateral chest, in the eighth or ninth intercostal space.
- If a patient has both air and fluid to drain, two tubes are inserted and may be joined with a Y connector before connecting to the tubing that leads to a drainage system.

Surgery - VATS

- RCTs show 80% can be managed medically
- VATS has widened the number of patients suitable for surgery
- Current guidance is for usage in treatment failure or advanced cases
- In advanced cases VATS treatment failure increases with conversion to decortication up to 61%
- VATS decortication mortality 2-6%
- Complication rate 9-40%
- Persistent air leak, pain, bleeding, infection, residual pleural space
- No data for best time for surgery

Thoracoscopy





Thank You So Much