Introduction To COPD

COLOR INDEX:

MAIN TEXT (BLACK)

FEMALE SLIDES (PINK)

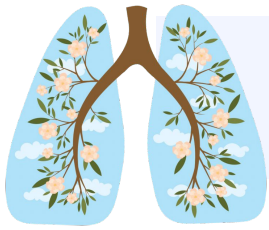
MALE SLIDES (BLUE)

IMPORTANT (RED)

DR'S NOTE (GREEN)

EXTRA INFO (GREY)

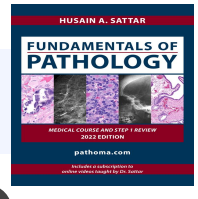
Editing file:



Objectives

- Know the major obstructive disorders and compare the major clinical and functional differences between them
- Aware that the symptom common to all these disorders is “dyspnea” (difficulty in breathing), but each have their own clinical and anatomical characteristics.
- Define emphysema and the following forms of emphysema: panacinar, emphysema, centriacinar, emphysema.
- Define chronic bronchitis, describe it’s pathogenesis and the morphological changes. Describe the mechanism of airway obstruction in a patient with chronic bronchitis.
- Define bronchiectasis, it’s causes , presentation, morphology, and significant.

If you want to read the lecture from Pathoma click on book



If you want videos of Pathoma click here

Obstructive Lung Diseases

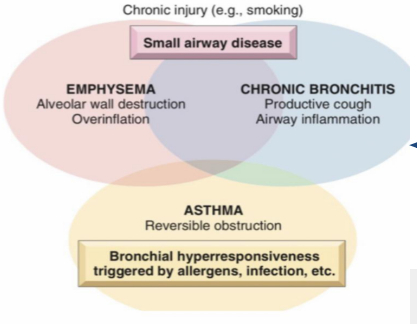
helpful video

Definition

Obstructive(airway) disease is characterized by an **increase in resistance to air flow** caused by partial or complete obstruction at any level. Each disease have distinct clinical and anatomic, but overlaps between emphysema, chronic bronchitis, and asthma are common.

Obstructive lung diseases

- Bronchial Asthma
- Bronchiectasis
- Chronic obstructive pulmonary disease (COPD)
  - Chronic bronchitis
  - Emphysema



-The anatomic distribution of these disorders also is somewhat different:

•chronic bronchitis initially involves the large airways

•emphysema affects the acinus.

Emphysema and chronic bronchitis often are grouped together under the rubric of chronic obstructive pulmonary disease (COPD).

Remember!

•Chronic obstruction of lung airflow that interferes with normal breathing and not fully reversible.

•Traditionally, regarded as a combination o smoking related lung disease: emphyseme, chronic bronchitis.

•Pulmonary function tests:

1. **Reduced FEV1**
2. **Normal or near-normal FVC**
3. **FEV/FVC ratio is reduced**

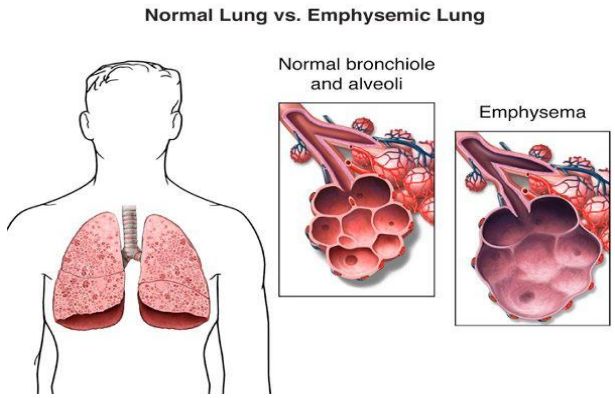
Comes In MCQ As a hint

Emphysema

helpful video

Definition

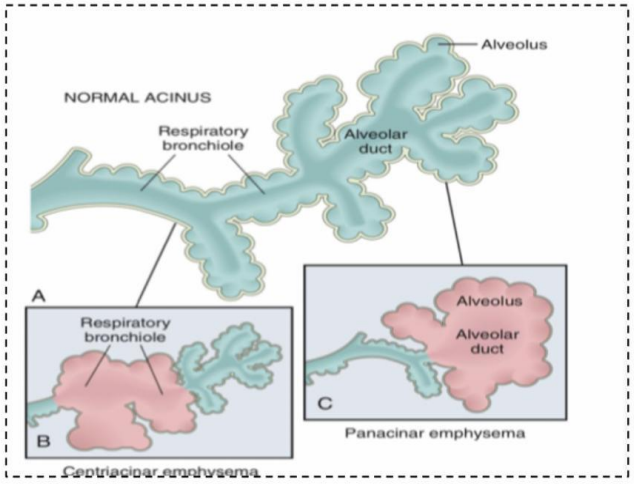
Is the abnormal **permanent enlargement** of air spaces distal to the terminal bronchioles accompanied by **destruction of their walls** without obvious fibrosis.



- Only the Centriacinar and panacinar types cause significant airway obstruction

Types

- Centriacinar
- Distal acinar/paraseptal
- Panacinar
- Irregular



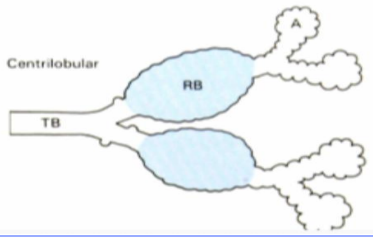
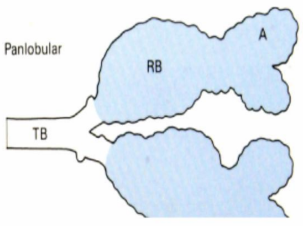
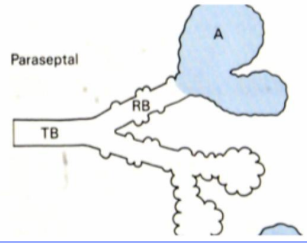
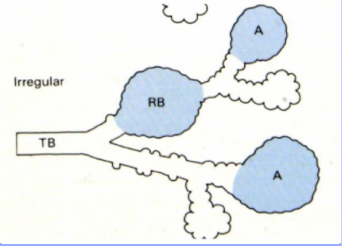
A Diagram of normal structure of the acinus, the fundamental unit of the lung

B Centriacinar emphysema with dilation that initially affects the respiratory bronchioles

C Panacinar emphysema with initial distention of all the peripheral structures (i.e., the alveolus and the alveolar duct

Soooooooo Important!

Classification of emphysema

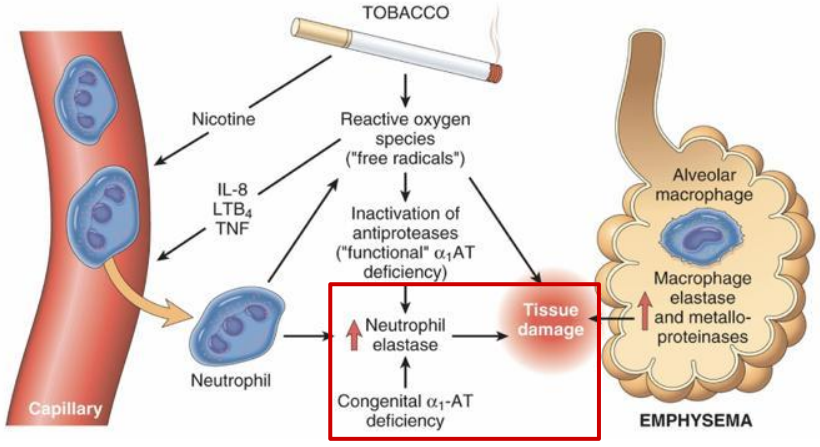
Whatever's in gray in the table is from robbins	Centriacinar	Panacinar	Distal acinar/parasaptal	Irregular
Etiology	Cigarette smoking	α-1 anti-trypsin Deficiency	Unknown but most often in young adults who present with spontaneous pneumothorax	
Location	Abnormal dilation of respiratory bronchioles and alveolar ducts with normal alveolar sacs	abnormal dilation of the whole acinus	Proximal portion of the acinus is normal, but the distal part is primarily involved	Acinus irregularly involved
Features	More Common and severe in **upper lobes**	Common in **lower zones**	More severe in the **upper half of the lungs**	May be most common form of emphysema
Notes	Often associated with **chronic bronchitis** 20 times more than panacinar	**Entire Acinus means** (from the terminal bronchiole to the terminal blind alveolus)	More adjacent to the **pleura,** along the lobular C.T septa. Occur adjacent to areas of **fibrosis**, scarring or atelectasis spontaneous pneumothorax in young adults	**Associated with scarring** **Resulting from healed inflammatory diseases**
Figures				

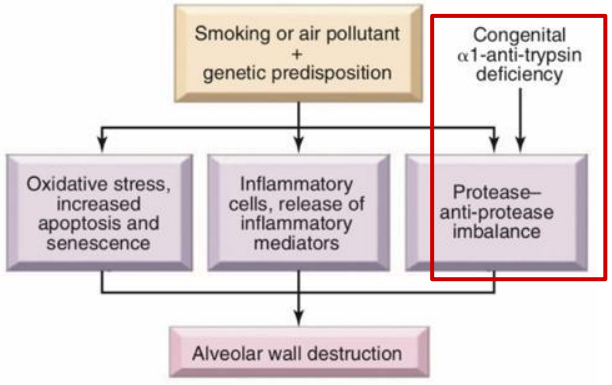
Emphysema

Thx to 443 🐐

Btw it’s extra slide to understand only pics are in the slide focus on red squares

Pathogenesis





1. Tobacco substances undergo phagocytosis by macrophages
2. Macrophages release cytokines that recruits neutrophils & reactive O2 species
3. Neutrophils release protease elastase: break down elastin. *note that elastin help in increase elasticity of the lungs
4. Lose the ability to recoil and increase lung compliance which cause air trapping

Smoking tobacco

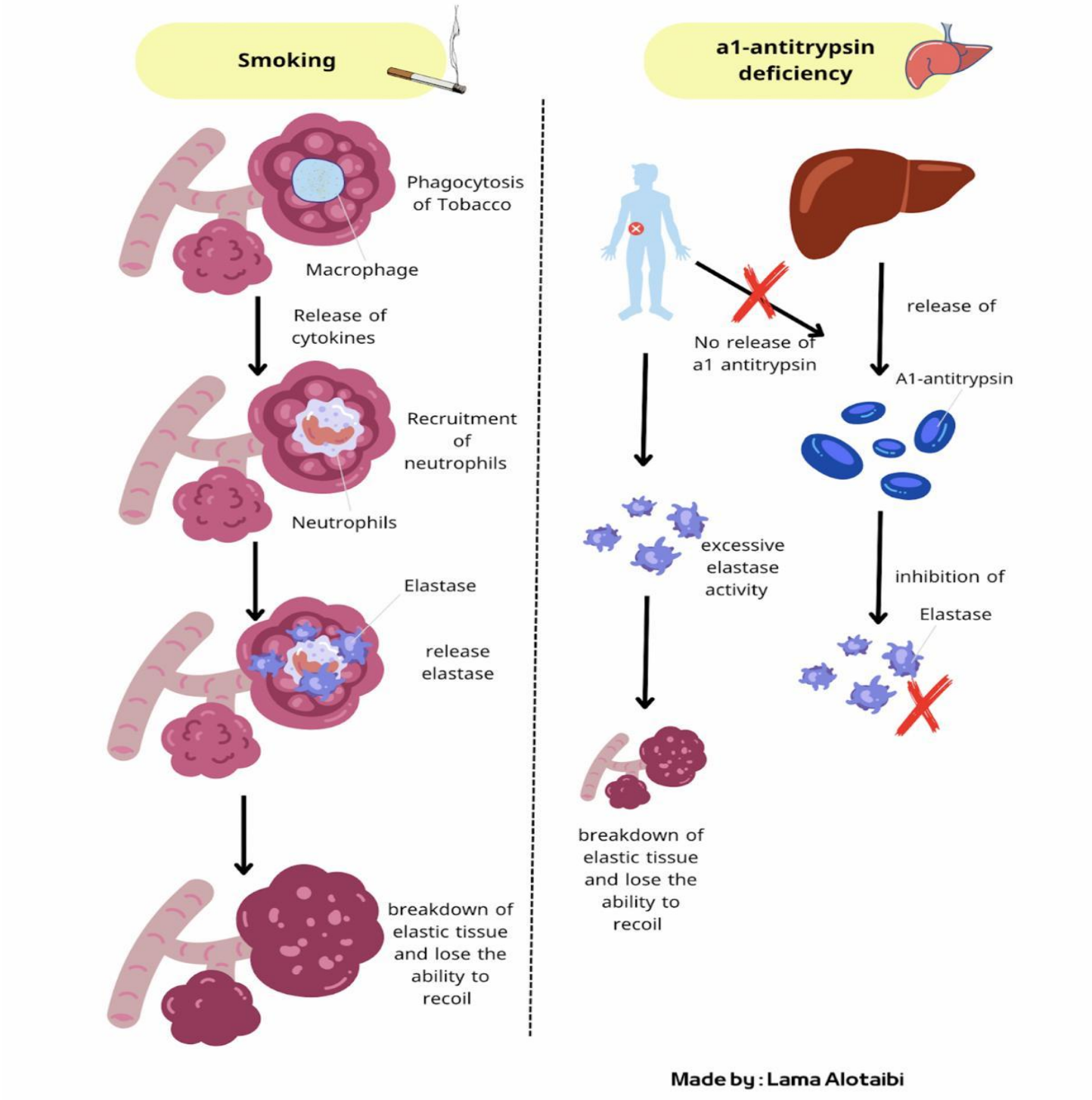
1. Liver release a1-antitrypsin which inhibits elastase enzymes
2. In people with a1-antitrypsin deficiency there is no inhibitions of elastase enzyme
3. Which cause excessive breakdown of elastic tissue and loss of elasticity
4. Loss of pressure cause air trapping with time the acinus become more expanded and lose the alveolar septa

A1- antitrypsin deficiency

Emphysema

Pathogenesis

Focus on elastase and a1 anti-trypsin
Thx 443 🐐

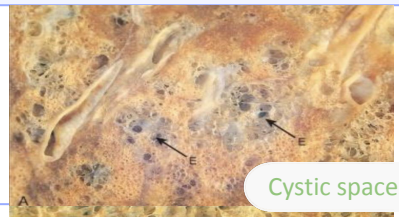
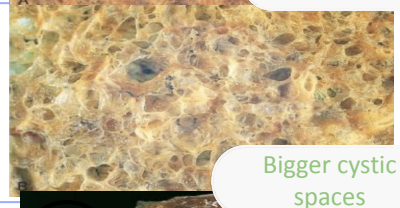
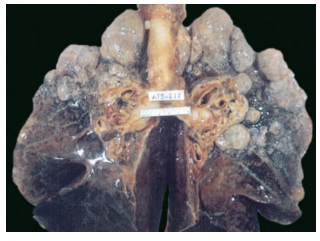
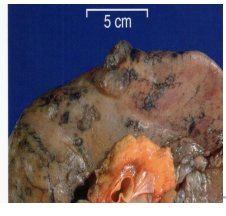
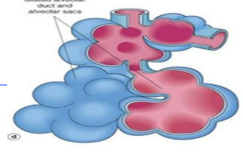
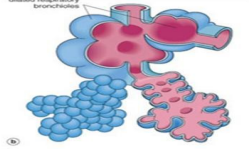
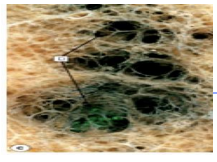


Made by : Lama Alotaibi

Emphysema

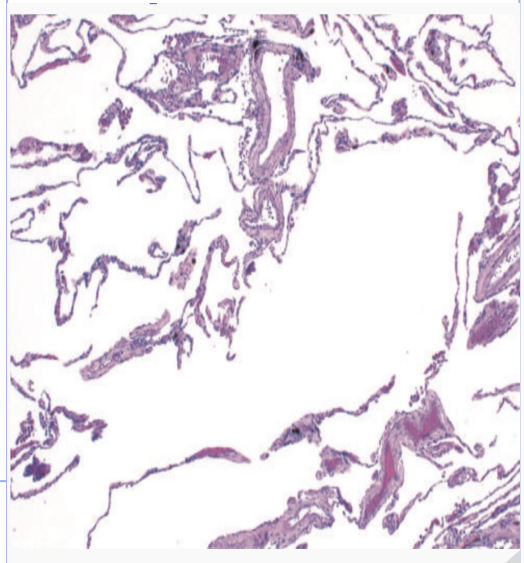
Morphology

سوو زوم للصور

	Description	Pictures
Gross	Centriacinar: Some parts are normal and some parts are dilated	 Cystic spaces
	Panacinar: pale, (because of air) voluminous lungs	 Bigger cystic spaces
	Bullous emphysema with large subpleural bullae (upper left)	 Bullous
	Large apical blebs or bullae are more characteristic of irregular emphysema secondary to scarring and if distal acinar emphysema	 Scarring & fibrosis
	Distal acinar/paraseptal: forming multiple cyst-like structures with spontaneous pneumothorax	 
		  

Emphysema

Morphology

	Description	Pictures
Microscopic	Thinning, dilated and destroyed alveolar walls (septa) with coalescence (fusion) of air spaces without fibrosis(if alveoli is associated with fibrosis we think of restrictive not COPD	
	There is no inflammation but maybe with smoking there will be some brown macrophages	

الalveoli تجي على شكل اكياس صغار بينهم جدار بسيط يمر فيه الدم ويصير تبادل الأكسجين لكن مع emphysema يختفي الجدار ويصير بدال أكياس صغار جنب بعض بيصيرون كيس كبير واحد أحيانا يسمونها كوصف (shopping bag) منطقيا بيقل عندنا الsurface area لأن صار الكيس بس blood supply واحد بدال اعداد كثيرة ف الexchange بيقل وبتصير مشاكل

Emphysema

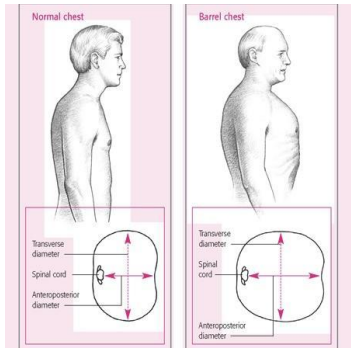
Clinical presentation of emphysema

Dyspnea usually is the first symptom; insidiously but is steadily progressive.

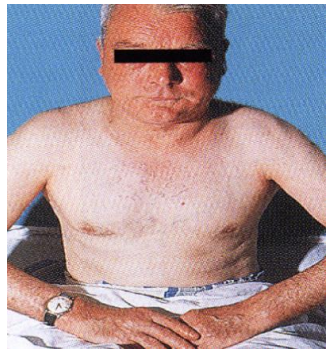
Cough and wheezing in patients with underlying chronic bronchitis or chronic asthmatic bronchitis.

Weight loss is common and may be severe enough to suggest an occult malignant tumor

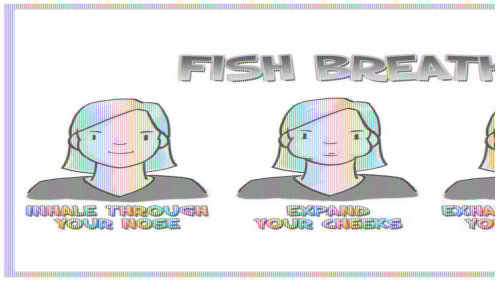
Barrel-shaped chest in a patient with Emphysema as a result from:
•Air-trapping with inflammatory changes.
•Hypersecretion of viscid contraction in The small airways.
Note that associated in drawing of the Intercostal muscles. Similar changes are seen in patients with chronic bronchitis and asthma



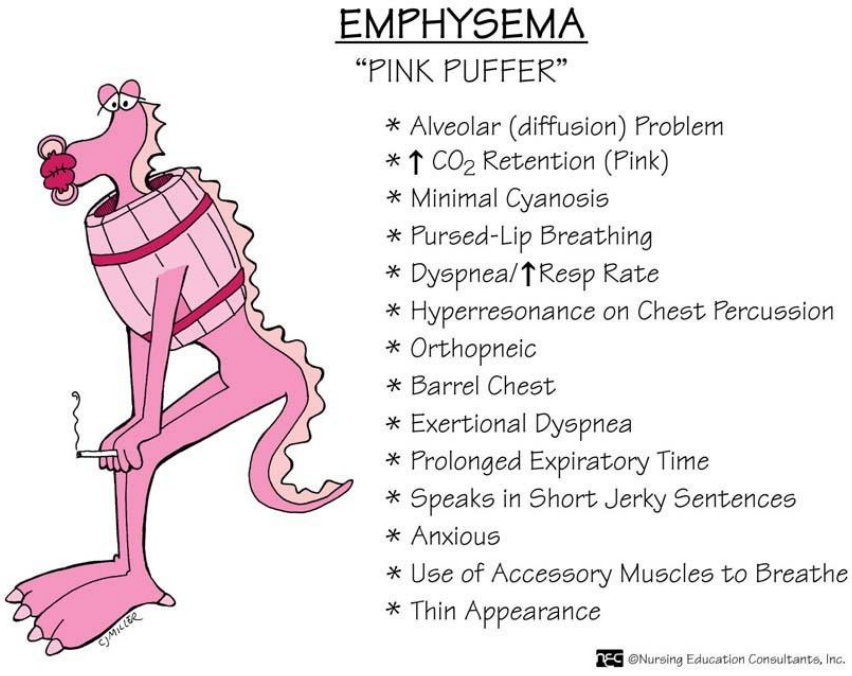
Fish mouthing breathing:
•Pursed lip expiration is a common maneuver adopted by patients with COPD.
•The patient starts to breathe out closed or nearly closed lips to keep the intrabronchial pressure high and prevent collapse of the bronchial wall and expiratory obstruction. Later in expiration the lips are blown forward and open with a grunt



الشهيق طبيعي والزفير مو طبيعي لان الBronchiole خسرت الelasticity فالهواء وهو طالع بيقفلها لكن لما يسوي طريقة فم السمكة بيوازن الضغط الهوائي الداخل والطالع فهذا الشي بيخلي الbronchiole ما تقفل

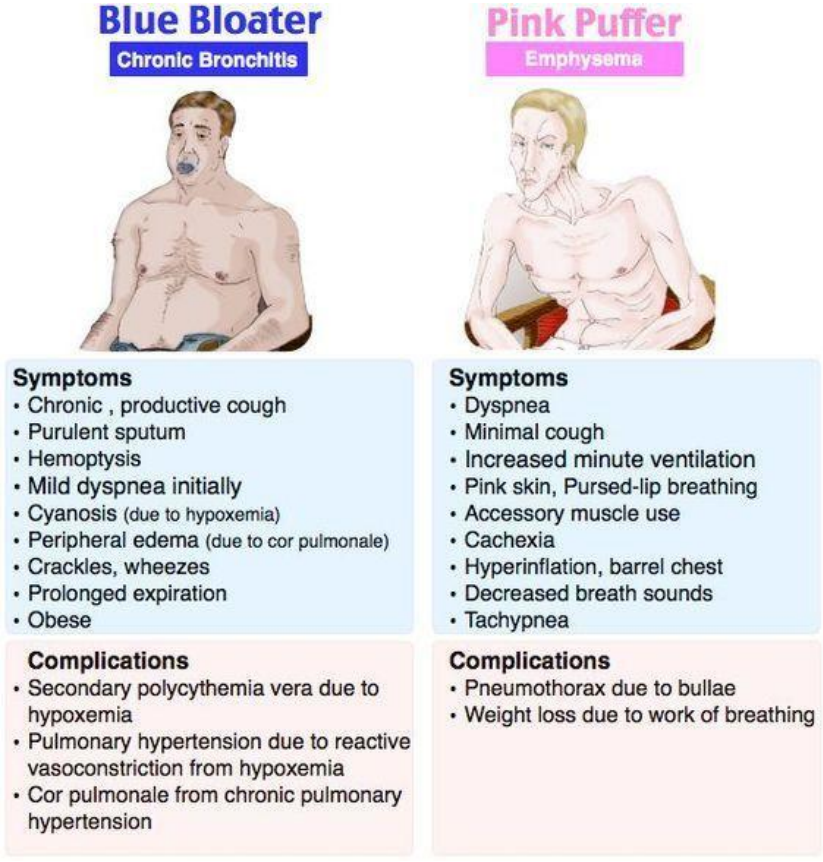


Emphysema



Pink puffer

Because of prominent Dyspnea and adequate oxygenation of hemoglobin



Emphysema

Conditions Related to Emphysema

Compensatory emphysema

The dilation of residual alveoli in response to loss of lung substance elsewhere, occurs after surgical removal of a diseased lung or lobe.

Obstructive overinflation

Refers to expansion of the lung due to air trapping. A common cause is subtotal obstruction of an airway by a tumor or foreign object. Can be life-threatening if expansion of the affected portion produces compression of the remaining normal lung.

Bullous emphysema

Refers to any form of emphysema that produces large subpleural blebs or bullae (spaces >1cm).

Mediastinal (interstitial) emphysema

Caused by entry of air into the interstitium of the lung, from where it may track to the mediastinum and sometimes the subcutaneous tissue.

- It may occur spontaneously if a sudden increase in intra alveolar pressure (as with vomiting or violent coughing) produces alveolar rupture
- Can develop in children with whooping cough
- Can occur in patients who have partial bronchiolar obstruction or in individuals with perforating injury. (E.g., fractured rib)

Subcutaneous emphysema

When the interstitial air gets into the subcutaneous tissue with marked swelling of the head and neck and crackling crepitation (abnormal sound during auscultation) over the chest

Emphysema

Pulmonary function test

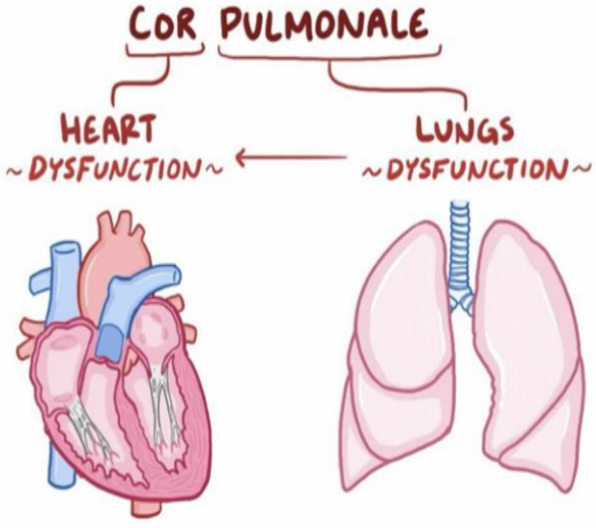
1	Reduced FEV1
2	Normal or near-normal FVC
3	FEV1/FVC ratio is reduced

Hyperinflated lung will push the spleen and the liver down , giving the false impression of splenomegaly and hepatomegaly , how to tell the difference? By Percussion of upper border of liver and spleen , if the upper border is normal this is hepatomegaly - splenomegaly if both the lower and the upper border are depressed this is emphysema

Complication

- Pneumothorax (comes with bullous)
- Death from emphysema is related to:

1.Pulmonary failure with respiratory acidosis, hypoxia, and coma.
2.Secondary pulmonary hypertension (arising from both hypoxia-induced pulmonary vascular spasm and loss of pulmonary capillary surface area from alveolar destruction).
3.Right-sided heart failure (Cor pulmonale).



Emphysema

Summary

Emphysema is a chronic obstructive airway disease characterized by enlargement of air spaces distal to terminal bronchioles.

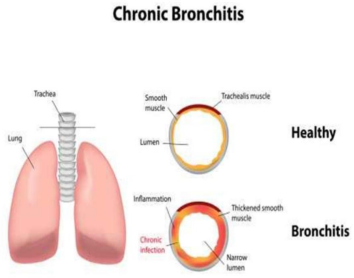
- Subtypes include centriacinar (most common: smoking- related), panacinar (seen in α1 -anti-trypsin deficiency), distal acinar, and irregular.
- Smoking and inhaled pollutants cause ongoing accumulation of inflammatory cells,
- which are the source of proteases such as elastases that irreversibly damage alveolar walls.
- Patients with uncomplicated emphysema present with increased chest volumes, dyspnea, and relatively normal blood oxygenation at rest ("pink puffers").
- Most patients with emphysema also have signs and symptoms of concurrent chronic bronchitis, since cigarette smoking is a risk factor for both.

Chronic Bronchitis

click here

Definition

Is diagnosed on clinical grounds: it is defined by the presence of a persistent productive cough for at least 3 consecutive months in at least 2 consecutive years Med43: المريض خلال آخر سنتين كان يكح 3 شهور متتالية



Common among cigarette smokers ; air pollutants also contribute

Pathogenesis

- Hypertrophy of mucous secreting cells in trachea and bronchi Increase goblet cells in bronchiole
- Chronic inflammation; macrophages, neutrophils.
- Secretion of IL-13 by T lymphocytes
- Chronic inflammation with increased mucus formation leads to bronchiolar obstruction along with fibrosis.(in the bronchi)
- As result; mild or significant airflow obstruction, which almost always complicated by emphysema.

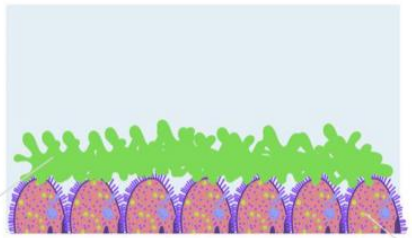
Chronic Bronchitis

I recommend u know this slide
Thx 443

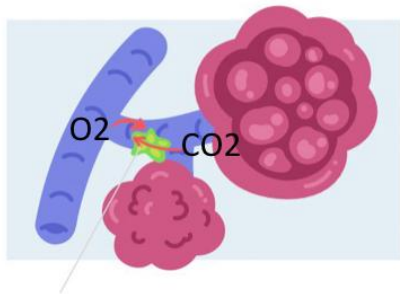
Pathogenesis

Increase mucus production because of inflammation and releasing of cytokines & hypertrophy + hyperplasia of mucin glands and goblet cells causing narrowing of the airways

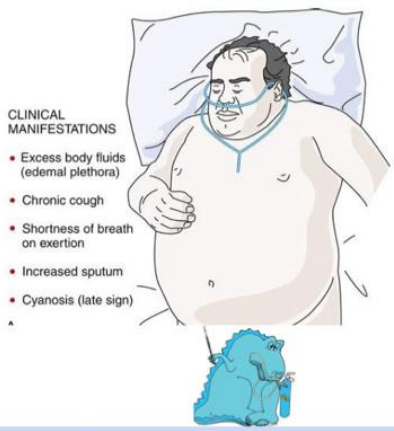
Ciliary dysfunction causes accumulation of mucus (mucus plug)

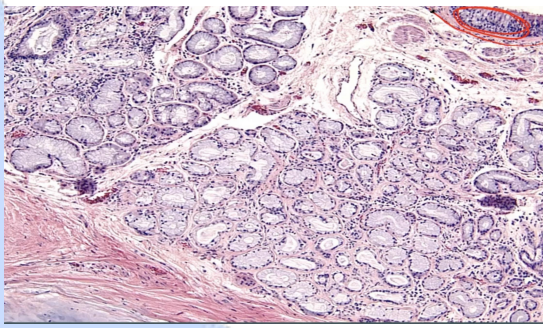


Mucus plug cause less O2 in and less CO2 out (air trapping)



This leads to V/Q mismatch cause hypoxia, hypercapnia and cyanosis





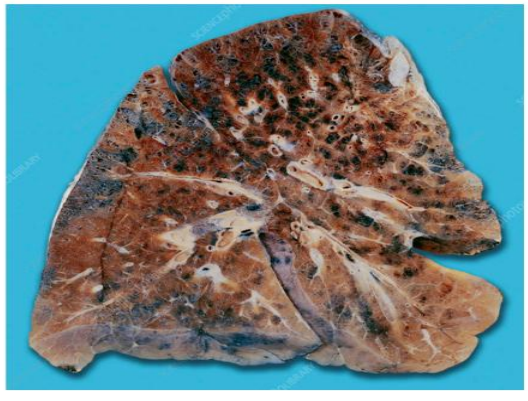
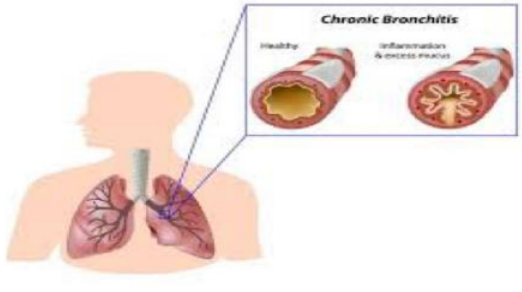
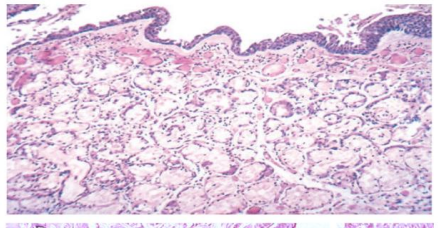
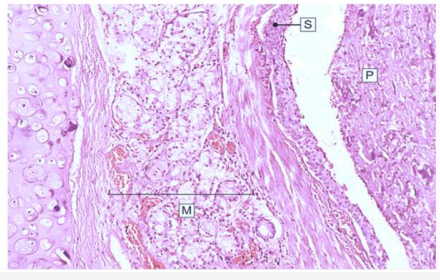
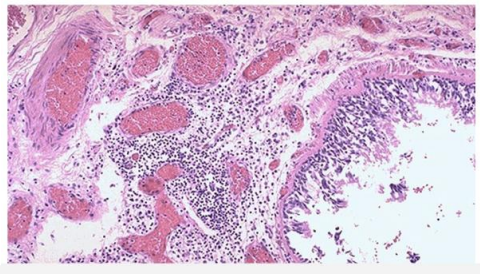
الطبقة حقت الglands مرة كبيرة
فهو شي غير طبيعي أكيد والدائرة
هي الrespiratory
epitheluim فشوفوا كيف صار
thick layer of mucous
glands فراح تنتج كمية كبيرة
وتسبب عندنا الmucous plug

Blue bloater

Made by: Lama Alotaibi
from Med443

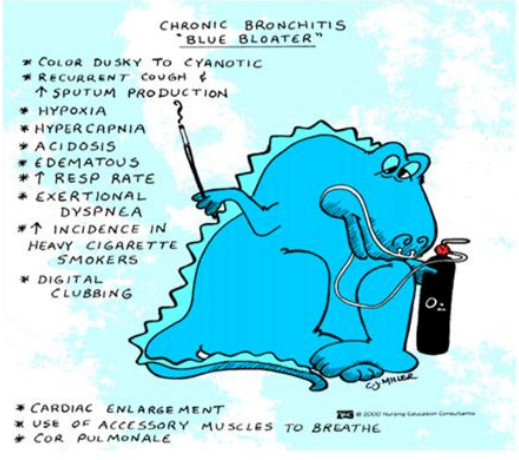
Chronic Bronchitis

Morphology

	Gross	Microscopic
Description	• Bronchial wall shows; Redness, swelling, covered by mucopurulent Secretion. • Thickening of the wall Mild narrowing of bronchial lumen	**Histologic examination:** 1.Chronic inflammation. 2.Enlargement of mucus secreting glands:The magnitude of the increase in size is assessed by the ratio of the thickness of the submucosal gland layer to that of the bronchial wall (Reid index-normally 0.4). 3. Goblet cell metaplasia. 4. Bronchiolar wall fibrosis.
Pictures	 	  The main abnormality is secretion of abnormal amounts of mucus, causing plugging of the airway lumen  Chronic inflammation in the bronchial wall

Clinical Presentation & Complications

1. Cough and sputum production persist indefinitely without ventilatory dysfunction.
2. Outflow obstruction marked by hypercapnia, hypoxemia, and cyanosis.
3. Patients with chronic bronchitis and COPD have frequent exacerbations, more rapid disease progression, and poorer outcomes than those with emphysema alone.
4. Death due to further impairment of respiratory functions after superimposed acute bacterial infections.



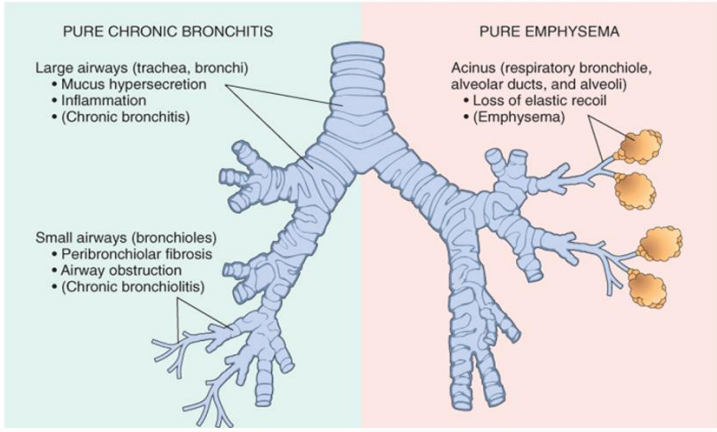
emphysema: Pink puffer
Chronic bronchitis: blue bloater

Blue bloater

Patient with emphysema who also has pronounced chronic bronchitis and a history of recurrent infections. Dyspnea usually is less prominent, and in the absence of increased respiratory drive the patient retains carbon dioxide, becoming hypoxic and often cyanotic

Chronic Bronchitis summary

- Chronic bronchitis is defined as persistent productive cough for at least 3 consecutive months in at least 2 consecutive years.
- Cigarette smoking is the most important underlying risk factor; air pollutants also contribute.
- Chronic airway obstruction largely results from small airway disease (chronic bronchiolitis) and coexistent emphysema.
- Histologic examination demonstrates enlargement of mucus- secreting glands, goblet cell metaplasia, and bronchiolar wall fibrosis.



	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*

Bronchiectasis

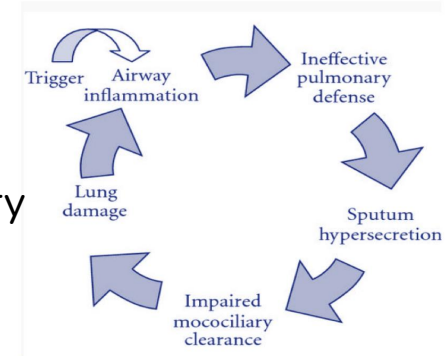
click here

Definition

Permanent dilation of bronchi and bronchioles caused by destruction of smooth muscle and the supporting elastic tissue.

it typically results from or is associated with chronic necrotizing infections

Not a primary disorder, as it always occurs secondary to persistent infection or obstruction caused by a variety of conditions.



Causes

Causes

Congenital = منذ الولادة

Congenital or hereditary conditions	Postinfectious conditions	Bronchial obstruction	Other conditions
cystic fibrosis, intralobar sequestration	bacteria, viruses and fungi	Tumors, forging bodies	RA, SLE

Most common causes

SLE= Systemic Lupus Erythematosus
RA = Rheumatoid arthritis

Pathogenesis

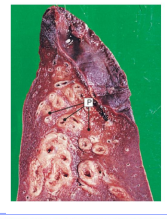
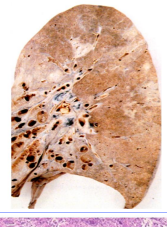
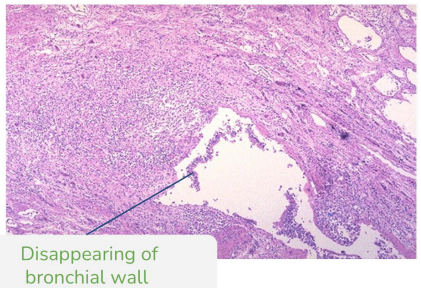
atelectasis= collapse=انكماش

Obstruction & infections are the major cause; After obstruction→ air is reabsorbed from the airways distal to the obstruction→ atelectasis. Accumulation Of intraluminal secretions → dilatation of the airways AND Bronchial wall inflammation.

If the obstruction persists during the period of growth, these changes become irreversible

When superadded suppurative necrotizing type of infection; Extensive bronchial and bronchiolar wall damage (destruction with loss of elastic and smooth muscle fibers).

Morphology

	Description	Pictures
Gross	• Usually, lower lobes or distal. • Dilatation about Four times the normal diameter • Mucosa: congestion, ulcers.	
	1-Dilatation of bronchi with destruction of bronchial walls 2-A lower lobe of lung surgically resected for bronchiectasis	2   1
Microscopic	• Acute and chronic inflammation. • Destruction with abscesses. • Epithelial ulcerations. • +/- squamous metaplasia. • Glandular atrophy • Fibrosis of bronchiolar wall.	 Disappearing of bronchial wall

Clinical Presentation

Severe persistent cough with sputum (mucopurulent sputum) sometime with blood.

Dyspnea

Hypoxia, hypercapnia, cyanosis.

Clubbing of fingers.

Complication

1

Significant obstructive ventilatory defects, with hypoxemia,hypercapnia, pulmonary hypertension, and cor pulmonale.

2

Lung abscess.
Most common complication

3

Brain abscess.

4

Amyloidosis
Accumulation of abnormal proteins

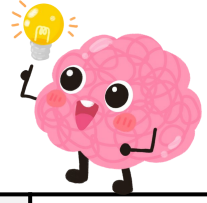
Summary +

Extra sources to understand

Clinical Entity	Anatomic Site	Major Pathologic Changes	Etiology	Signs/Symptoms
Chronic bronchitis	Bronchus	Mucous gland hypertrophy and hyperplasia, hypersecretion	Tobacco smoke, air pollutants	Cough, sputum production
Bronchiectasis	Bronchus	Airway dilation and scarring	Persistent or severe infections	Cough, purulent sputum, fever
Asthma	Bronchus	Smooth muscle hypertrophy and hyperplasia, excessive mucus, inflammation	Immunologic or undefined causes	Episodic wheezing, cough, dyspnea
Emphysema	Acinus	Air space enlargement, wall destruction	Tobacco smoke	Dyspnea

Robbins COPD Click here

Flash cards for this lecture that might help you right here



KEYWORDS

Chronic bronchitis	Cigarette smoking Persistent productive cough (with mucous) for 3 months to 2 years Hypertrophy of mucous secreting cells goblet cell metaplasia Fibrosis of bronchiolar wall Blue bloaters
Emphysema	Dilation of air spaces with destruction of alveolar walls Neutrophil elastase: cause elastic tissue damage **Centriacinar**: cigarette smoking **Panlobular (panacinar)**: a1 anti-trypsin deficiency Pink puffer
Bronchiectasis	Destruction of smooth muscle and elastic tissue **Location** : Bronchiole and bronchi Dilation of bronchial wall Not primary disorder Mucopurulent sputum (can be with blood)

Answers:
1-B 2-D 3-A 4-B 5-A

MCQ

1- Which of the following is complication to chronic bronchitis?			
A) Cystic fibrosis	B) Cor pulmonale	C) lung cancer	D) Pneumothorax
2- Which of the following is associated with formation of bullae?			
A) Parasepta	B) Panacinar	C) Centriacinar	D) irregular
3- Ahmad 21 years old came to hospital, because he had productive cough for last 8 months, also he is a smoker. We did sputum culture, we found macrophages full of carbon, and a lot of neutrophil. What is the diagnosis?			
A) Chronic Bronchitis	B) Asthma	C) Emphysema	D) Bronchiectasis
4-The main enzyme that increases in the pathophysiology of emphysema and responsible for the destruction of the elastic fibers is			
A) a1 antitrypsin deficiency	B) Elastase	C) Both A&B	D) Carbonic anhydrase
5- Which one is characterized with a PINK buffer?			
A) Emphysema	B) Asthma	C) Bronchiectasis	D) Chronic Bronchitis

Answers:
1-B 2-D 3-C

Cases

1- A 55-year-old man was admitted to the hospital with a chief complaint of increasing shortness of breath over the past several years. The patient was a heavy smoker over the past 40 years. Physical examination reveals cyanosis, elevated jugular venous pressure, and peripheral edema. A high-resolution CT scan shows bullae over both lungs. Chronic intra-alveolar exposure to which of the following proteins is most likely associated with the pathogenesis of chronic obstructive pulmonary disease in this patient?			
A) α1-Antitrypsin	B) Elastase	C) Collagenase	D) Alkaline phosphatase
2- A 48-year-old man with a history of heavy smoking presents with a 3-year history of persistent cough and frequent upper respiratory infections, associated with sputum production. Physical examination reveals prominent expiratory wheezes and peripheral edema. Analysis of arterial blood gases reveals hypoxia and CO 2 retention. Which of the following is the appropriate diagnosis?			
A) Atelectasis	B) Usual interstitial pneumonia	C) Hypersensitivity pneumonitis	D) Chronic obstructive pulmonary disease
3-A 34-year-old man who used to smoke two packs of cigarettes per day for 14 years. For the past 2 years, he has had a chronic productive cough that usually lasts for 3.5 month for each year. The patient came to the clinic complaining from shortness of of breath and tightness of the chest. The Clinical presentation showed audible wheezing. a biopsy that has been taken showed enlargement of mucous secreting glands causing plugging of the airway lumen (P). Which of the following pathologic conditions is most likely responsible for his clinical condition?			
A) Emphysema	B) bronchiectasis	C) chronic bronchitis	D) Asthma

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