RS

PATHOLOGY

MODIFIED NO. 2

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OBSTRUCTIVE LUNG DISEASES

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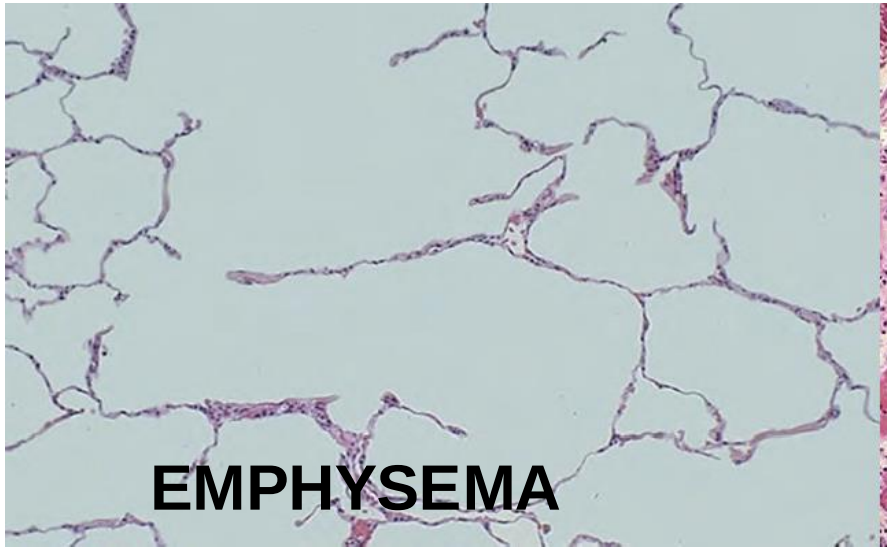
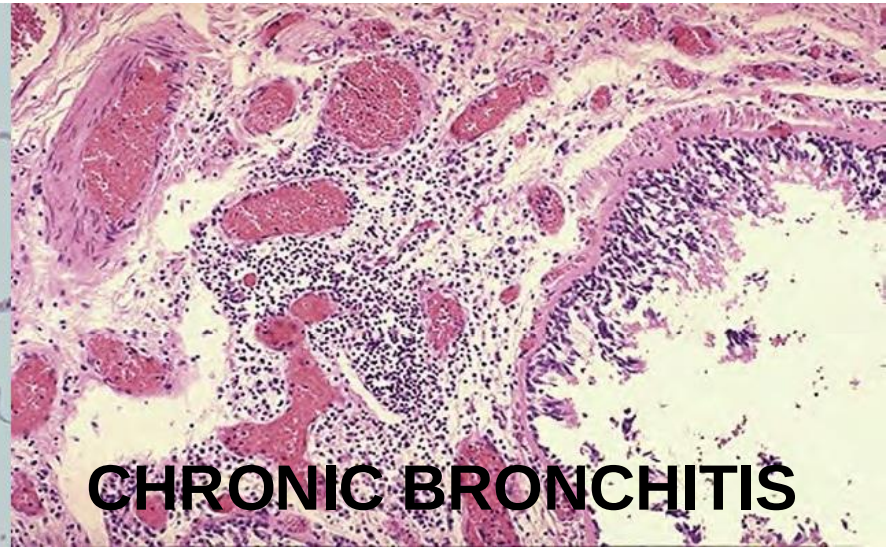
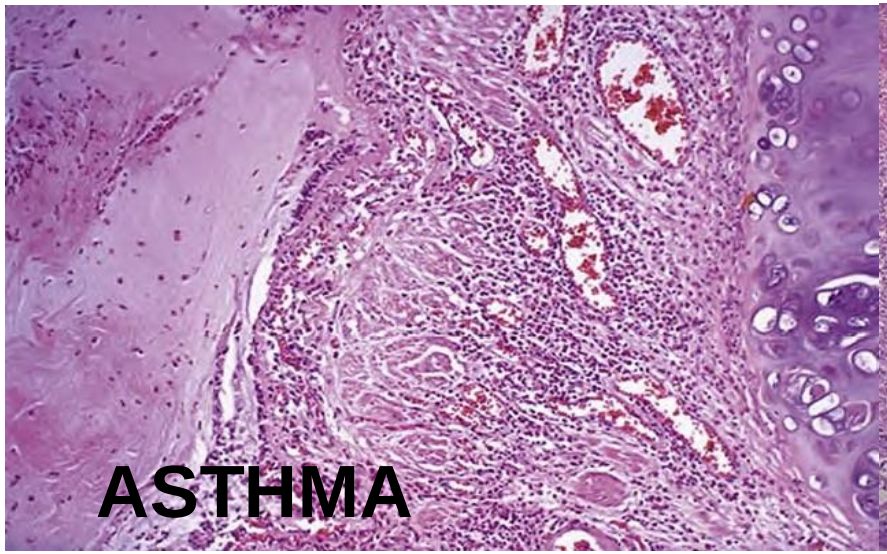
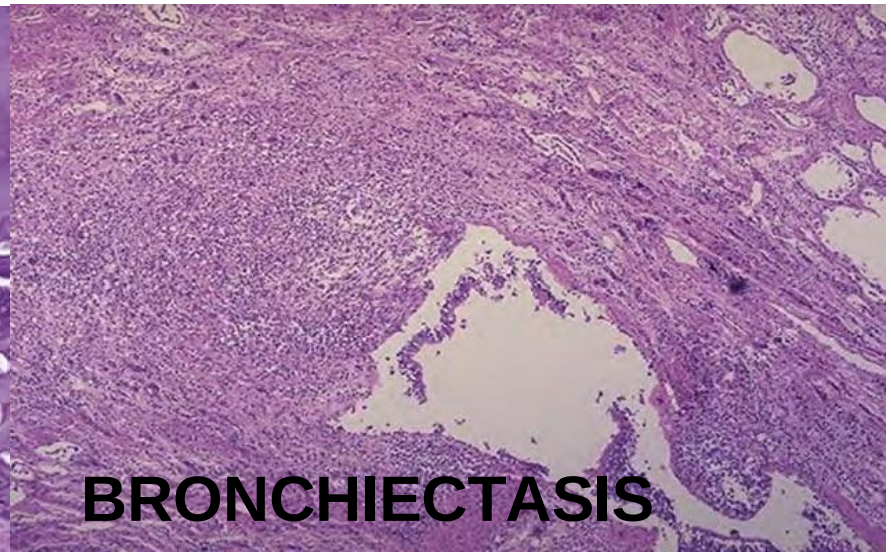
CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)









- Although each one of these diseases has its own characteristic features in terms of clinical features, morphology and radiography. However, **the overlap between Emphysema, Asthma and chronic bronchitis is considered common.**

some general information regarding obstructive pulmonary diseases :

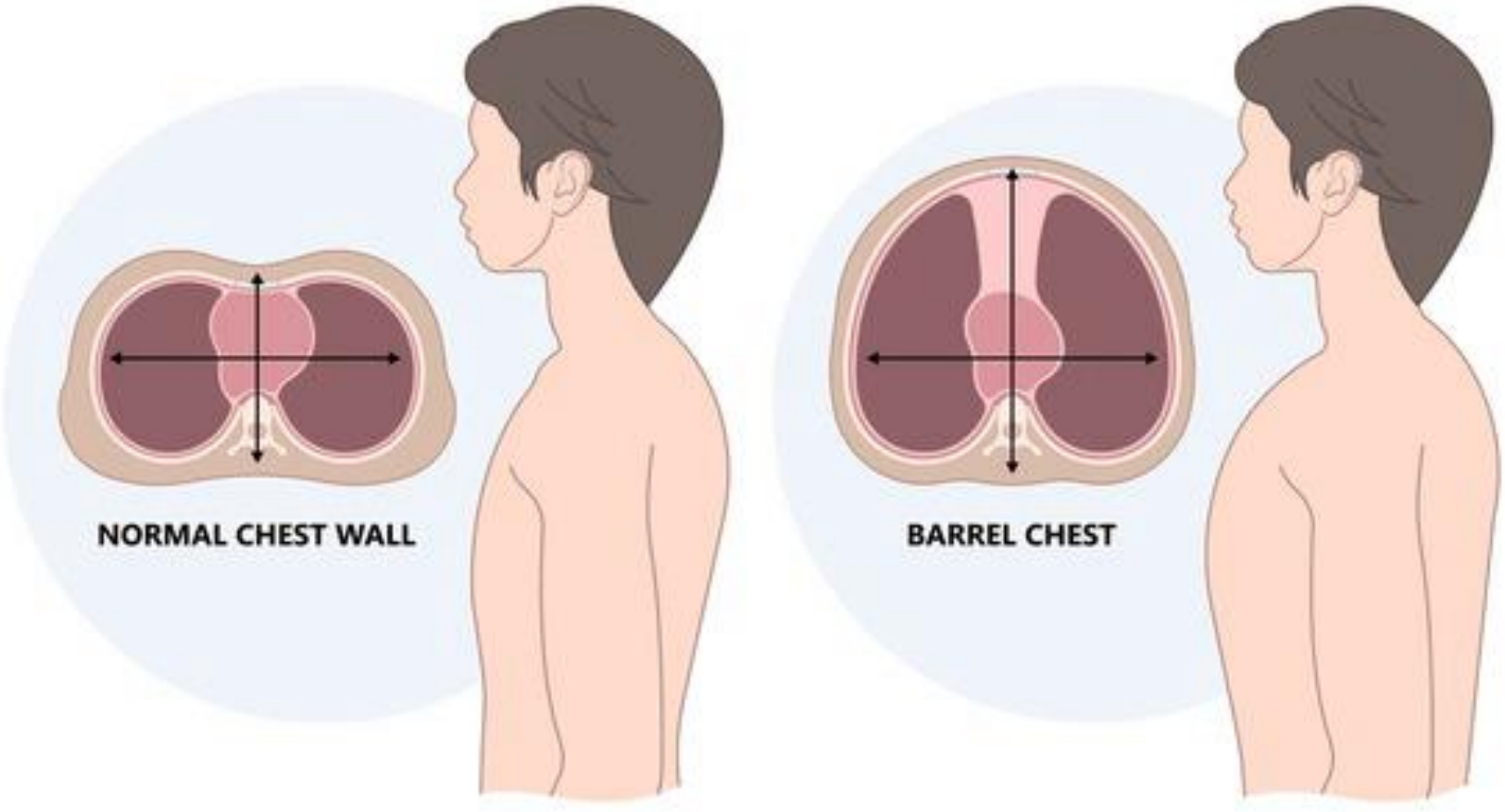
- In these diseases **It's hard to Exhale, and to get the air out**. As a result the **air accumulates** in the lungs causing **air trapping** > resulting in **hyperinflated lungs,** [but it could be normal].

- The hyperinflated lungs cause **Anterior posterior dilatation of the chest wall**, and it might be described as **Barrel Chest wall** , due to air trapping.

- When talking about **obstructive pulmonary** diseases, we can imagine the lungs as a **very old rubber band**. If we stretch it > is a **stretchable** and the **compliance is easily increased**. So it is **easy** to **inhale and fill the lung with air**.However, If we removed this stretching Force, the **lungs will not go back to their initial size**, due to **defects in the elastic recoil** > resulting in **defect in exhalation > ending up in accumulation of air.**

- In contrast , in **restrictive pulmonary diseases ,** we can imagine the lung as a **brand new rubber band**, if we stretch it **it's not stretchable** > **the compliance is low**. So there is a **difficulty in inhalation and filling the lung with air**. So the problem is in **inhalation**.

Extra pic



- **Because Emphysema and Chronic bronchitis share in many cases the same underlying etiology , which is the tobacco smoking. As a result, they are typically grouped together under the broader term chronic obstructive pulmonary disease (COPD).**

- **Total lung capacity (or total lung volume)** : **(TLC) is the volume of air in the lungs upon the maximum effort of inspiration.**
- **lung compliance : is a measure of the lung's ability to stretch or expand**

- In **obstructive** pulmonary diseases , **TLC or TLV is normal or increased** (high compliance, no problem in inhalation)
- In **restrictive** pulmonary diseases , **TLC or TLV is decreased** (low compliance, can't fill the lungs with air)

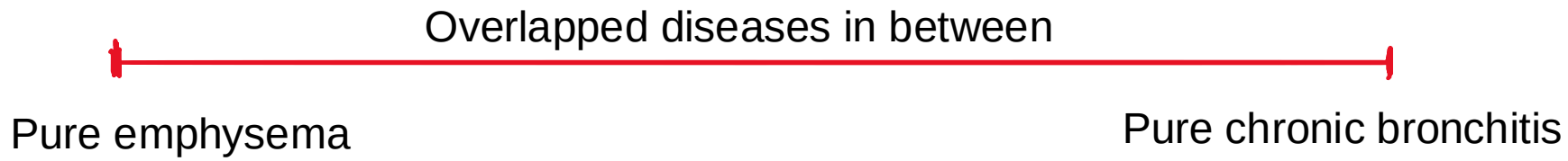
"In **obstructive** pulmonary diseases , **TLC or TLV is normal or increased** "
- Why it might be increased?
- there is often air trapping, which can increase residual volume (RV) and contribute to the observed increase in TLC.

It’s hard to get the air OUT

It’s hard to EXHALE

Lungs are hyperinflated

CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)



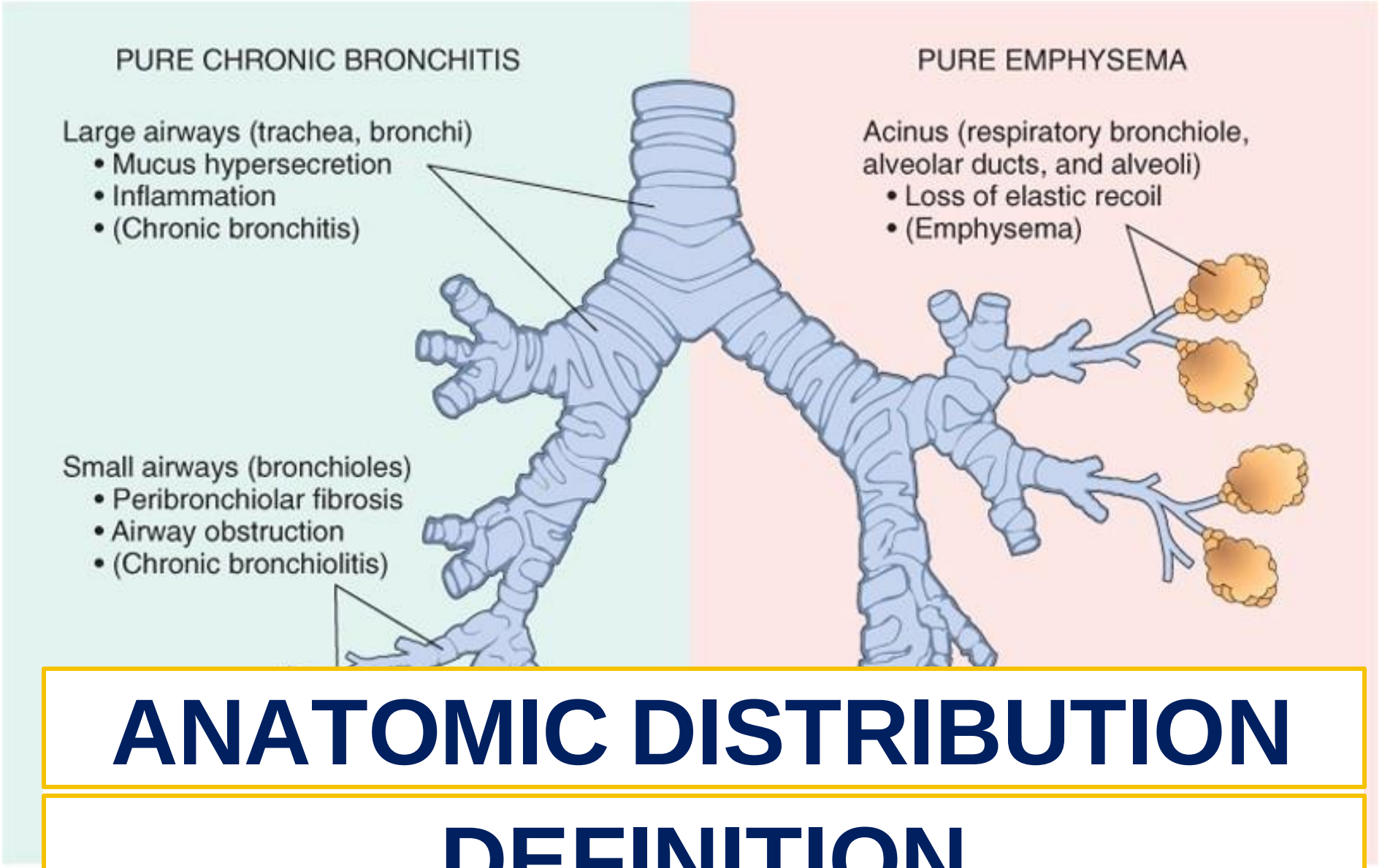
- COPD contains two main entities : **emphysema and the chronic bronchitis.**
- We can think about COPD as a **spectrum,** at one extreme we have **pure chronic bronchitis** , And on the other extreme we have **pure emphysema**. In between them **overlapped** cases are seen .
- Examples on overlapping:
- Dominant emphysema with minimal bronchitis
- Dominant chronic bronchitis with minimal Emphysema

- COPD patients are not the same in many terms , due to this wide spectrum.

COPD:

- **defined by the WHO as** “a common, **preventable and treatable** disease that is characterized by **persistent** (**indicating its chronic**) respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities caused by exposure to noxious particles or gases.” This will result in a process that eventually will result in obstruction.
- **4th leading cause of death in the world**
- There is a strong association between heavy cigarette smoking and COPD.
  - 35% to 50% of heavy smokers develop COPD .
  - 80% of COPD is attributed to smoking.

- These are clues that the disease is common and a treatable.
- Treatable : because smoking is a modifiable factor
- common because smoking is very common

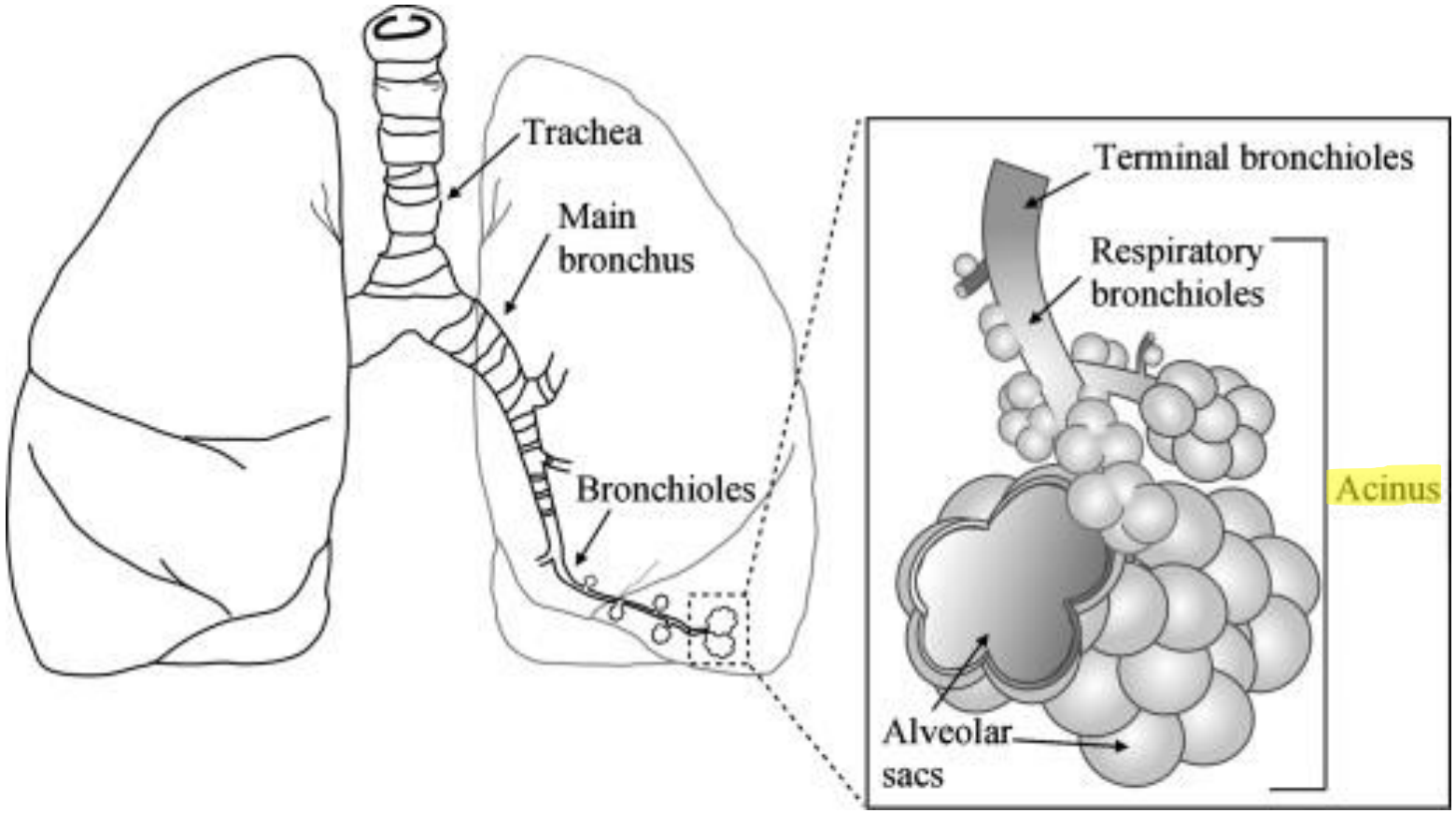


ANATOMIC DISTRIBUTION

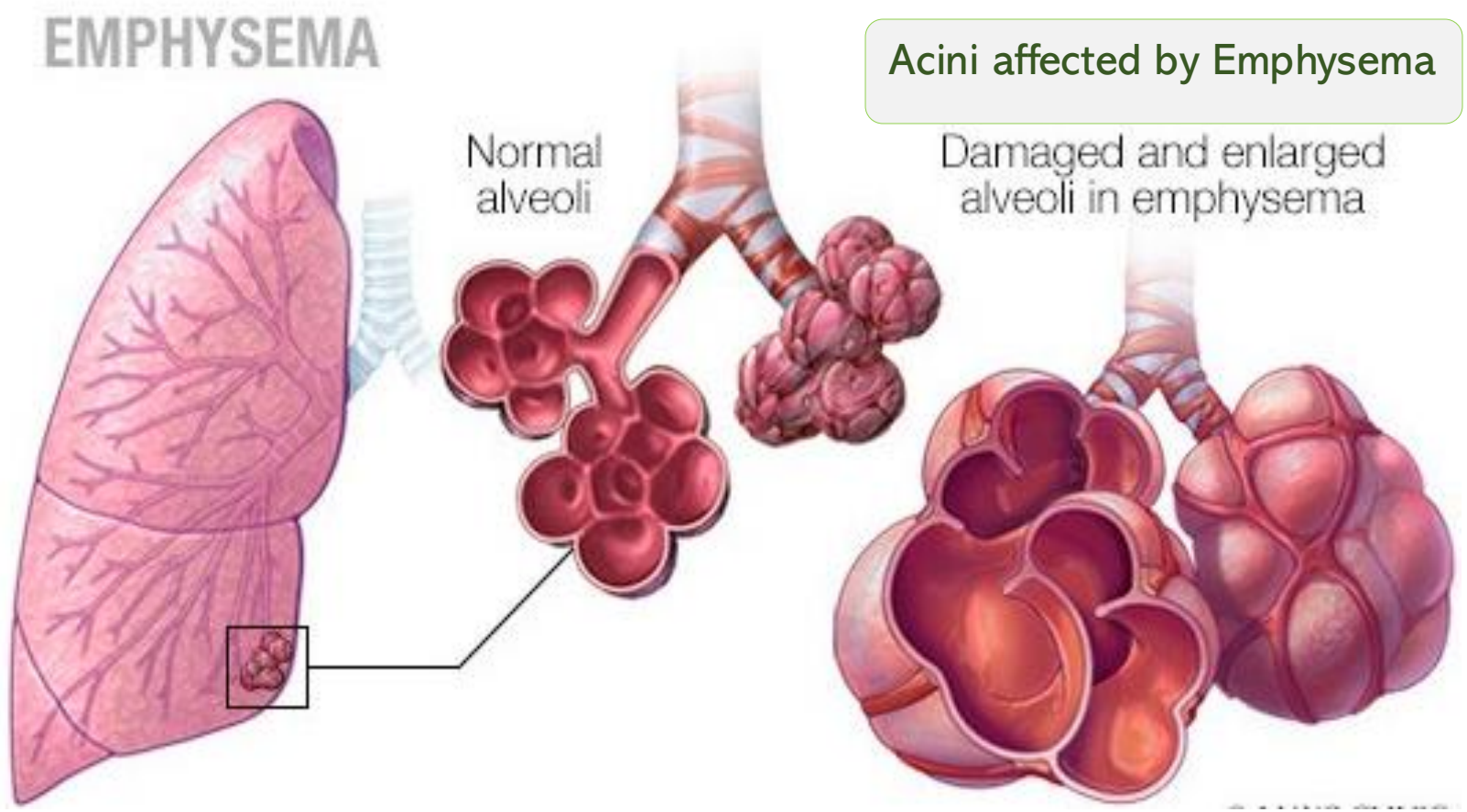
DEFINITION

	Pure Chronic bronchitis	Pure Emphysema
Anatomical distribution	**Large** airways (trachea , bronchi)	**Acinus** (respiratory bronchioles , alveolar ducts , alveoli)
Definition	Defined on the basis of **clinical findings** (a certain clinical scenario is enough for definition)	Defined on certain **morphological and radiographic findings** (we must make a bunch of tests , hand to hand with radiography)

- Later, when the diseases progressed, they can affect also the small bronchioles , causing chronic bronchiolitis .



Extra pic , showing the Acinus



Acini affected by Emphysema

Acini affected by Emphysema : **Permanant** enlargement of the airways **distal to the terminal bronchioles** (**irreversible process**) caused by distruction of the alveolar walls.

- Remember that the part of the airway **distal to the terminal bronchioles is called Acinus**

1. EMPHYSEMA

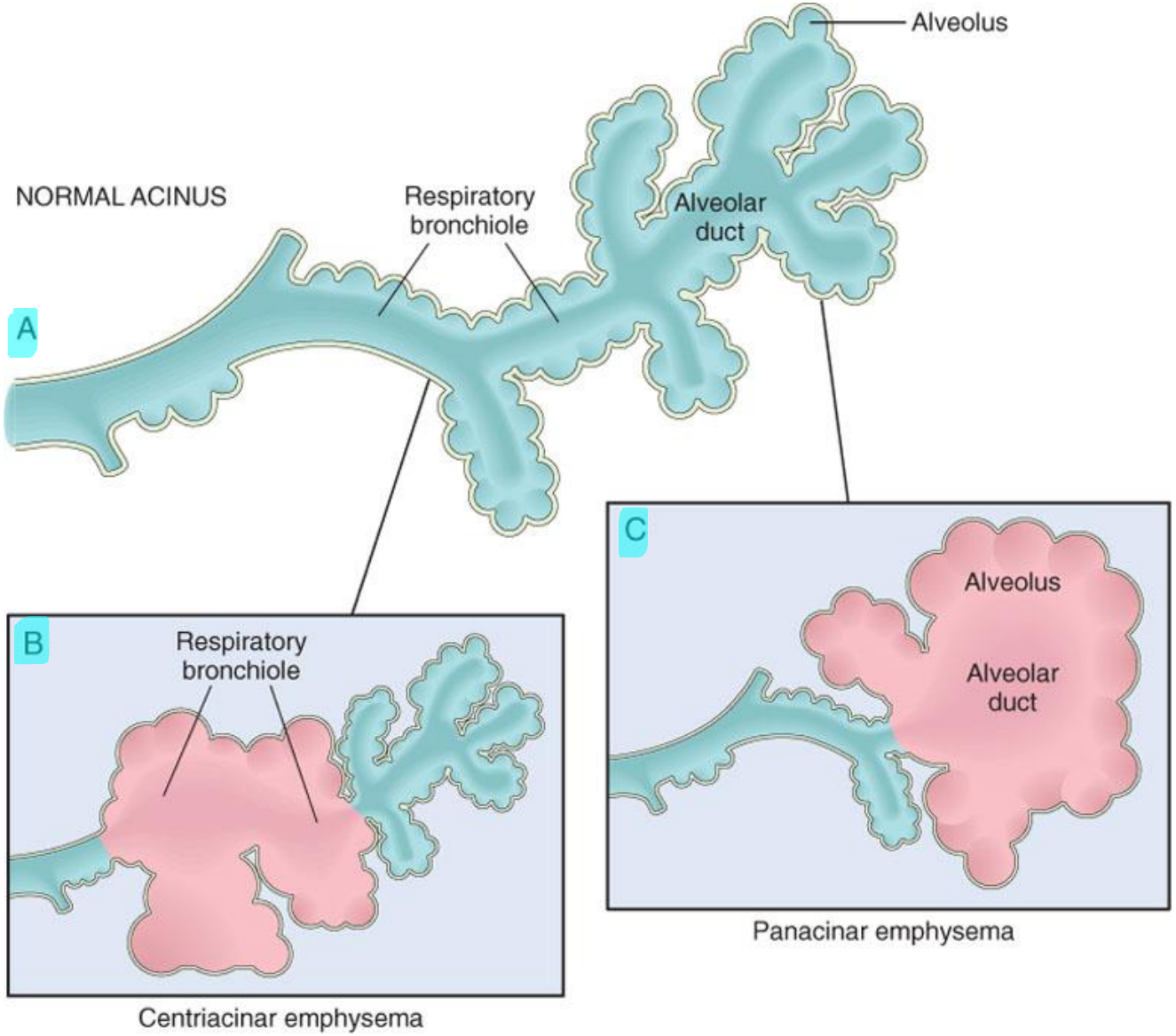
- **Permanent** (irreversible) enlargement of the airspaces **distal** to the terminal bronchioles with destruction of their walls.
- **Subtle but functionally important small airway fibrosis** → significant contributor to airflow obstruction.(patients with Emphysema always have airway obstruction due to this fibrosis that is associated with the process)
- Classified according to its anatomic distribution:

(1) Centriacinar (centrilobular)

(2) panacinar (panlobular)

(3) distal acinar (paraseptal)

(4) irregular



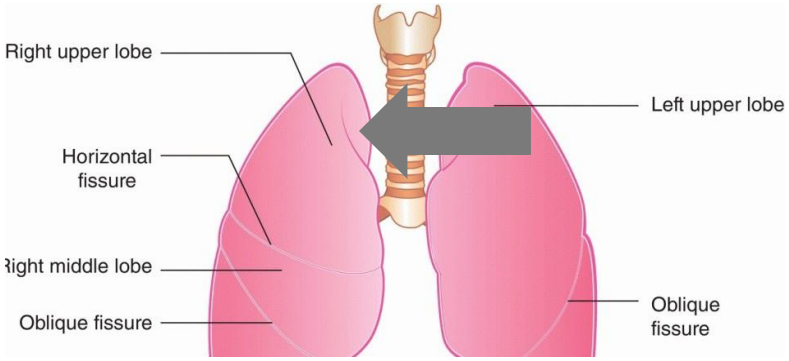
Normal acinus (A) :
starting from **respiratory bronchioles**, (which is a branch of the terminal the bronchioles) > **alveolar duct** > **alveolar sac.**

Centriacinar Emphysema (B) : it affects the **central part (proximal part)** which is the respiratory bronchioles, **while alveolar duct and sac are spared (not affected)**, specially on early disease
-Note: if the disease becomes advanced the whole all Acinus will be involved, and becomes indistinguishable from panacinar emphysema.

Panacinar emphysema (C) : it will start from the alveolar sac and duct then it will involve the respiratory bronchioles , **so it involves the entire acinus**

Centriacinar emphysema (the most common type of emphysema)

Strongly associated with **tobacco smoking**, that's why it is the most common (since tobacco smoking is common)



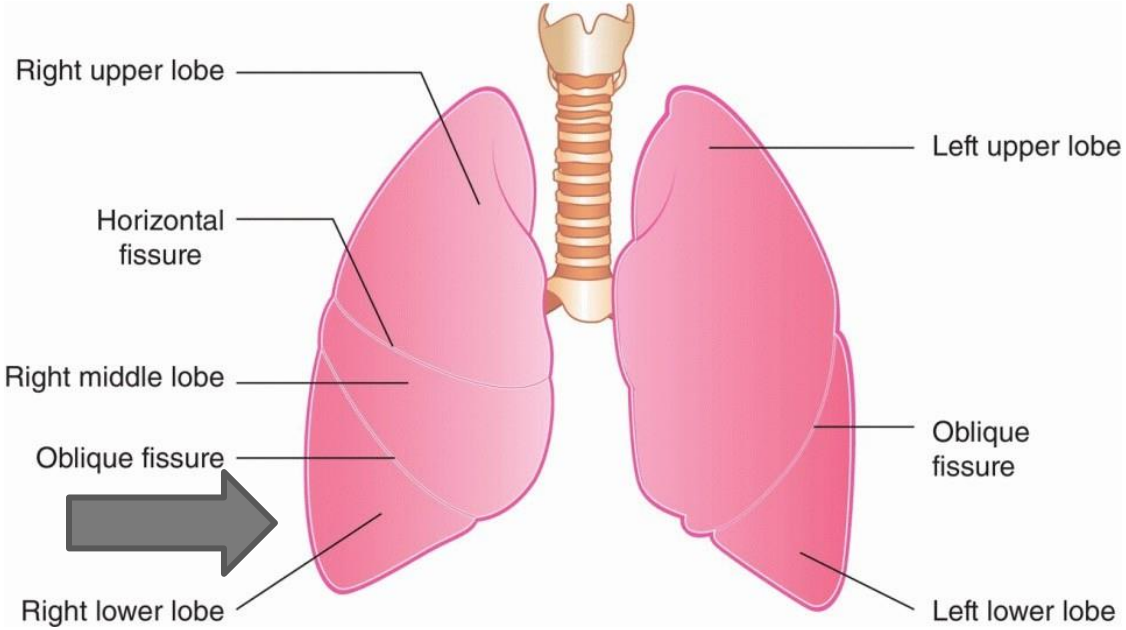
Because 90% of **chronic bronchitis is associated with tobacco smoking**, <u>centriacinar emphysema is the most common type to be associated with chronic bronchitis</u>

In terms of lung involvement Centriacinar emphysema is much more common and more severe in <u>**the upper half of the lung specially the apical segments**</u>

Panacinar emphysema

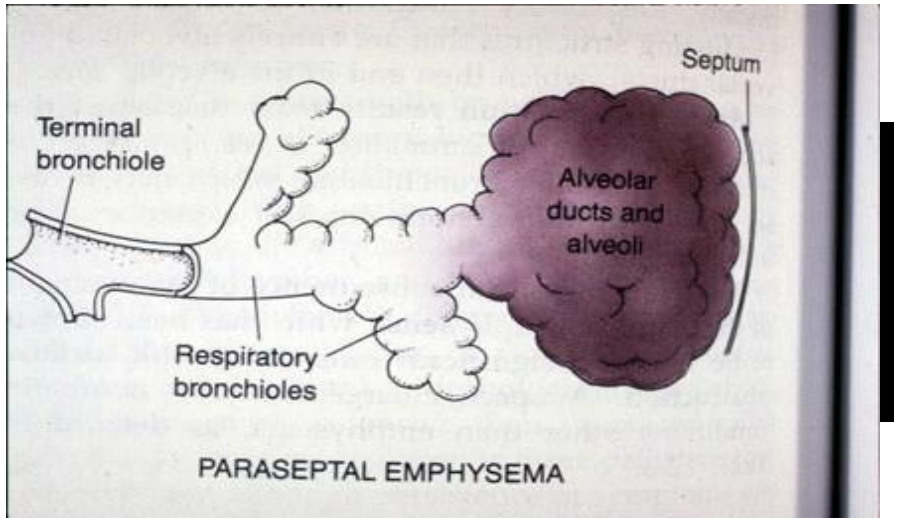
α_1-antitrypsin deficiency

Panacinar emphysema is associated with **alpha-1 antitrypsin deficiency** (which is a genetic disease associated with liver and lung diseases)



The **lower part** of the lung is more common and severely affected, specially the **lung base and anterior margin of the lung**

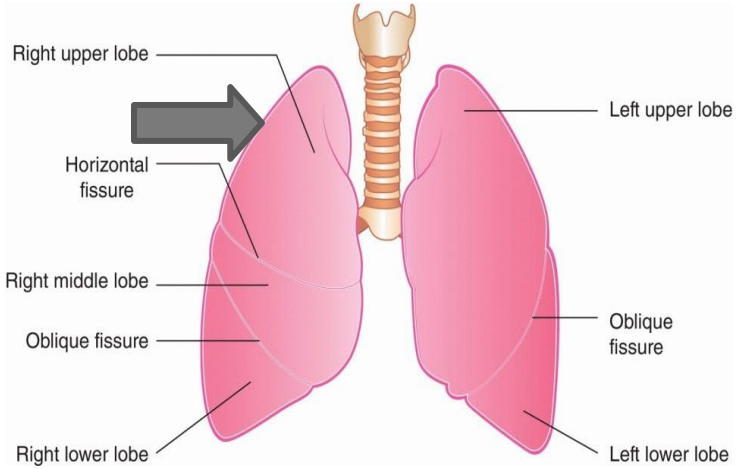
Paraseptal or distal acinar emphysema



Distal : it involves the **alveolar duct and sac**, however, **respiratory bronchioles are spared**

Paraseptal : adjacent to: the septae, margins of the lobules, lobular CT septae, to areas of scarring fibrosis and atelectasis.

- Most importantly: **Adjacent to pelural cavity**
- Etiology : unknown



The **upper part** of the lung is more common and severely affected, especially the apical segment.

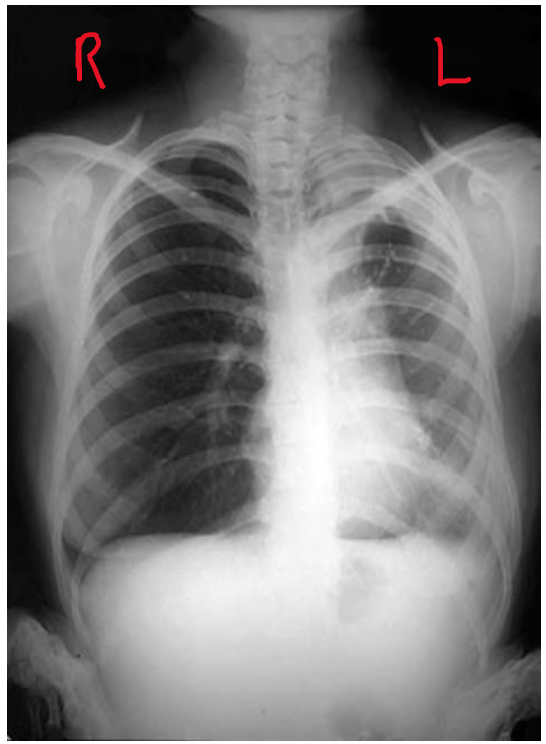
Paraseptal or distal acinar emphysema

As we said it is adjacent to **the pleural cavity, it might cause cystic spaces (.5 - 2 cm)**
These spaces **contain air**, and are prone to **spontaneous rupture**
Once they rupture, they leak air ito the pleural cavity, causing spontaneous pneumothorax

It is **The most common type of emphysema associated with spontaneous pneumothorax in young adults**

In other words :
In Paraseptal emphysema: Destruction occurs in the **distal alveoli** and alveolar ducts, near the terminal bronchioles.Creates enlarged airspaces (**cysts or bullae**) near the pleura.

Paraseptal or distal acinar emphysema



What is pneumothorax :

pneumothorax, commonly referred to as a collapsed lung, occurs when air escapes from the lung and accumulates in the space between the lung and the chest wall (the pleural space). This buildup of air can create pressure on the lung, causing it to collapse either partially or fully.

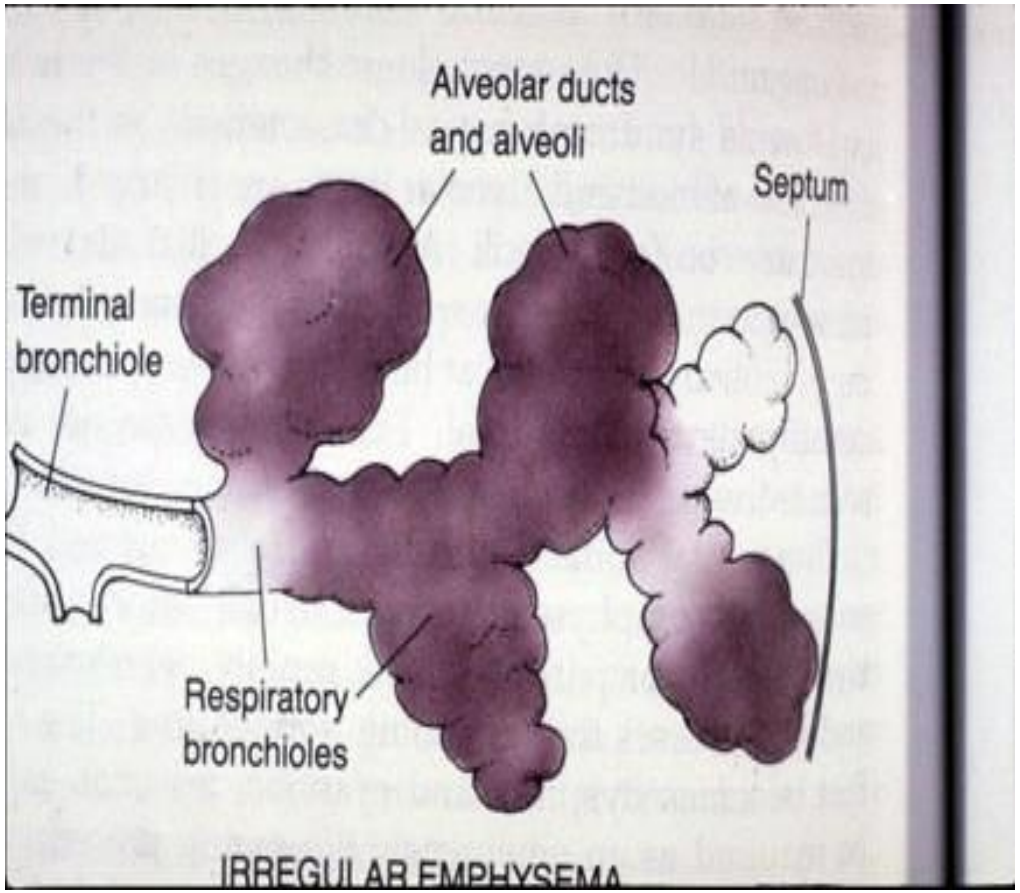
-The **right** side is affected by pneumothorax.
-The black color indicates that there is a loss in lung markings (pneumothorax is covering the whole right lung).

Heart and mediastinum are shifted to the left due to accumulation of air within the pleural cavity of the right side.

Causes of pneumothorax :

1.Mechanical ventilation
2. Direct penetrating injury
3. rupture of cyctic spaces (bullae)

Irrigular emphysema



- No specific pattern, any part of acinus can be involved
- it is almost invariably associated with **scarring**

Although it's common, but **it's clinically asymptomatic and insignificant**, usually diagnosed at **the autopsy** level

Remember : the Most common clinically diagnosed or symptomatic emphysema is **the centriacinar emphysema**

A 20-year-old, previously healthy gentleman is jogging one morning when he falls to the ground.He suddenly becomes markedly short of breath. In ER no breath sounds audible over the Rt side of the chest. A CXR shows shift of the mediastinum from right to left. A chest tube is inserted on the right side, and air rushes out. Which of the following underlying diseases is most likely to have produced this complication?

A. Centriacinar emphysema
B. B Chronic bronchitis
C. C. Distal acinar emphysema
D. D. Panlobular emphysema

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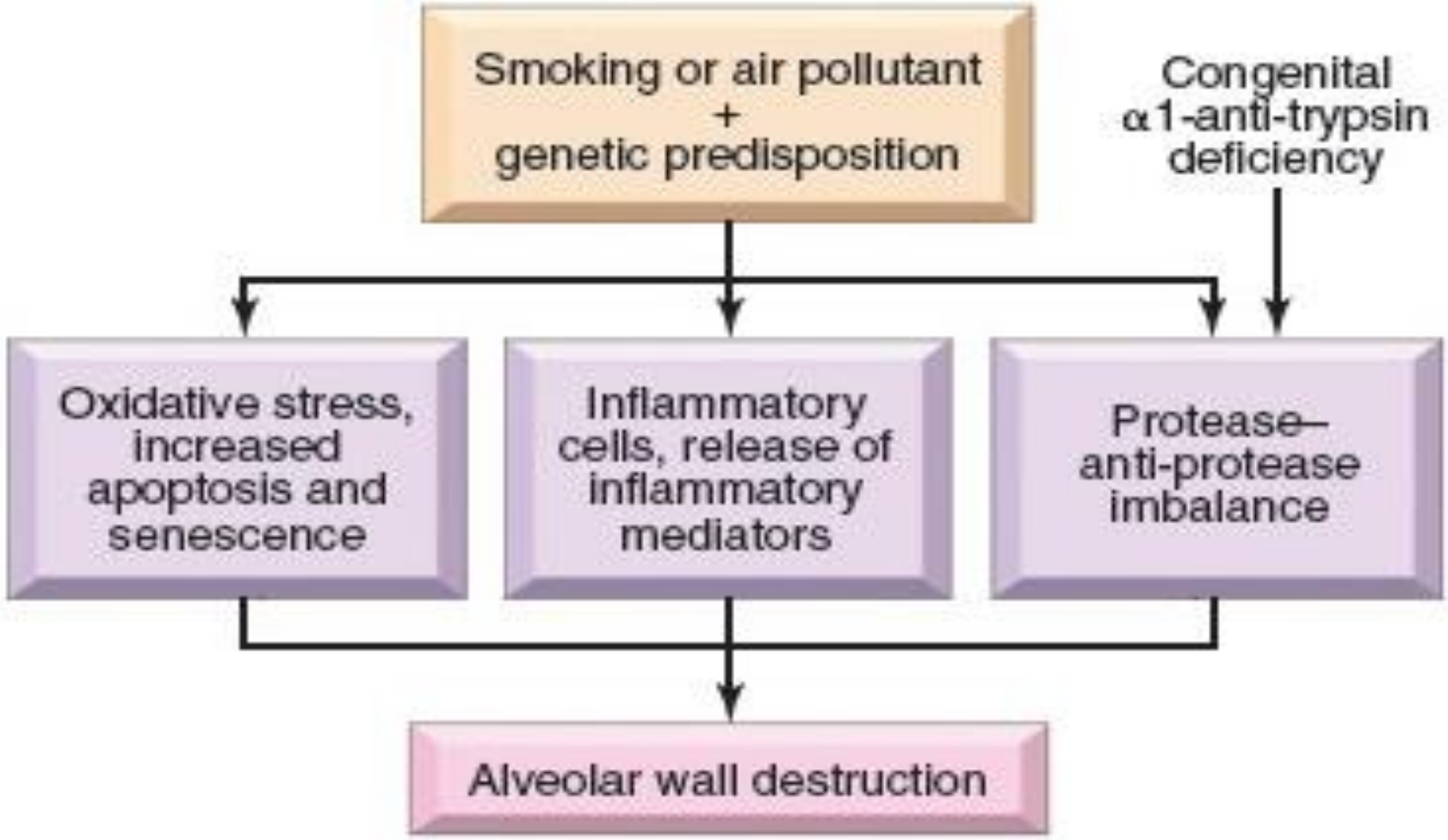
PATHOGENESIS



infections

Infections **cannot** directly cause emphysema
but can **exacerbate the condition** or trigger acute attacks in patients with existing disease.
These infections perpetuate the inflammatory response, further contributing to alveolar damage

Emphysema is a condition with two important rules to remember.

Rule 1: Symptomatic Threshold

For a patient to present with symptoms and seek medical attention, they typically need to lose at least **one-third** of their functioning lung parenchyma. This significant loss of functional tissue is necessary for the patient to experience noticeable symptoms.

Rule 2: Major Causes

Although there are multiple causes or underlying etiologies, the most significant ones that can lead to this extent of lung damage are **smoking** and **alpha-1 antitrypsin deficiency**. Smoking, in particular, is the primary cause, while alpha-1 antitrypsin deficiency represents a **genetic predisposition**. These two factors cause alveolar wall destruction through different mechanisms.

Emphysema is characterized by the **irreversible and permanent enlargement of air spaces**, resulting from the destruction of alveolar walls.

Mechanisms in Smoking-Induced Emphysema

1. Oxidative Stress

Tobacco smoke and the inflammatory response produce ROS and reactive nitrogen species (RNS), compounding the oxidative damage.

2. Inflammation and Protease Activity

Tobacco smoke contains noxious particles that trigger an inflammatory reaction. Neutrophils and macrophages are activated, releasing proteases (e.g elastase) and reactive oxygen species “ROS”, which directly damage alveolar walls.

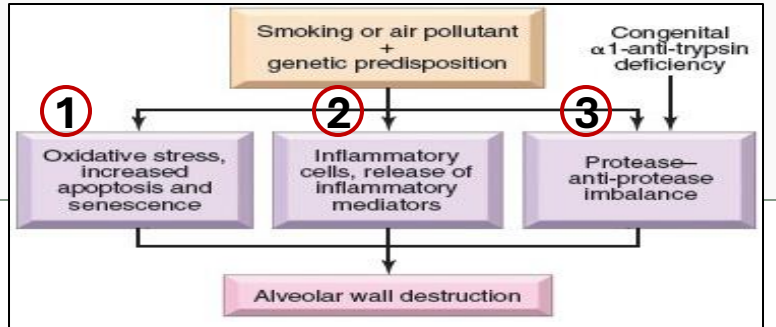
3. Protease-Antiprotease Imbalance

Normally, the body maintains a balance between proteases (destructive enzymes) and anti-proteases (protective enzymes). In emphysema, this balance is disrupted, either due to smoking or genetic predisposition, allowing unchecked protease activity and tissue destruction.

Genetic Predisposition: Alpha-1 Antitrypsin Deficiency

Patients with this condition have a deficiency in alpha-1 antitrypsin, a key anti-protease that inhibits elastase. Elastase destroys elastic fibers, which are essential for alveolar integrity.

When both smoking and alpha-1 antitrypsin deficiency are present, the damage is compounded, leading to severe disease.

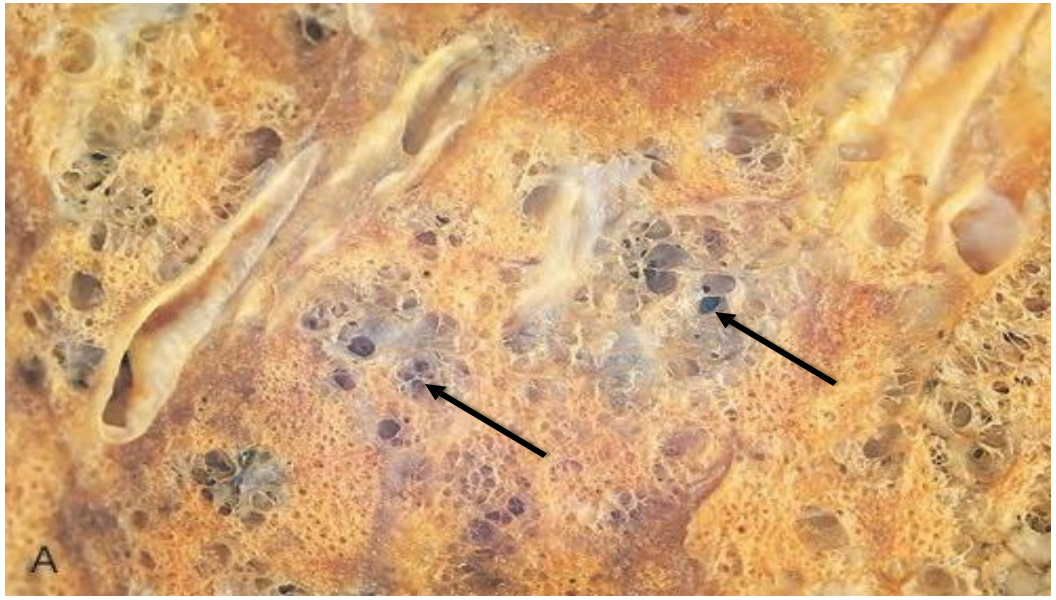


MORPHOLOGY Macroscopic:

Advanced emphysema→ voluminous lungs

Centriacinar emphysema . Central areas show marked emphysematous damage (arrows) surrounded by relatively spared alveolar spaces.

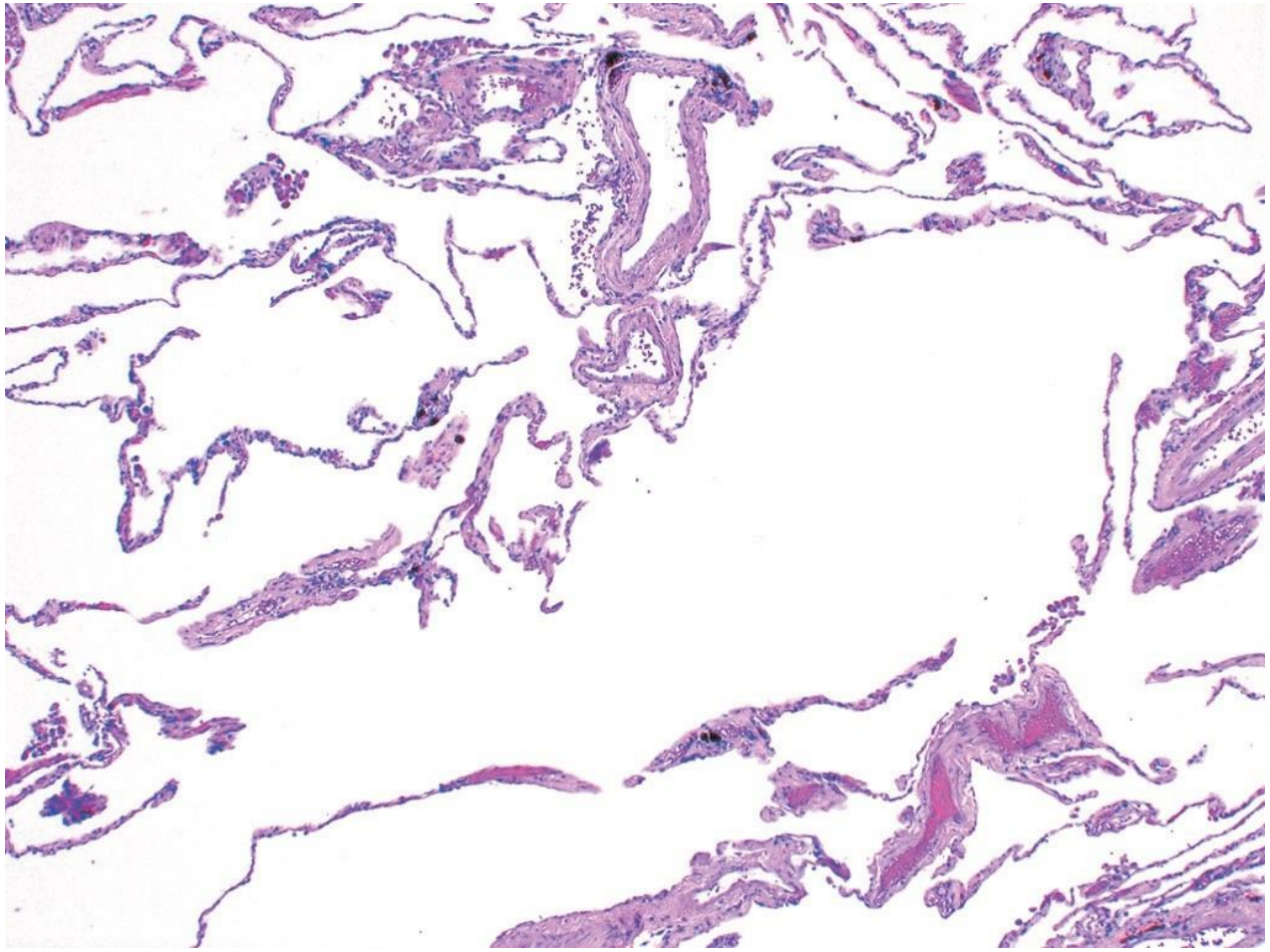
If we look at this figure, we will see that areas of acinus are dilated, while adjacent areas are not dilated. What is the possibility of this type of emphysema? It can be irregular, but irregular emphysema is not symptomatic, so it usually won't be a clinical case. It can be distal acinar or centriacinar emphysema because there is variation. You don't need to distinguish these two.



Panacinar emphysema involving the entire pulmonary lobule.

all spaces converted into cystically dilated spaces. This is the main difference between centriacinar or distal acinar emphysema and panacinar emphysema.

- **Microscopic examination of the lung:**
- abnormally large alveoli are separated by thin septa with only focal centriacinar fibrosis.

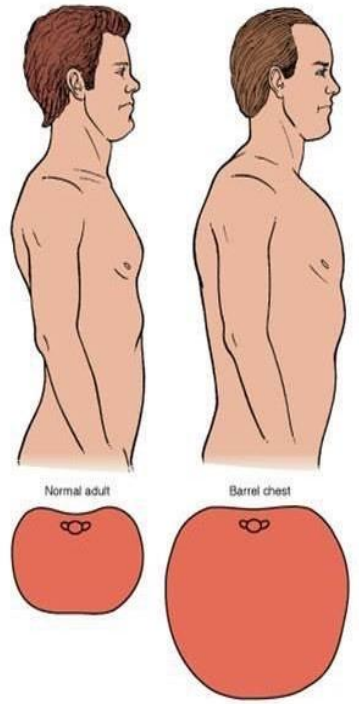


In terms of histology, there is enlargement of the alveolar spaces. Look at the central alveolar spaces, which are enlarged. There are breaks in the walls, and the walls are not thickened, although minimal and subtle fibrosis may be present, contributing to airflow obstruction

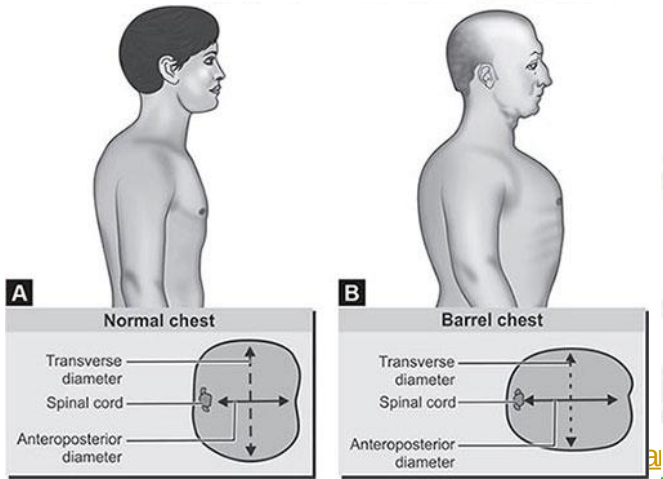
EMPHYSEMA, PRESENTATION:

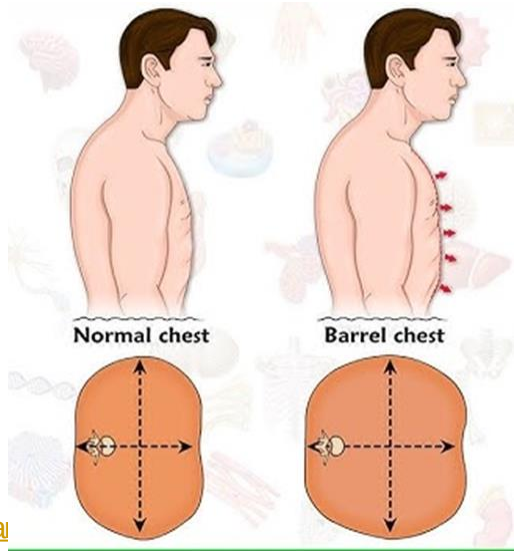
- **Symptoms do not appear until at least 1/3 of the functioning pulmonary parenchyma is damaged**
- **Dyspnea: appears first, beginning insidiously but progressing steadily. It's the most important symptom.** It begins gradually but increases steadily over time. This means the patient today will be better than six months or a year later, as dyspnea progresses continuously
- **Weight loss; common**
- **barrel-chested**

- The term “barrel-chested” refers to an increased anterior-posterior diameter of the chest due to air trapping and lung hyperinflation.
- Comparing a normal adult chest wall with a patient who has emphysema or another obstructive lung disease, the anterior-posterior diameter is visibly increased, giving this appearance.



Additional pics :





- **prolonged expiration,**

- **sitting forward in a hunched-over Position (look at this extra pic)**

- **breathes through pursed lips**
- **Hyperventilation**
- **adequate oxygenation of Hemoglobin especially at rest and prominent dyspnea → "pink puffers."**

The term "pink" refers to their well-oxygenated hemoglobin, as they are not cyanosed early in the disease. The term "puffer" reflects their effort to expel air, often through pursed lips, due to dyspnea.

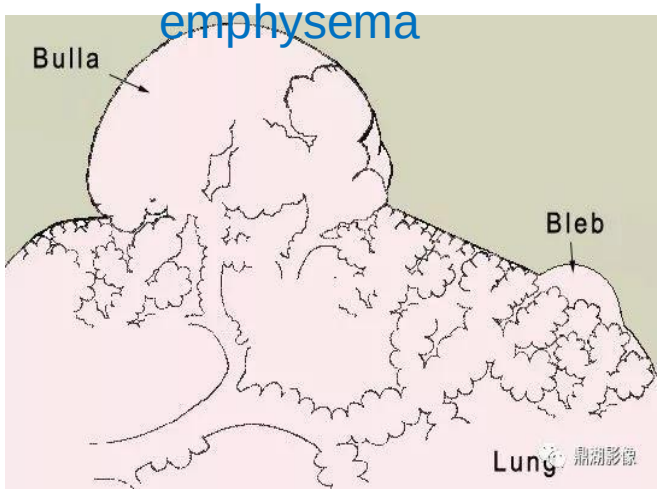
- **Cough and wheezing if Coexistent asthma & chronic bronchitis. there is an overlap between emphysema, chronic bronchitis, and asthma. If a patient has an element of chronic bronchitis, and we assume they are a smoker, they will likely have a cough with sputum. If there is an element of asthma, the patient will present with wheezing and coughing.**

Symptom	Cause
Prolonged expiration	• Because of the destruction of the walls and air trapping, the lungs are dilated, and the acini are filled with air. The patient feels short of breath and tired as a result. To compensate, the patient tries to get as much air as possible, leading to prolonged expiration This also causes the patient to use all the muscles in the chest wall to expel air.
Sitting forward in a hunched-over position	Often, the patient adopts a specific position, sitting at the edge of a chair, leaning forward, and pressing their chest to aid air expulsion. Additionally, they breathe through pursed lips, tightening their lips to improve airflow.
Breathing through pursed lips	
Hyperventilation	• This disease is associated with obstruction from the very beginning, so patients develop hyperventilation, with rapid, deep breaths to maintain oxygenation, especially early in the disease.
Adequate oxygenation of Hb	
“pink puffers.” 	The term “pink” refers to their well-oxygenated hemoglobin, as they are not cyanosed early in the disease. The term “puffer” reflects their effort to expel air, often through pursed lips, due to dyspnea.

OUTCOME:

- Decreased capillary bed area due to:
  - ✓ Destruction of alveolar walls
  - ✓ enlarged airspaces (bullae and blebs) in advanced disease causing Compression of the respiratory bronchioles and lung vasculature.
  - ✓ Tobacco smoke exposure also can induces Inflammatory changes in small airways
- Decreased capillary bed area→ hypoxia
- Hypoxia-induced pulmonary vascular spasm →gradual development of secondary pulmonary hypertension→ in 20-30% right-sided congestive heart failure (cor pulmonale).

Extra:
bullae and blebs Abnormal air-filled spaces in the lungs caused by alveolar wall destruction in emphysema



Additional Explanation for last point

🔴Why Hypoxia-Induced Pulmonary Vascular Spasm Leads to Cor Pulmonale❓

1.Hypoxia-Induced Pulmonary Vascular Spasm:

In response to hypoxia, pulmonary arteries undergo **vasoconstriction**. This is a physiological mechanism aimed at redirecting blood flow to better-ventilated areas of the lung to optimize oxygenation.

However, in emphysema, hypoxia is widespread, leading to **global pulmonary vasoconstriction**, which increases **pulmonary vascular resistance**

2.Gradual Development of Secondary Pulmonary Hypertension:

Persistent vasoconstriction causes chronic elevation in pulmonary artery pressure.

This increase in pressure strains the **right ventricle**, which must pump against higher resistance in the pulmonary circulation.

3.Right-Sided Congestive Heart Failure (Cor Pulmonale):

Over time, the right ventricle **hypertrophies** and eventually fails due to the sustained pressure overload.

The failing right ventricle leads to systemic venous congestion.

✔ **Hypoxia → Pulmonary vasoconstriction → Increased pulmonary vascular resistance → Pulmonary hypertension → Right ventricular hypertrophy → Right-sided heart failure (Cor Pulmonale).**

II. CHRONIC BRONCHITIS

Defined clinically as Persistent Chronic productive cough (minimal amount of clear sputum) for AT LEAST 3 consecutive months in AT LEAST 2 consecutive years in the absence of any other identifiable cause. For example, if a patient had a persistent cough with sputum for four months in 2020 and seven months in 2023, this would **not** meet the diagnostic criteria for chronic bronchitis..

- **90% cigarette smokers; air pollutants also contribute.**
- **chronic bronchitis is one end of the spectrum of COPD, with emphysema being the other.**
- Emphysema involves airway obstruction from day one, while chronic bronchitis does not initially present with obstruction. This distinction is critical to understanding their pathogenesis.

PATHOGENESIS

The primary factor in the genesis is exposure to irritating inhaled substances such as tobacco smoke (90%of pt) and dust from grain, cotton, and silica.

it primarily affects the **large airways**, including the trachea and main bronchi. Diagnosis is based on **clinical features**, and 90% of cases are associated with tobacco smoking, which is why it is commonly linked with **centriacinar emphysema**

- **hypersecretion of mucus:**
  - **The earliest feature of chronic bronchitis**
  - **beginning in the large airways.**
  - Why does this happen? It is due to goblet cell proliferation and thickening of the submucosal gland layer

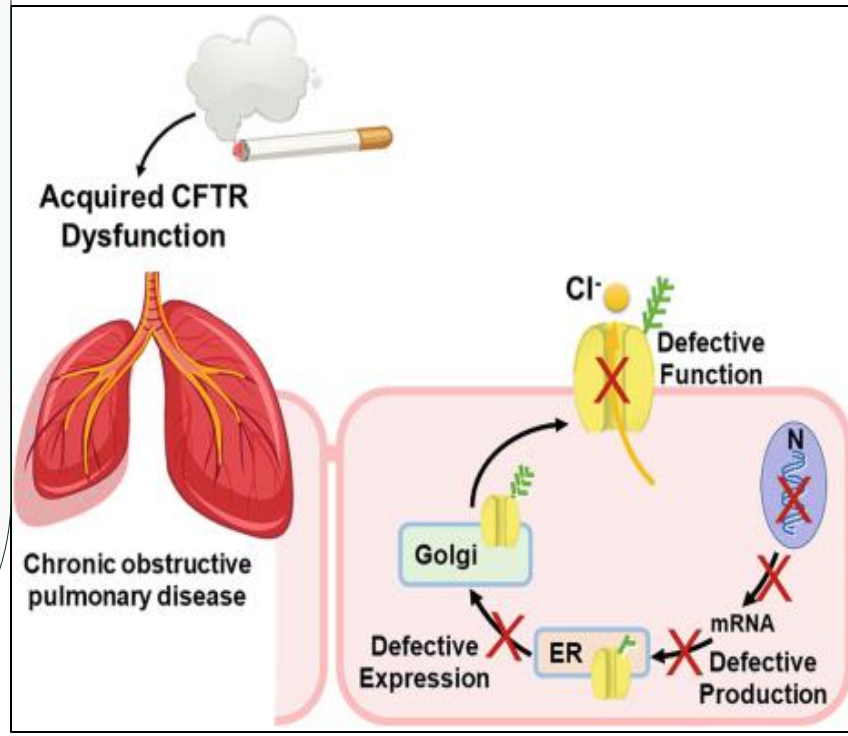
- **Acquired cystic fibrosis transmembrane conductance regulator (CFTR) dysfunction.**
  Exposure to noxious particles or substances, such as those in tobacco smoke, leads to acquired dysfunction of (CFTR). Which changes the quality of the mucus, making it sticky and thick, making it difficult for the patient to expel. Over time, the mucus accumulates and causes obstruction.
  - **smoking leads to acquired CFTR dysfunction**
  - **secretion of abnormal dehydrated mucus**
  - **increases the severity of chronic bronchitis.**

Inflammation. 1.Due to the Inhalants 2.No eosinophils , involves lymphocytes, macrophages, and neutrophils, but not eosinophils, as they are not a feature of chronic bronchitis.

Additional for ur better understanding

Acquired CFTR dysfunction in chronic bronchitis, particularly in **smokers**, arises from factors like tobacco smoke exposure and chronic inflammation. Tobacco smoke directly inhibits CFTR function by reducing its expression and activity on airway epithelial cells, while oxidative stress and inflammatory cytokines (e.g IL-1β, TNF-α) further impair its function. This dysfunction leads to **decreased chloride secretion** and water transport, resulting in dehydrated, thick mucus that is difficult to clear. Impaired mucociliary clearance promotes mucus plugging, airway obstruction, and recurrent infections, creating a cycle of inflammation and progressive lung damage.

The consequences of CFTR dysfunction include **reduced mucus clearance, bacterial colonization, and worsening airway obstruction**, resembling some features of cystic fibrosis. This dysfunction contributes to the chronic infections and progressive structural lung damage characteristic of chronic bronchitis. Interventions like smoking cessation and therapies targeting CFTR activity could help mitigate these effects and improve patient outcomes.



- Long-standing inflammation and fibrosis involving small airways (small bronchi and bronchioles, less than 2 to 3 mm in diameter) ➞ chronic airway obstruction.
- Infection:
  - ✓ Infection does not initiate chronic bronchitis but is probably significant in maintaining it
  - ✓ Produces acute exacerbations.

Over time, the chronic inflammation and accumulation of sticky mucus in the airways result in obstruction. Initially, the patient may be able to expel mucus from the large or upper airways, but as it accumulates in the small airways, it can cause mucus blocks that close these airways. This leads to further inflammation, fibrosis, and eventually chronic airway obstruction. In the **early** stages of chronic bronchitis, the process is still **reversible**, as there is no significant obstruction. However, over time, the nature of the mucus changes, it becomes sticky and accumulates in the small airways, such as the small bronchi and bronchioles. This leads to inflammation, fibrosis, and obstruction, which results in airflow limitation.

Again, The role of infections in chronic bronchitis is significant but not in initiating the disease. (important &past info)

Infections can induce acute exacerbations or prolong attacks in patients with chronic bronchitis. By themselves, infections do not cause chronic bronchitis, but they can worsen symptoms or trigger acute episodes.

- **In early stages airflow is not obstructed.**

- **airflow obstruction in chronic bronchitis** results from: **(important)**

1. Small airway disease

chronic bronchiolitis: results in mild airflow obstruction. Induced by mucus plugging of the bronchiolar lumen, inflammation, and bronchiolar wall fibrosis.

As we said, the mucus progressively accumulates in the small airways, making it harder for the patient to expel it. This is compounded by impaired ciliary function, which further promotes mucus accumulation, leading to obstruction, inflammation, and fibrosis.

2. Coexistent emphysema: The cause of **significant airflow obstruction. Chronic bronchitis alone typically results in mild airflow obstruction. If a patient presents with significant airflow obstruction before two years of chronic bronchitis symptoms, you should suspect coexisting emphysema.**

- **When chronic bronchitis persists for years:**

  - decline in lung function, leading to cor pulmonale
  - cause atypical metaplasia and dysplasia of the respiratory epithelium, providing a rich soil for cancerous transformation.

    ➞ Another risk for these patients, due to tobacco smoking as the primary etiology, is the development of squamous metaplasia and dysplasia of the bronchial lining, increasing the risk of **squamous cell carcinoma**.

- May coexist with hyper-responsive airways with intermittent bronchospasm and wheezing ➞ **asthmatic bronchitis**

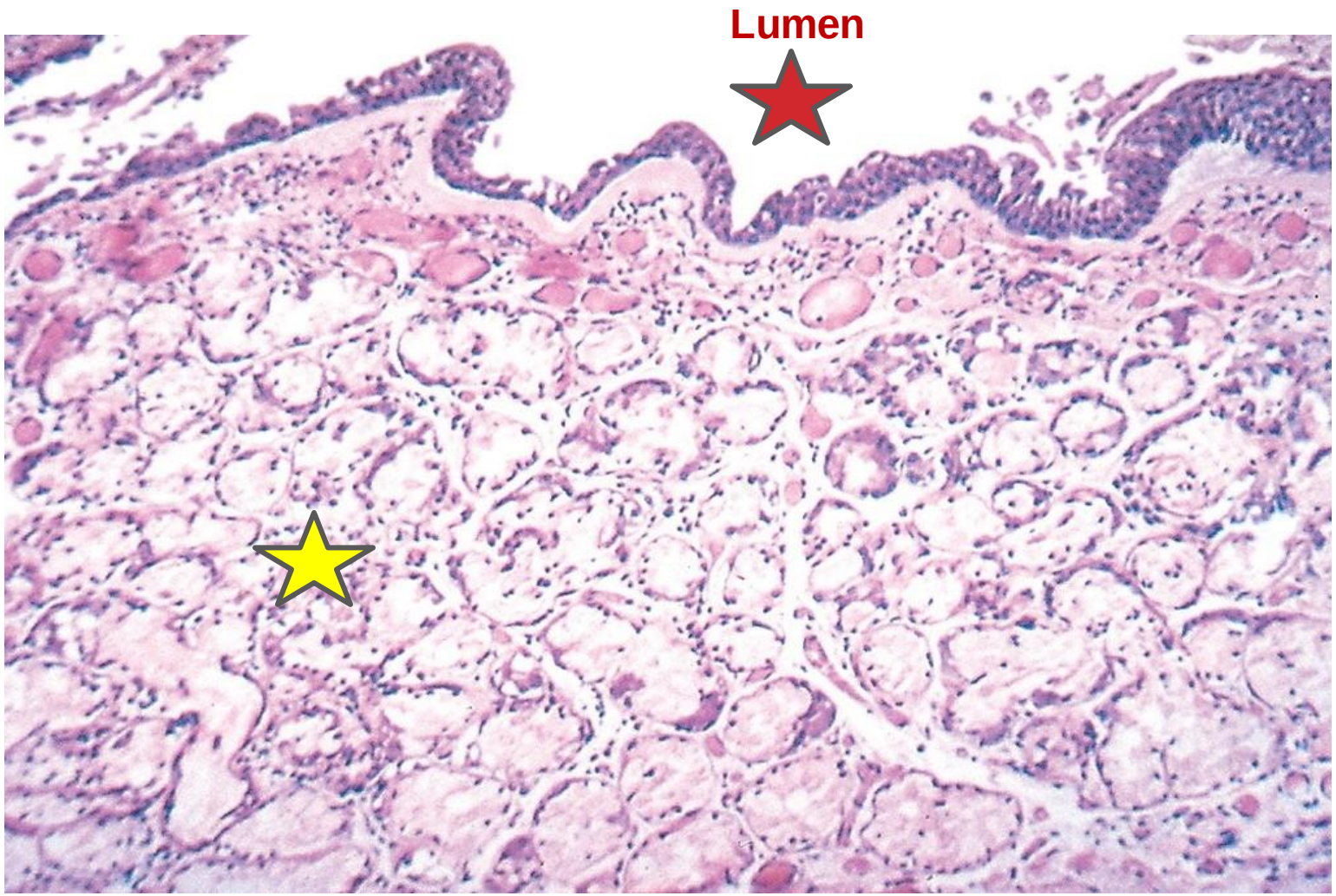
MORPHOLOGY

Macroscopic: Bronchoscopy reveals inflammation such as:

- **Mucosal lining is hyperemic and swollen**

- **Layers of mucinous or mucopurulent secretions**

- You can see a red star highlighting the lumen, and beneath this lumen, we have the epithelial lining. On the left side, there is pseudostratified ciliated columnar epithelium with many goblet cells, and on the right side, squamous metaplasia is present due to exposure to smoking-related substances.
- Just beneath the epithelial lining, the area is sprinkled with purple-blue dots, which represent lymphocytes. In chronic bronchitis, lymphocytes, macrophages, and neutrophils can be observed.
- The yellow star highlights the thickening of the submucosal glands, which are at least twice the normal size of submucosal glands.



Chronic bronchitis. The lumen of the bronchus is above. Note the marked thickening of the mucous gland layer (approximately twice-normal) and squamous metaplasia of lung epithelium.

MICROSCOPIC:

- **mild chronic inflammation of the airways (predominantly lymphocytes)**
  - **Hyperplasia of the mucus-secreting glands of the trachea and bronchi**
  - **squamous metaplasia and dysplasia of the bronchial epithelium**
- **Changes of emphysema often co-exist**

CLINICAL FEATURES:

- **<u>persistent cough with production of sparse sputum,</u>**
  The cough occurs daily, producing a minimal amount of clear sputum, which is not purulent or infected.
- For many years no respiratory functional impairment is present, but eventually dyspnea on exertion develops.
- chronic bronchitis and COPD patients show frequent exacerbations, rapid disease progression, and poorer outcomes than emphysema alone

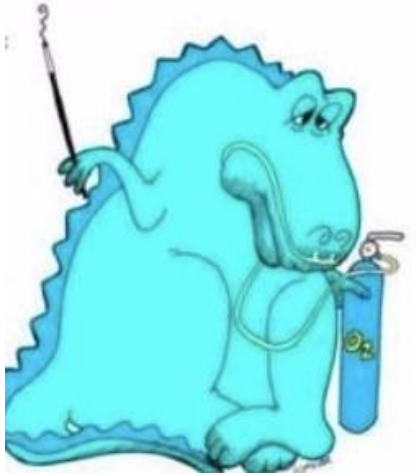
OUTCOME:

- Progressive disease is marked by the development of pulmonary hypertension, cardiac failure, recurrent infections due to mucus blocks in the airways; and ultimately respiratory failure

- Death may also result from further impairment of respiratory function due to superimposed acute infections.

- **Less dyspnea** compared to a patient with emphysema because, initially, there is no significant airflow obstruction. As a result, shortness of breath is not as prominent as it is in emphysema, where patients often suffer from excess air trapping.
- absence of increased respiratory drive → the **lungs retain carbon dioxide→ hypoxic and cyanotic.**
- For unclear reasons, patients with chronic bronchitis tend to be **obese**
- oxygenation of hemoglobin is less effective and inadequate compared to that in emphysema. This insufficient oxygenation leads to the characteristic **"blue bloater"**
- hence the designation **“blue bloaters”**

→ carbon dioxide retention, hypoxia, and cyanosis



Dr did not mention it

Table 15-4 Emphysema and Chronic Bronchitis

	Predominant Bronchitis	Predominant Emphysema
Age (yr)	40-45	50-75
Dyspnea	Mild; late	Severe; early
Cough	Early; copious sputum	Late; scanty sputum
Infections	Common	Occasional
Respiratory insufficiency	Repeated	Terminal
Cor pulmonale	Common	Rare; terminal
Airway resistance	Increased	Normal or slightly increased
Elastic recoil	Normal	Low
Chest radiograph	Prominent vessels; large heart	Hyperinflation; small heart
Appearance	Blue bloater	Pink puffer

Let's view some p.p questions . .

Which one of the following is a correct association concerning the pathogenesis of smoking-induced emphysema?

A) Destruction of distal acinus - centrilobular emphysema
B) Destruction of distal acinus - paraseptal emphysema
C) Destruction of entire acinus - panlobular emphysema
D) Destruction of proximal acinus - centrilobular emphysema
E) Destruction of proximal acinus - paraseptal emphysema

Answer: D

A 35-year-old gentleman he is a non smoker and barrel chested suffers from dyspnea and his lower zone of the lung has something , his brother has similar manifestations what of the following is most likely to be the reason for his disease
A) increase in proteases
B) lack of anti elstases
C) active pulmonary infection

Answer: B

U can revise general lec's info by this Extra table ➞

Category	Disease	Definition	Pathogenesis	Clinical Features	Morphology	Outcome
Obstructive Lung Diseases	1.Emphysema	Permanent (irreversible) enlargement of airspaces distal to terminal bronchioles with destruction of walls.	Small airway fibrosis, destruction of alveolar walls, decreased capillary bed area.	Dyspnea, weight loss, barrel chested, hyperventilation , prolonged expiration, “pink puffers.”	Voluminous lungs, abnormally large alveoli, enlarged airspaces (bullae and blebs).	Hypoxia, pulmonary hypertension, right-sided heart failure (cor pulmonale).
	2.Chronic Bronchitis	Persistent productive cough for at least 3 consecutive months over 2 years in the absence of any other identifiable cause.	Hypersecretion of mucus, acquired CFTR dysfunction, inflammation, small airway disease.	Persistent cough, sparse sputum, dyspnea on exertion, cyanosis (“blue bloaters”).	Hyperemic swollen mucosa, thickened mucus gland layer, squamous metaplasia.	Pulmonary hypertension, cardiac failure "cor pulmonale", respiratory failure, recurrent infections.

من إدلب العزّ حتّى غزّة الحرّة
حرٌ يورّثُ حُرًّا بعدهُ ثأرًا
هذه الحياةُ ممرٌّ ، لا لنحيا بها
إنّا نموت لنحيا بعدها عمرًا

VERSIONS	SLIDE #	BEFORE CORRECTION	AFTER CORRECTION
V1→ V2			
V2→V3			

امسح الرمز و شاركنا بأفكارك لتحسين أدائنا !!