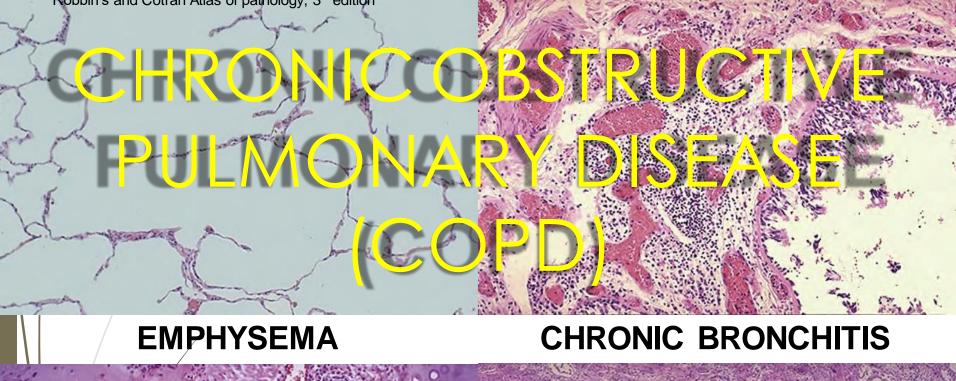
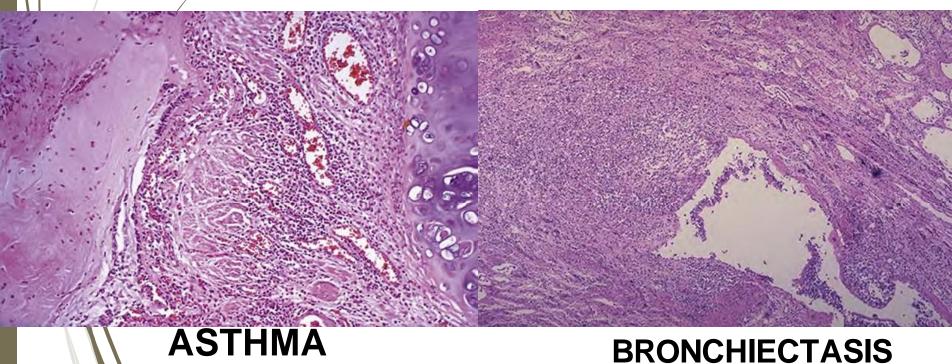
Obstructive lung disease

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CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

It's hard to get the air OUT It's hard to EXHALE

Lungs are hyperinflatted

- 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

PURE CHRONIC BRONCHITIS

PURE EMPHYSEMA

ANATOMIC DISTRIBUTION

MORPHOLOGIC CHARACTERISTICS

- Peribronchiolar fibrosis
- Airway obstruction

DEFINITION

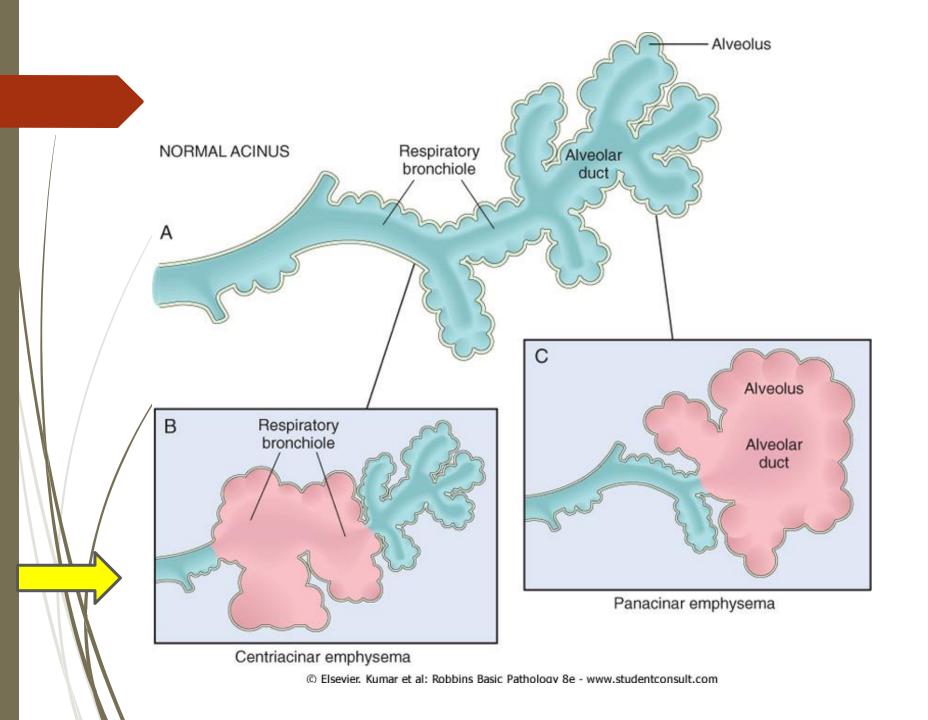




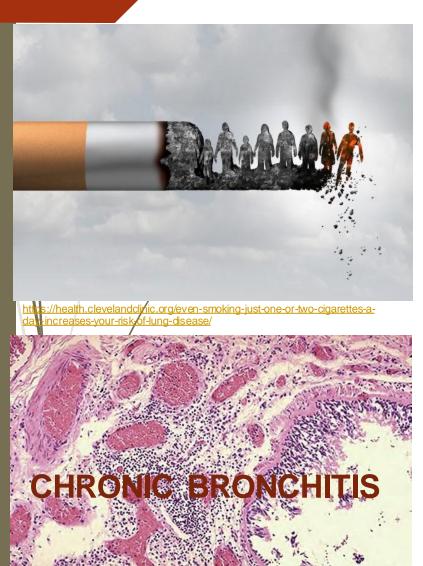
1. EMPHYSEMA

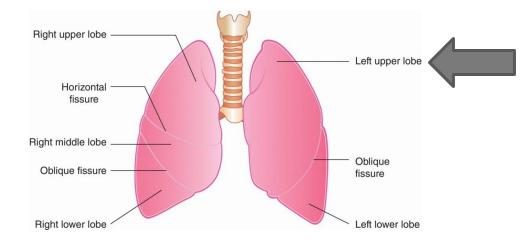
 Permanent enlargement of the airspaces distal to the terminal bronchioles with destruction of their walls and without significant fibrosis.

- Classified according to it's anatomic distribution
- (1) centriacinar, (2) panacinar, (3) distal acinar, and
- (4) *i*rregular



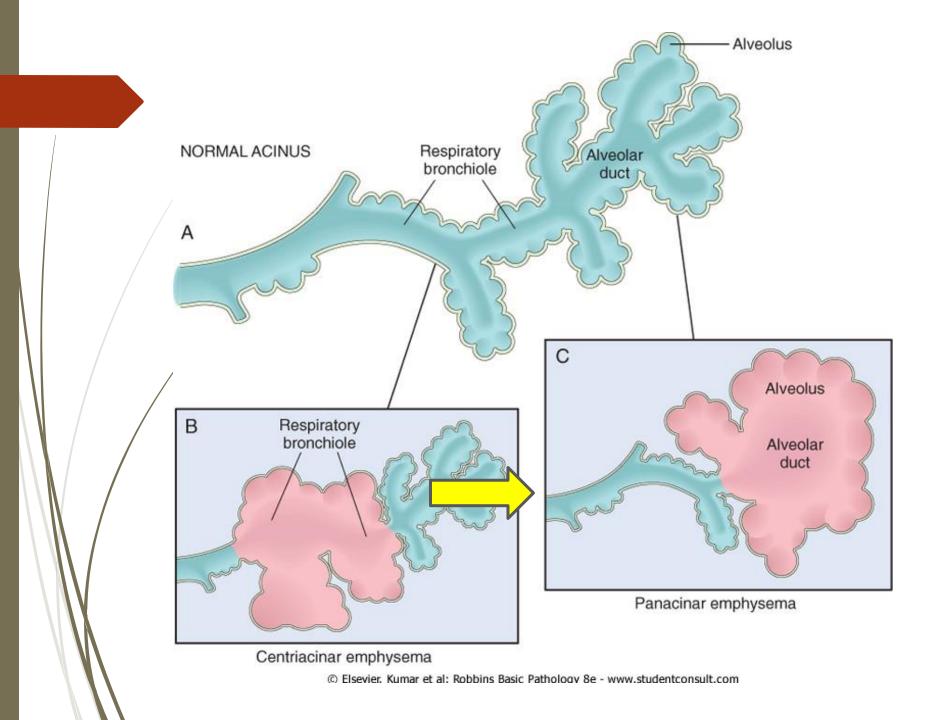
CENTRIACINAR (CENTRILOBULAR) EMPHYSEMA





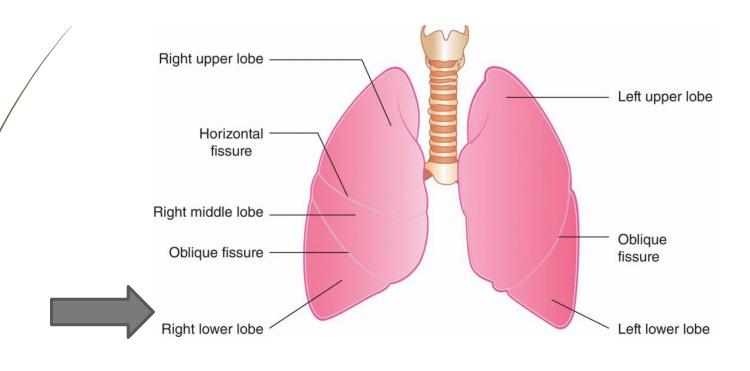
• Centriacinar (centrilobular) emphysema:

- affects the central or proximal parts of the acini first, formed by respiratory bronchioles, while distal alveoli are spared.
- cigarette smokers
- associated with chronic bronchitis
- more common and severe in the upper lobes, particularly in the apical segments.



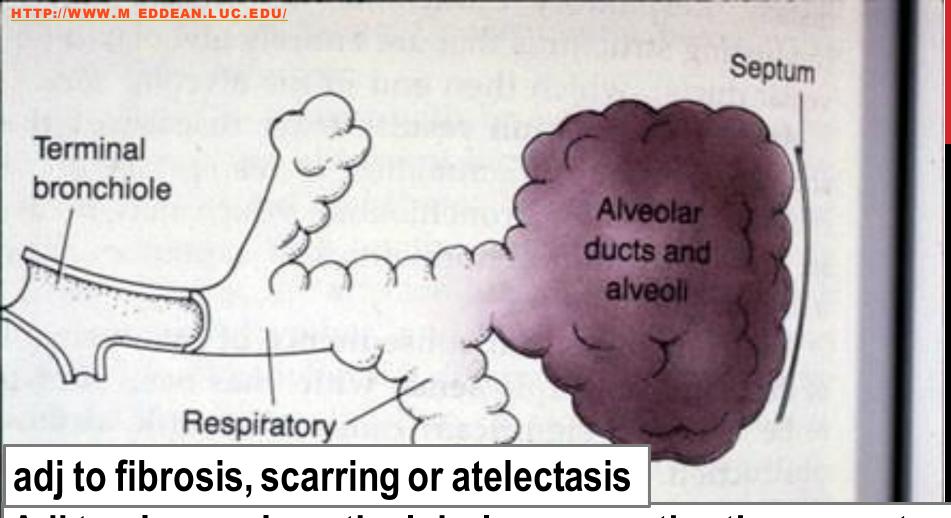
TYPES OF EMPHYSEMA

α₁-antitrypsin deficiency



Panacinar (panlobular) emphysema:

- the acini are uniformly enlarged, from the level of the respiratory bronchiole to the terminal blind alveoli.
- / associated with α_1 -antitrypsin deficiency more common in the lower lung zones.

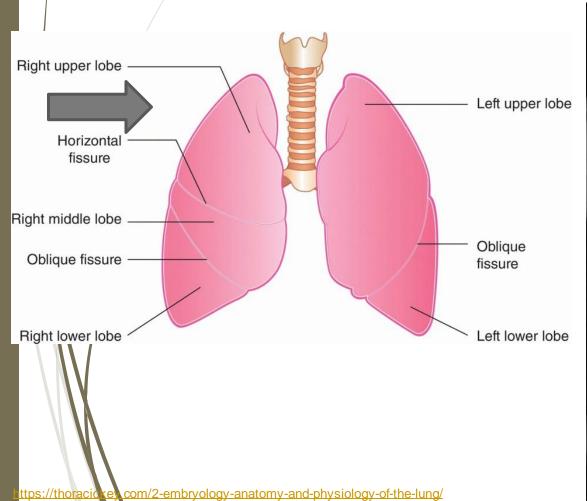


Adj to pleura, along the lobular connective tissue septa, & at the margins of the lobules

DISTAL ACINAR EMPHYSEMA

https://thoracickev.com/2-embryology-anatomy-and-physiology-of-the-lung/

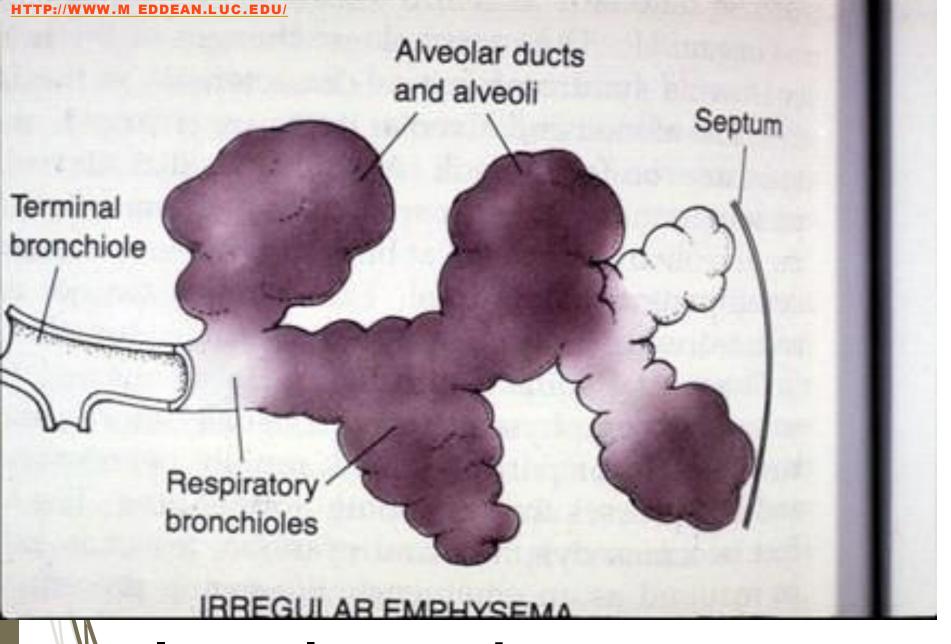
DISTAL ACINAR EMPHYSEMA





<u>Distal Acinar (Paraseptal) Emphysema:</u>

- involves the distal portion of the acinus while the proximal part is normal.
- present adjacent to the pleura, along the lobular connective tissue septa, at the margins of the lobules
- /adjacent to fibrosis, scarring or atelectasis.
- more severe in the upper half of the lungs.
- The cause is unknown.
- The presence of multiple, enlarged air spaces may form large cystic structures that give rise to bullae.
- the most common cause of spontaneous pneumothorax in young adults.



Irregular emphysema

Almost invariably associated with scarring

clinically asymptomatic, but the commonest form of emphysema

Irregular emphysema:

- The acinus is irregularly involved
- almost invariably associated with scarring
- | clinically it's asymptomatic
- considered the commonest form of 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. Ranlobular emphysema

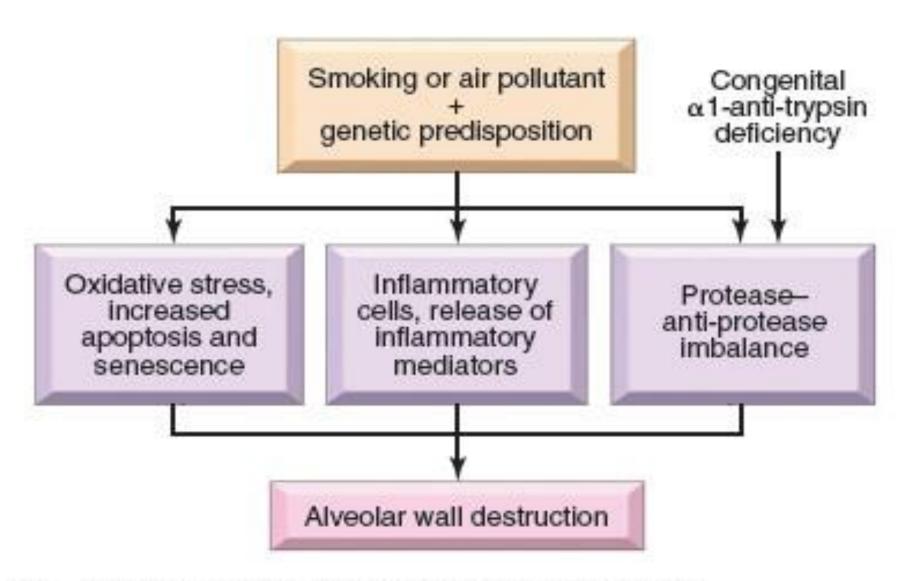


Fig. 13.6 Pathogenesis of emphysema. See text for details.

MORPHOLOGY

Macroscopic:

- Panacinar emphysema:
 - ✓ Pale, voluminous lungs

- **Centriacinar emphysema**
 - ✓ Less impressive changes
 - ✓ Deeper pink and less voluminous lungs



CENTRIACINAR EMPHYSEMA

Robbin's and Cotran Atlas of pathology, 3rd edition

☐ Microscopic examination of the lung:

destruction of alveolar walls & enlarged air spaces

No significant fibrosis

• small airways collapse due to loss of elastic tissue in the surrounding alveolar septa during expiration (chronic airflow obstruction).

Bronchiolar inflammation in advanced cases.

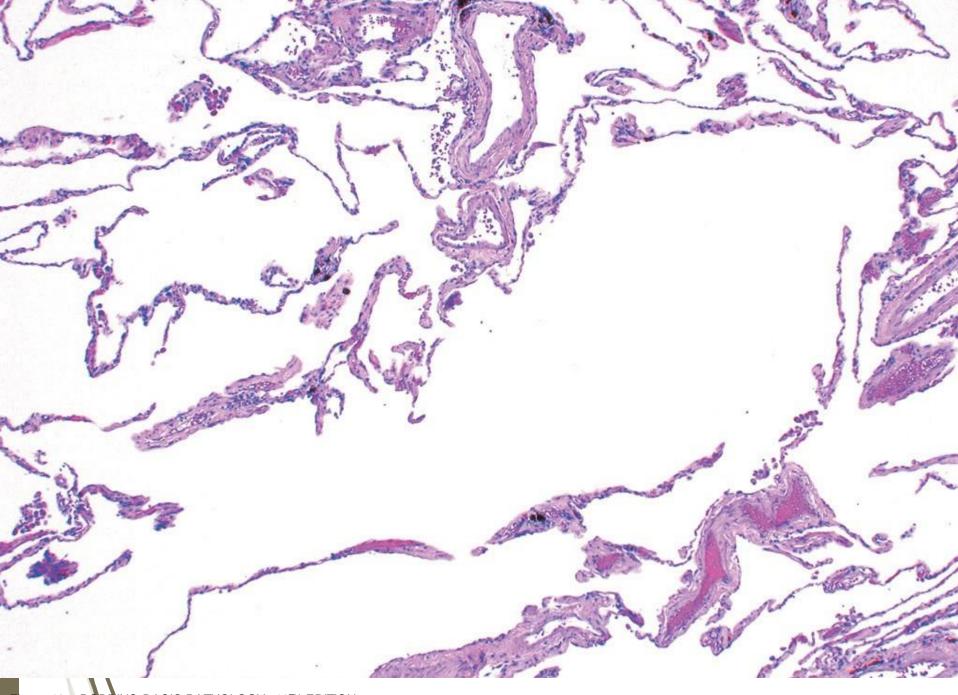


Figure 13.5 ROBBINS BASIC PATHOLOGY, 10[™] EDITION

THE CLASSIC PRESENTATION OF EMPHYSEMA WITH NO

"BRONCHITIC" COMPONENT

- Dyspnea
- barrel-chested
- prolonged expiration
- sitting forward in a hunchedover position
- Hyperventilation.
- adequate oxygenation of hemoglobin and prominent dyspnea
 - → "pink puffers."
- Cough and wheezing if
 coexistent asthma and chronic



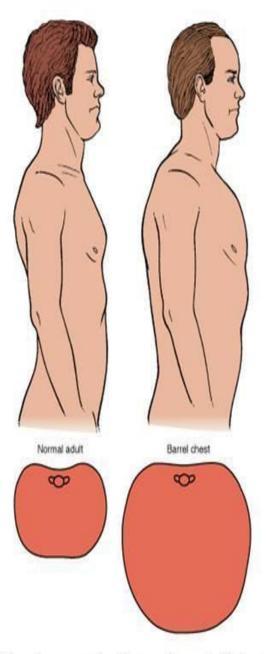


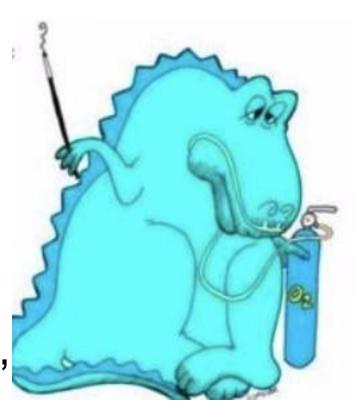
Figure 25-31 Profile and anteroposterior diameter of normal adult chest and barrel chest.



https://ratedmedicine.wordpress.com/barrel-chest/

THE OTHER END OF THE SPECTRUM: EMPHYSEMA WITH PRONOUNCED CHRONIC BRONCHITIS AND A HISTORY OF RECURRENT INFECTIONS.

- Less dyspnea
- absence of increased respiratory
 drive 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



COMPLICATIONS

Destruction of the walls distal to the terminal bronchioles → hypoxia → Hypoxia-induced pulmonary vascular spasm → gradual development of secondary pulmonary hypertension → in 20-30% right-sided congestive heart failure (cor pulmonale).

Death from emphysema is related to either respiratory failure or right-sided heart failure.

CONDITIONS RELATED TO EMPHYSEMA

Compensatory emphysema:

 Compensatory dilation of alveoli in response to loss of lung substance.

 As hyper-expansion of residual lung parenchyma following surgical removal of a diseased lung

Obstructive overinflation:

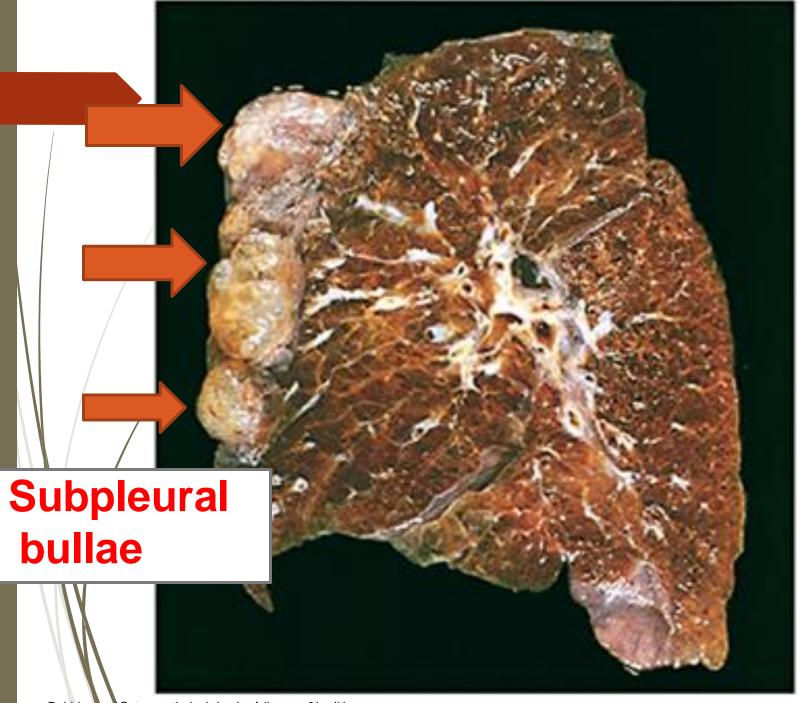
- Lung expands because air is trapped within it.
- Subtotal obstruction by a tumor or foreign object.
- Can be Life-threatening emergency if distends sufficiently to compress the remaining normal lung.

Bullous emphysema:

Any form of emphysema, Most are subpleural

Large subpleural blebs or bullae

Pneumothorax if rupture



Mediastinal (interstitial) emphysema:

Air in connective tissue of the lung, mediastinum, and subcutaneous tissue.

II. CHRONIC BRONCHITIS

• Common in cigarette smokers; air pollutants also contribute.

Clinical diagnosis

• Persistent productive cough for AT LEAST 3 consecutive months in AT LEAST 2 consecutive years.

In early stages airflow is not obstructed.

 Heavy smokers: develop chronic outflow obstruction, usually with associated emphysema

 May coexist with hyper-responsive airways with intermittent bronchospasm and wheezing

 asthmatic bronchitis

PATHOGENESIS

hypersecretion of mucus

airflow obstruction

- hypersecretion of mucus, beginning in the large airways.
 - cigarette smoking, other air pollutants:
 - hypertrophy of mucous glands in the trachea and bronchi

- increase in mucin-secreting goblet cells in the epithelial surfaces of smaller bronchi and bronchioles
- inflammation without eosinophils

- airflow obstruction results from:
 - 1. Small airway disease chronic bronchiolitis: results in early and mild airflow obstruction. Induced by mucus plugging of the bronchiolar

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

lumen, inflammation, and bronchiolar wall fibrosis

MORPHOLOGY

Macroscopic:

- Mucosal lining is hyperemic and swollen
- Layers of mucinous or mucopurulent secretions ,The smaller bronchi and bronchioles also may be involved

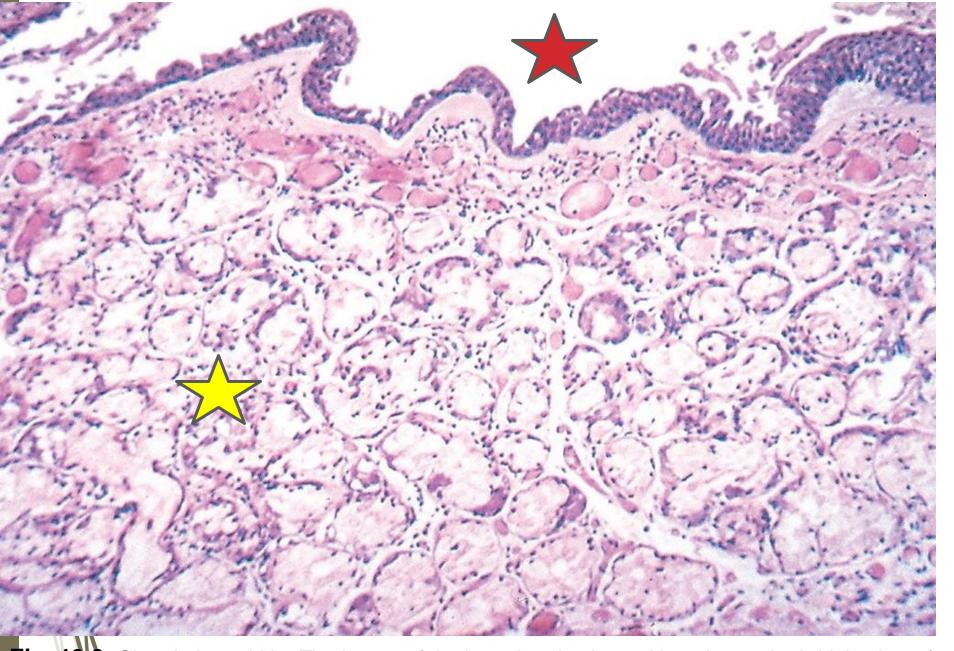


Fig. 13.9 Chronic bronchitis. The lumen of the bronchus is above. Note the marked thickening of the mucous gland layer (approximately twice-normal) and squamous metaplasia of lung epithelium. (From the Teaching Collection of the Department of Pathology, University of Texas, Southwestern Medical School,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

MICROSCOPIC:

Enlargement of the mucus-secreting glands

Inflammatory cells, largely mononuclear and neutrophils.

- Chronic bronchiolitis (small airway disease),
 characterized by goblet cell metaplasia, mucous plugging,
 inflammation, and submucosal fibrosis
- Bronchiolitis obliterans in severe cases: complete obliteration of the lumen as a consequence of fibrosis
- Changes of emphysema often co-exist

CLINICAL FEATURES:

Prominent cough with production of sputum

 chronic bronchitis and COPD patients show frequent exacerbations, rapid disease progression, and poorer outcomes than emphysema alone.

 Progressive disease is marked by the development of pulmonary hypertension, cardiac failure, recurrent infections; and ultimately respiratory failure



THANK YOU!