

OBSTRUCTIVE LUNG DISEASES

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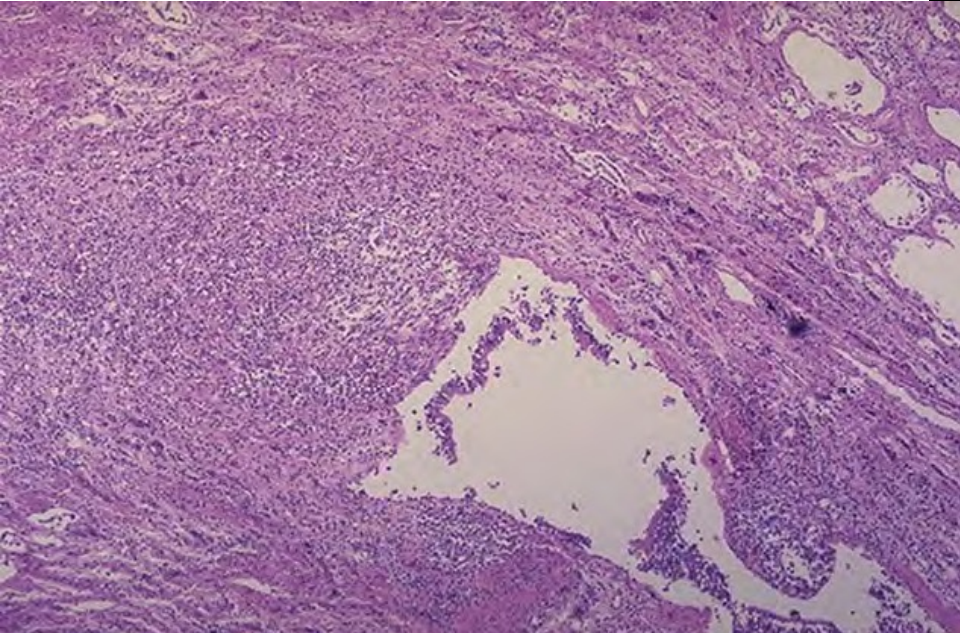
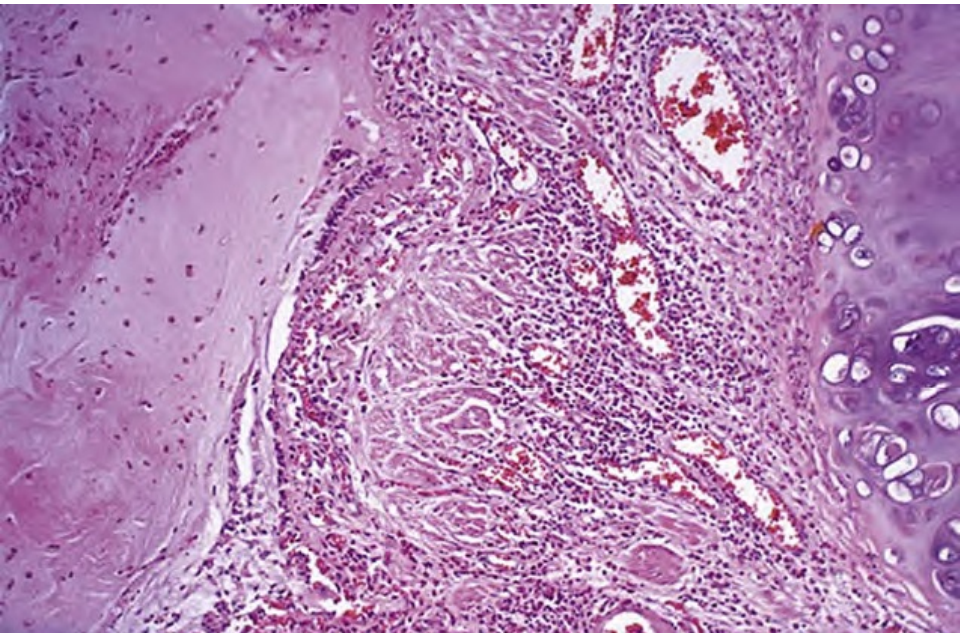
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CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

EMPHYSEMA

CHRONIC BRONCHITIS



ASTHMA

BRONCHIECTASIS

It's hard to get the air OUT

It's hard to EXHALE

Lungs are hyperinflated

- Total lung capacity: (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

**CHRONIC OBSTRUCTIVE
PULMONARY DISEASE
(COPD)**

COPD:

- **defined by the WHO as** “a common, preventable and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities caused by exposure to noxious particles or gases.”
- 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.

PURE CHRONIC BRONCHITIS

Large airways (trachea, bronchi)

- Mucus hypersecretion
- Inflammation
- (Chronic bronchitis)

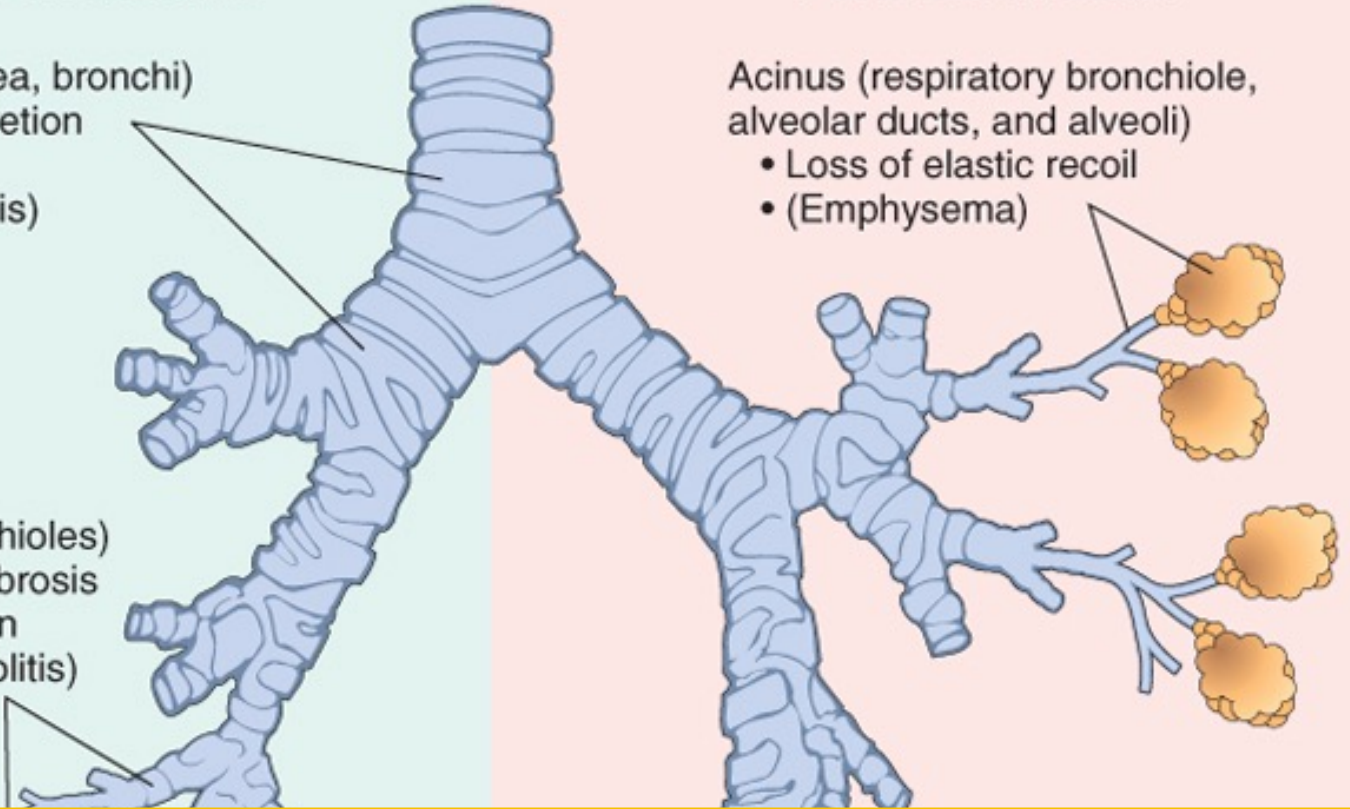
Small airways (bronchioles)

- Peribronchiolar fibrosis
- Airway obstruction
- (Chronic bronchiolitis)

PURE EMPHYSEMA

Acinus (respiratory bronchiole, alveolar ducts, and alveoli)

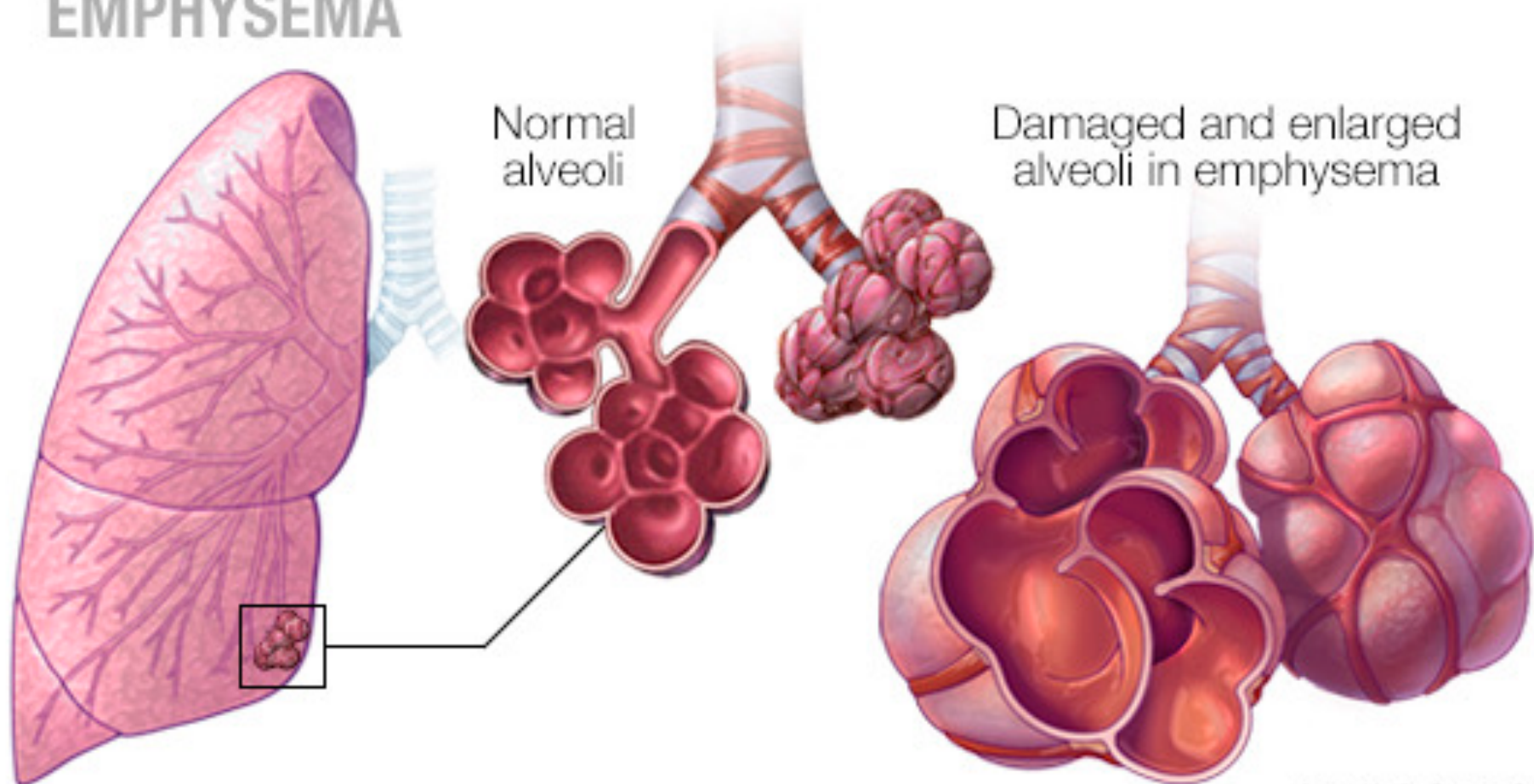
- Loss of elastic recoil
- (Emphysema)



ANATOMIC DISTRIBUTION

DEFINITION

EMPHYSEMA



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.
- Classified according to its anatomic distribution:
(1) centriacinar, (2) panacinar, (3) distal acinar, and (4) irregular

EMPHYSEMA

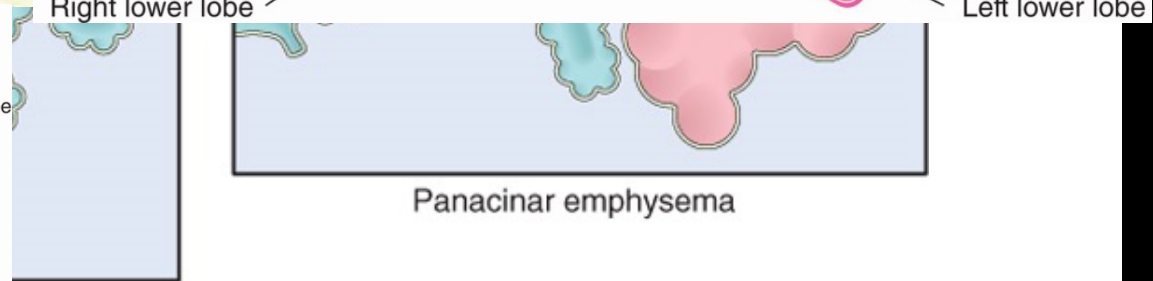
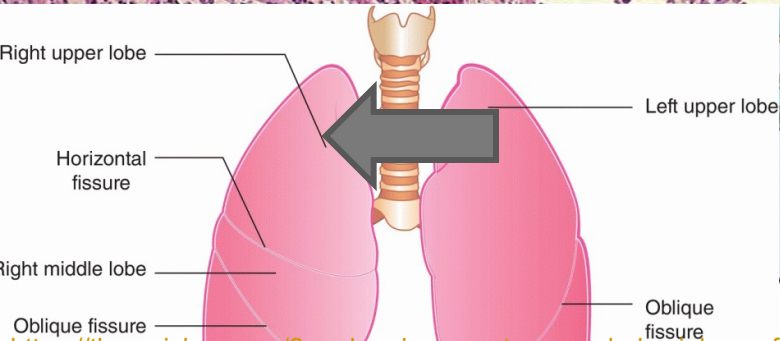
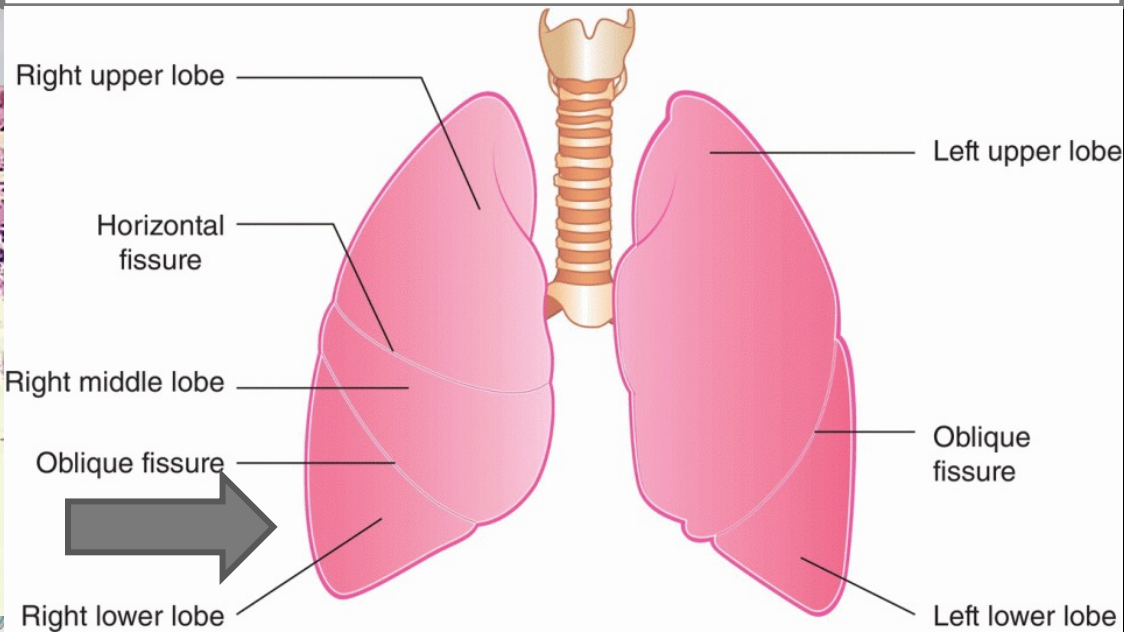
α_1 -antitrypsin deficiency



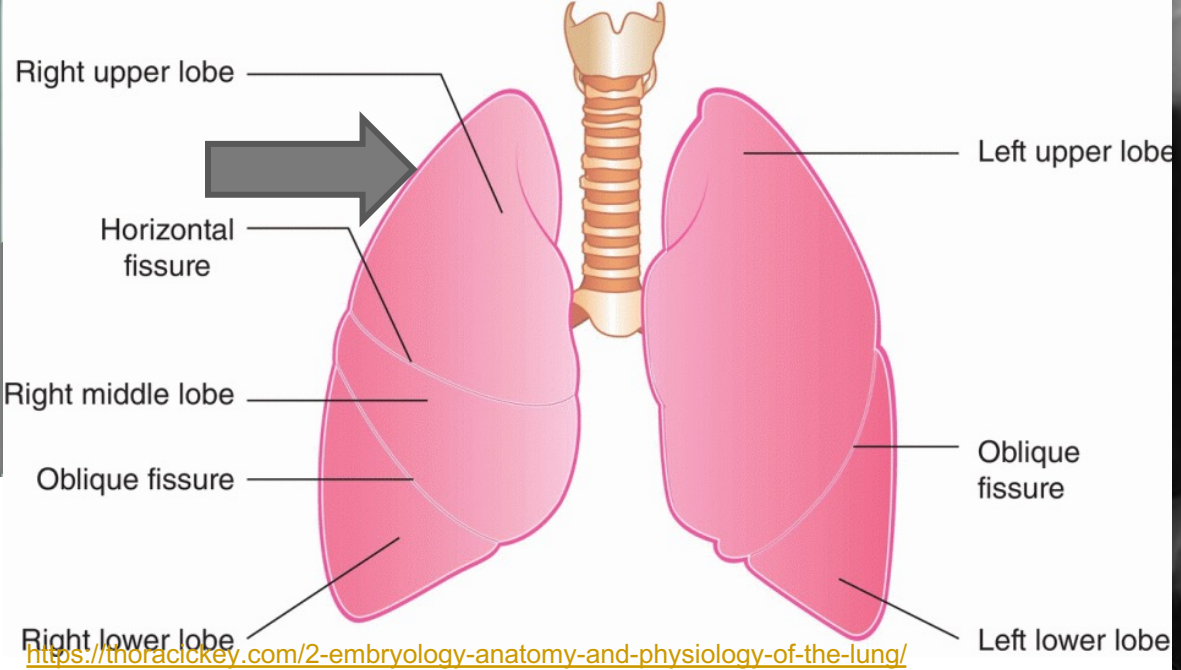
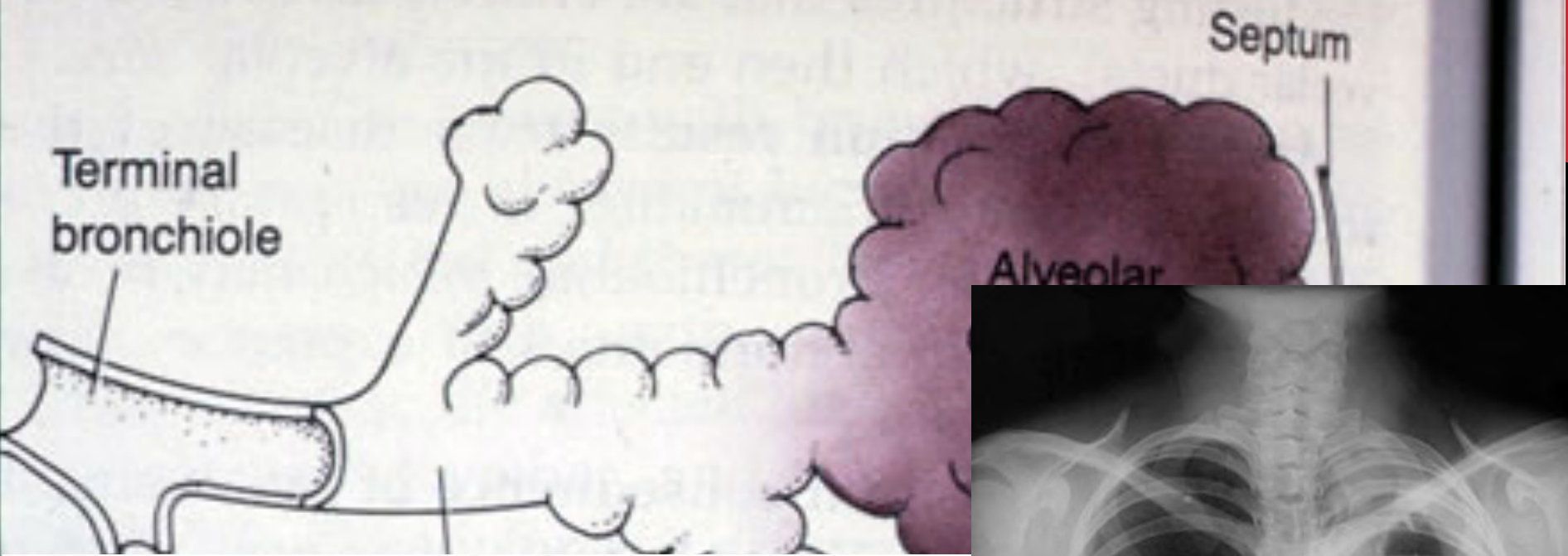
<https://health.clevelandclinic.org/even-smoking-just-one-or-two-cigarettes-a-day-increases-your-risk-of-lung-disease/>



CHRONIC BRONCHITIS



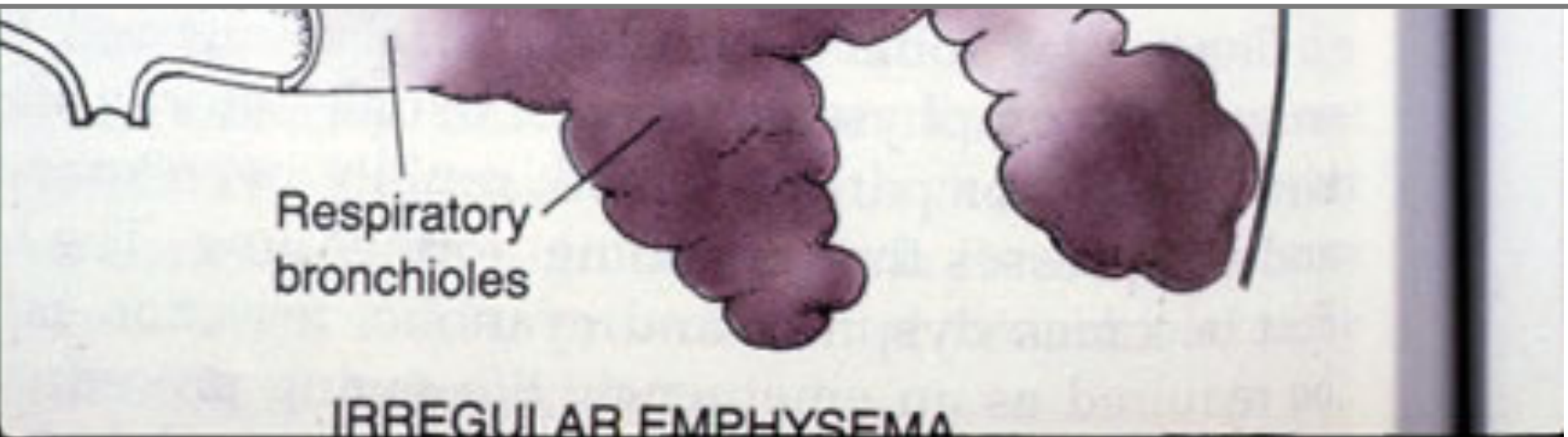
Panacinar emphysema



**Almost invariably associated
with scarring**



clinically asymptomatic and insignificant



Irregular 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. Chronic bronchitis
- C. Distal acinar emphysema
- D. Panlobular emphysema

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PATHOGENESIS

infections

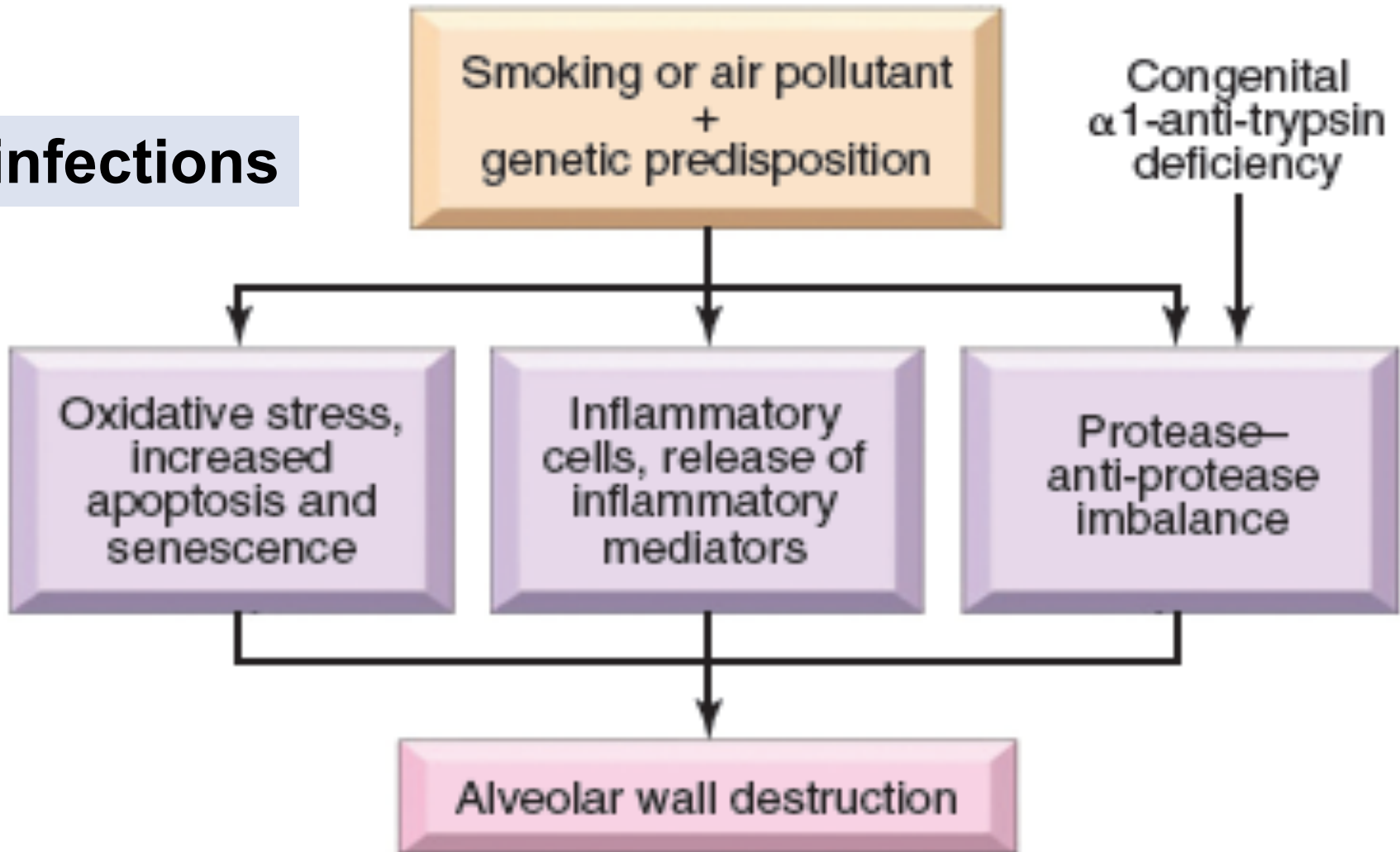
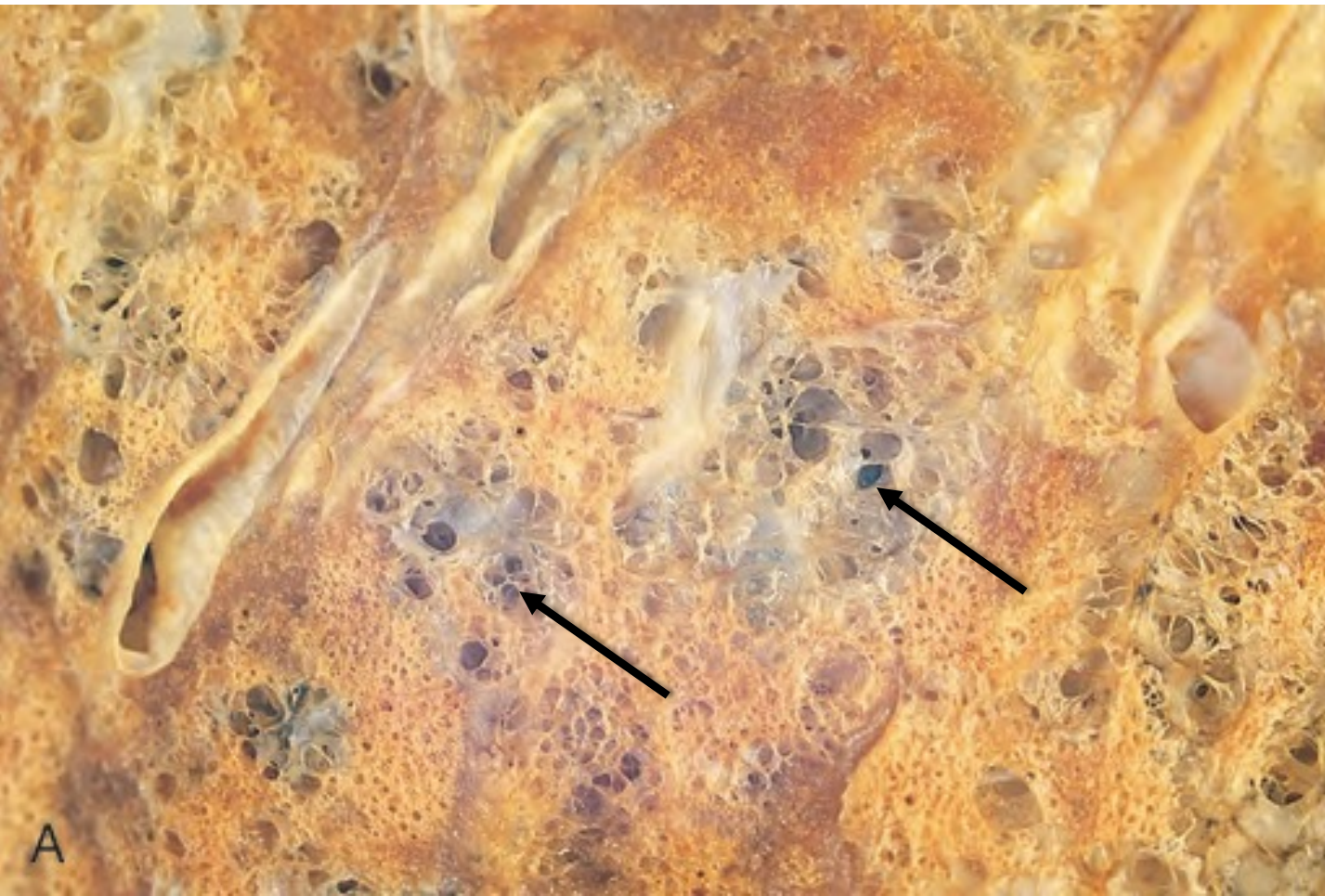


Fig. 13.6 Pathogenesis of emphysema. See text for details.

MORPHOLOGY

Macroscopic: Advanced emphysema → voluminous lungs



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



Panacinar emphysema involving the entire pulmonary lobule.

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

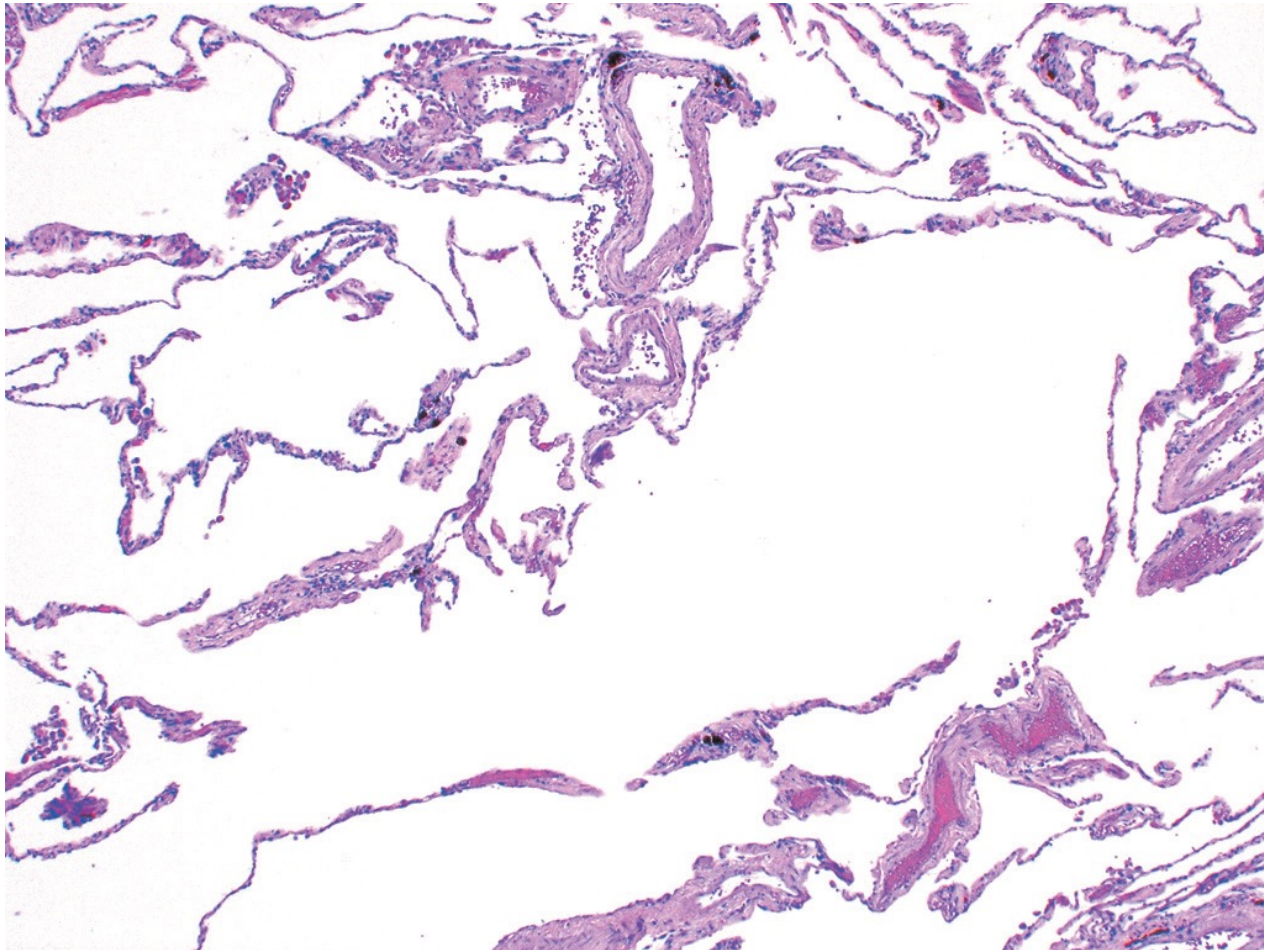


Figure 13.5 ROBBINS BASIC PATHOLOGY, 10TH EDITION

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
- Weight loss; common
- barrel-chested

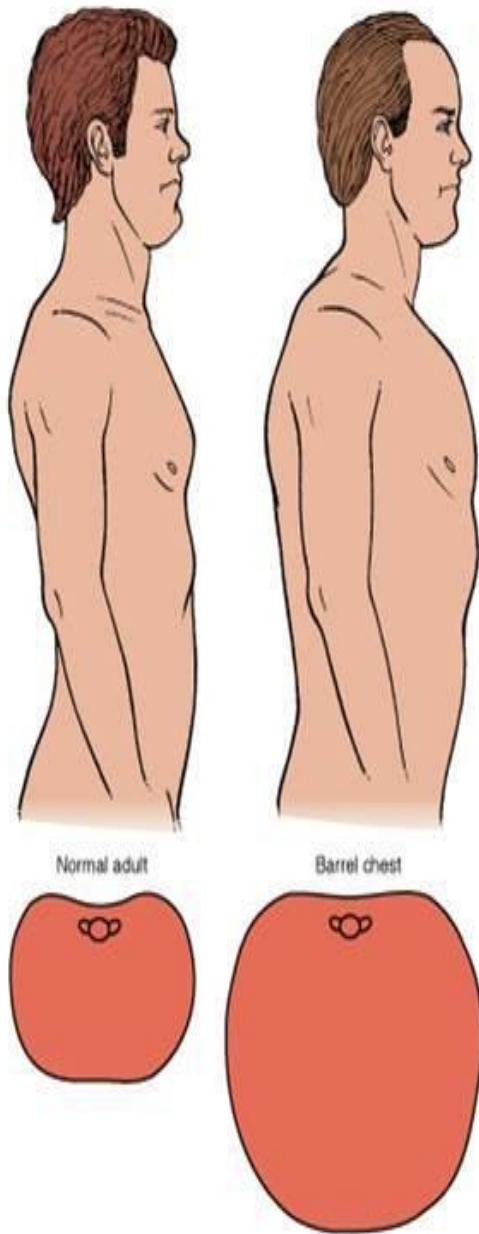


Figure 25-31 Profile and anteroposterior diameter of normal adult chest and barrel chest.

- prolonged expiration
- sitting forward in a hunched-over Position
- breathes through pursed lips
- Hyperventilation
- adequate oxygenation of Hemoglobin especially at rest and prominent dyspnea
→ “pink puffers.”
- Cough and wheezing if Coexistent asthma & chronic bronchitis.



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.
 - ✓ 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).

II. CHRONIC BRONCHITIS

Defined clinically as Persistent productive cough for **AT LEAST** 3 consecutive months in **AT LEAST** 2 consecutive years in the absence of any other identifiable cause.

- 90% cigarette smokers; air pollutants also contribute.
- chronic bronchitis is one end of the spectrum of COPD, with emphysema being the other.

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.

- **hypersecretion of mucus:**
 - The earliest feature of chronic bronchitis
 - beginning in the large airways.
- **Acquired cystic fibrosis transmembrane conductance regulator (CFTR) dysfunction.**
 - ✓ smoking leads to acquired CFTR dysfunction → secretion of abnormal dehydrated mucus → increases the severity of chronic bronchitis.
- **Inflammation.**
 - Due to the Inhalants
 - No eosinophils

- 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.

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

- **airflow obstruction in chronic bronchitis** results from:

- 1. Small airway disease**

chronic bronchiolitis: results in mild airflow obstruction.

Induced by mucus plugging of the bronchiolar lumen, inflammation, and bronchiolar wall fibrosis

- 2. Coexistent emphysema: The cause of significant airflow obstruction.**

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

MORPHOLOGY

Macroscopic:

- **Mucosal lining is hyperemic and swollen**
- **Layers of mucinous or mucopurulent secretions**

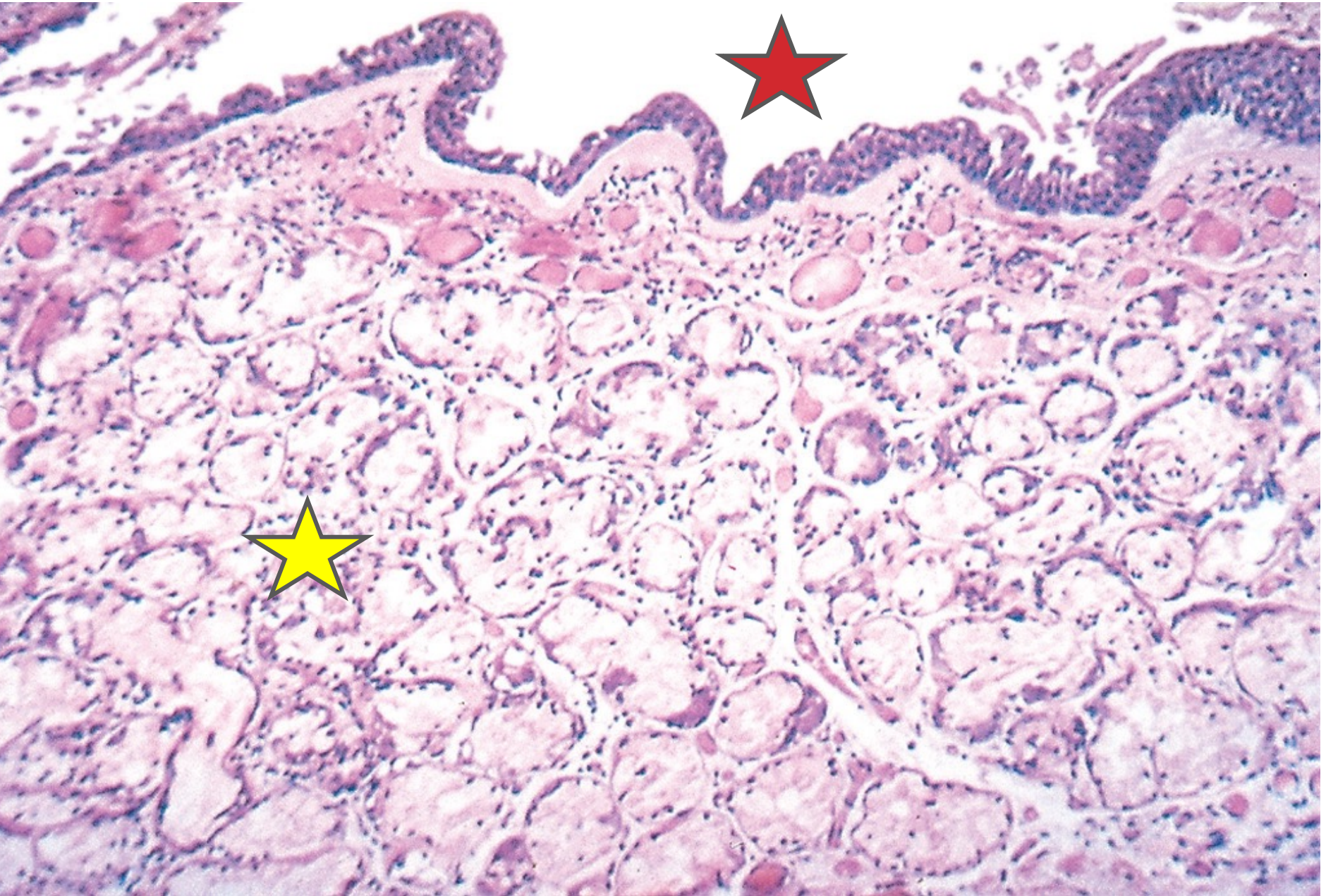


Fig. 13.9 Chronic bronchitis. The lumen of the bronchus is above. Note the marked thickening of the mucous gland layer (approximately twice-normal) and squamous metaplasia of lung epithelium. (From the Teaching Collection of the Department of Pathology, University of Texas, Southwestern Medical School, Dallas, Texas.)

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:

- persistent cough with production of sparse sputum
- 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; and ultimately respiratory failure
- Death may also result from further impairment of respiratory function due to superimposed acute infections.

- **Less dyspnea**
- absence of increased respiratory drive → the **lungs retain carbon dioxide** → **hypoxic and cyanotic.**
- For unclear reasons, patients with chronic bronchitis tend to be **obese** hence the designation “**blue bloaters**” → carbon dioxide retention, hypoxia, and cyanosis



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

THANK YOU!