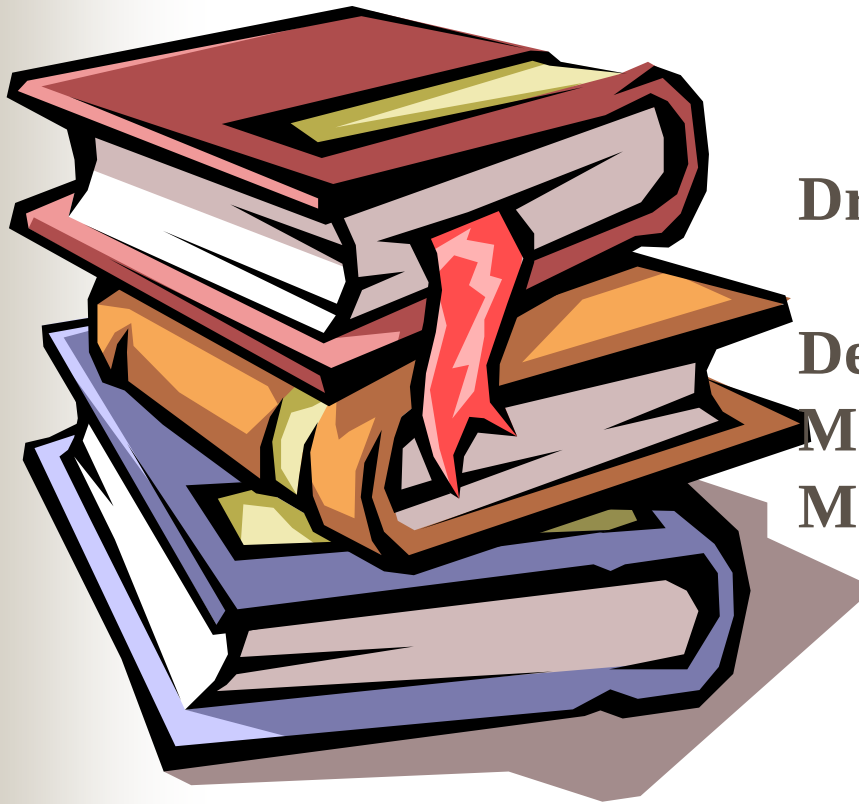


Chronic Bronchitis & Emphysema



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Chronic Bronchitis

Definition

- Persistent cough with expectoration on most days for at least 3 months of the year for two or more consecutive years.
- Middle aged $M > F$
- Is associated with emphysema

Etiopathogenesis

- Cigarette Smoking
- Atmospheric pollution(SO₂,NO₂,Dust,toxins,etc)
- Other factors — occupational(Pneumoconiosis), familial and genetic factor.

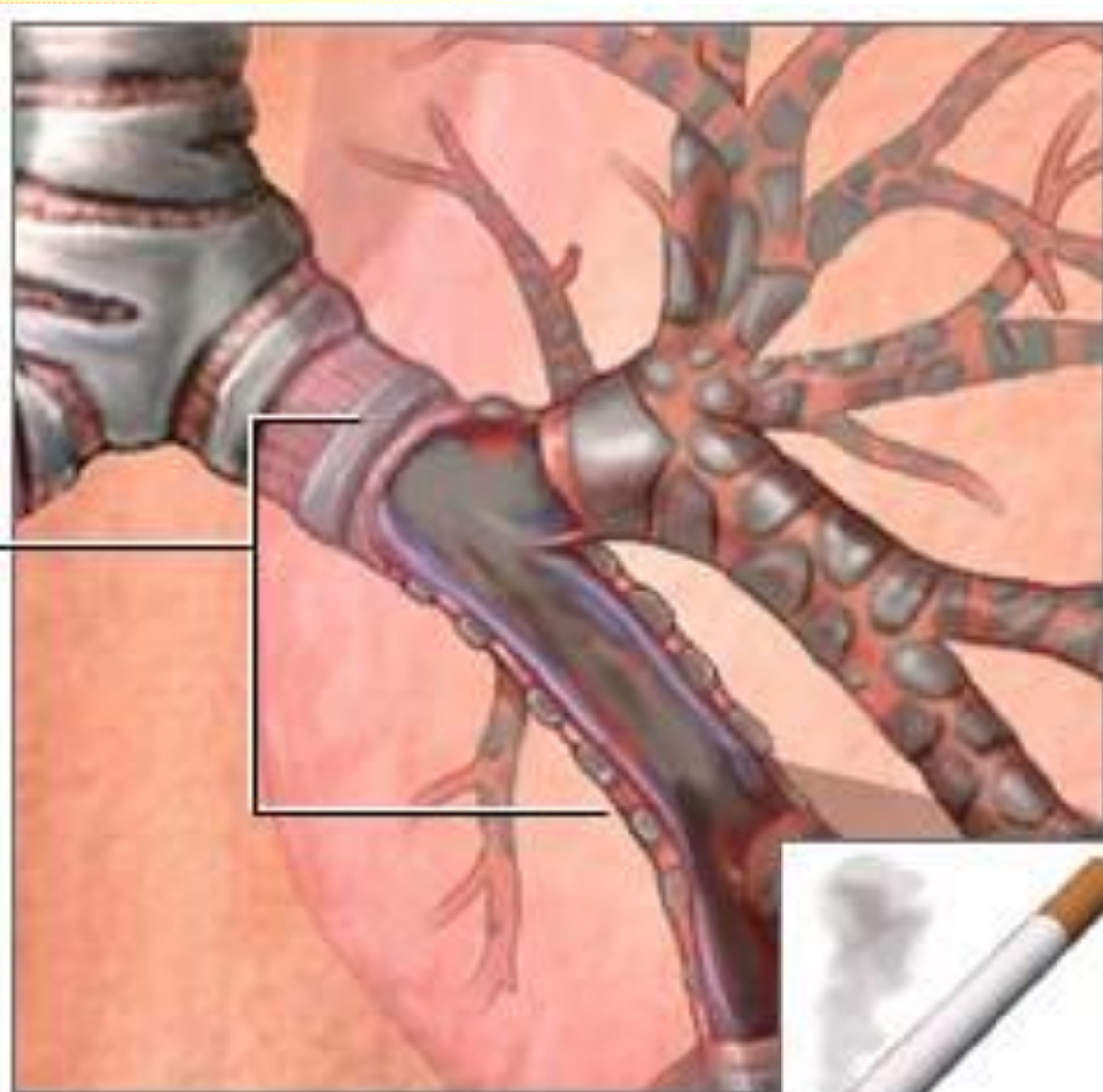
Note :*Cigarette impairs ciliary motility. Inhibits formation of alveolar macrophages, leads to hypertrophy and hyperplasia of mucus secreting glands -> obst of small airways -> stimulates vagus and causes bronchoconstriction. Bact,viral and mycoplasmal do not initiate but usually are seen in bronchi of cig. Smokers and predisposes to bronchitis.*

Cigarette smoking, or exposure to second-hand cigarette smoke, is the primary cause of chronic bronchitis symptoms.



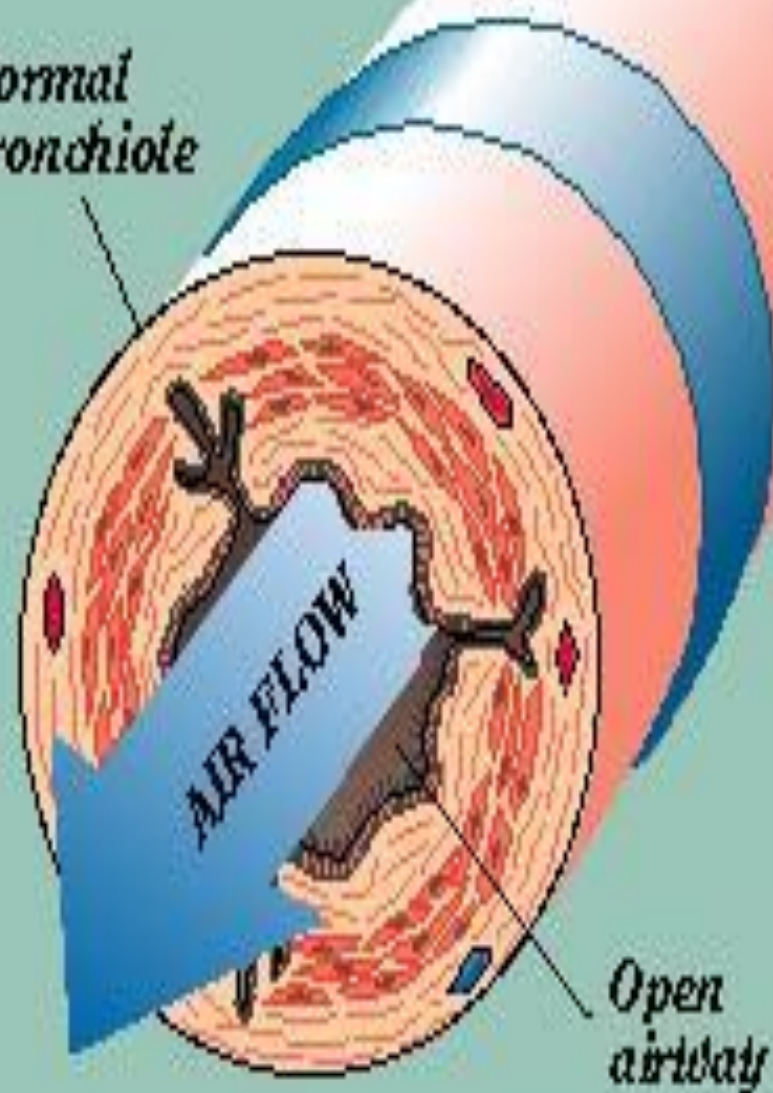


Inflammed
primary and
secondary
bronchi



Chronic bronchitis is caused most often
by exposure to airborne pollutants such
as cigarette smoke

*Normal
bronchiole*



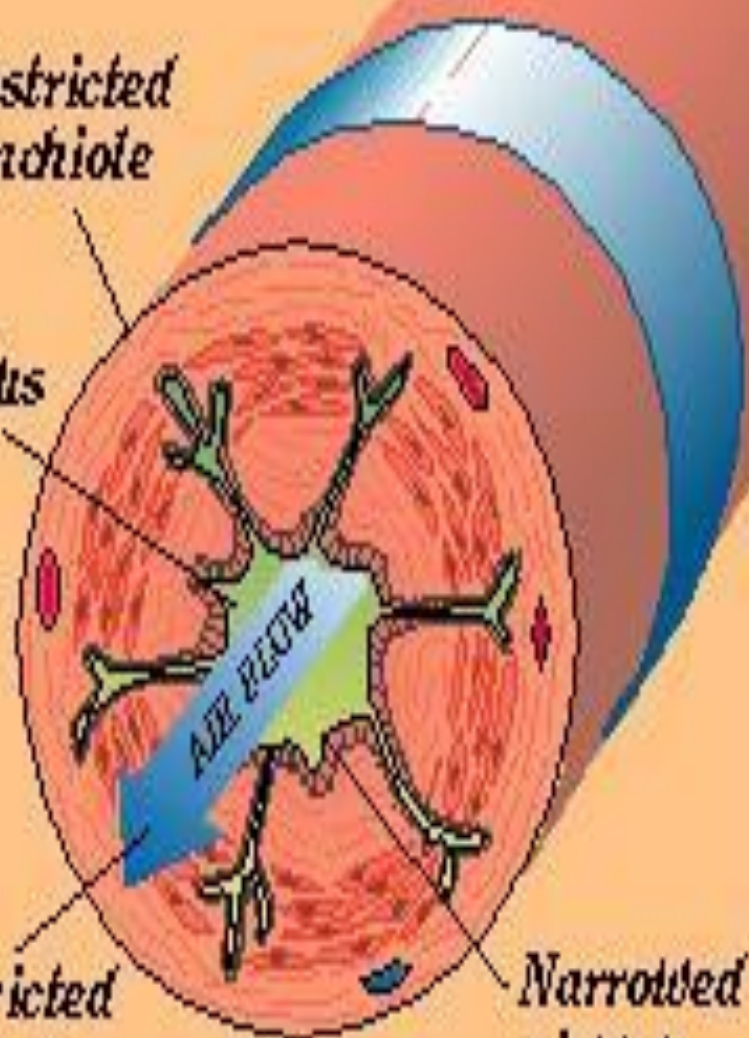
NORMAL AIRWAY

*Constricted
bronchiole*

Mucus

*Restricted
air flow*

*Narrowed
airway*



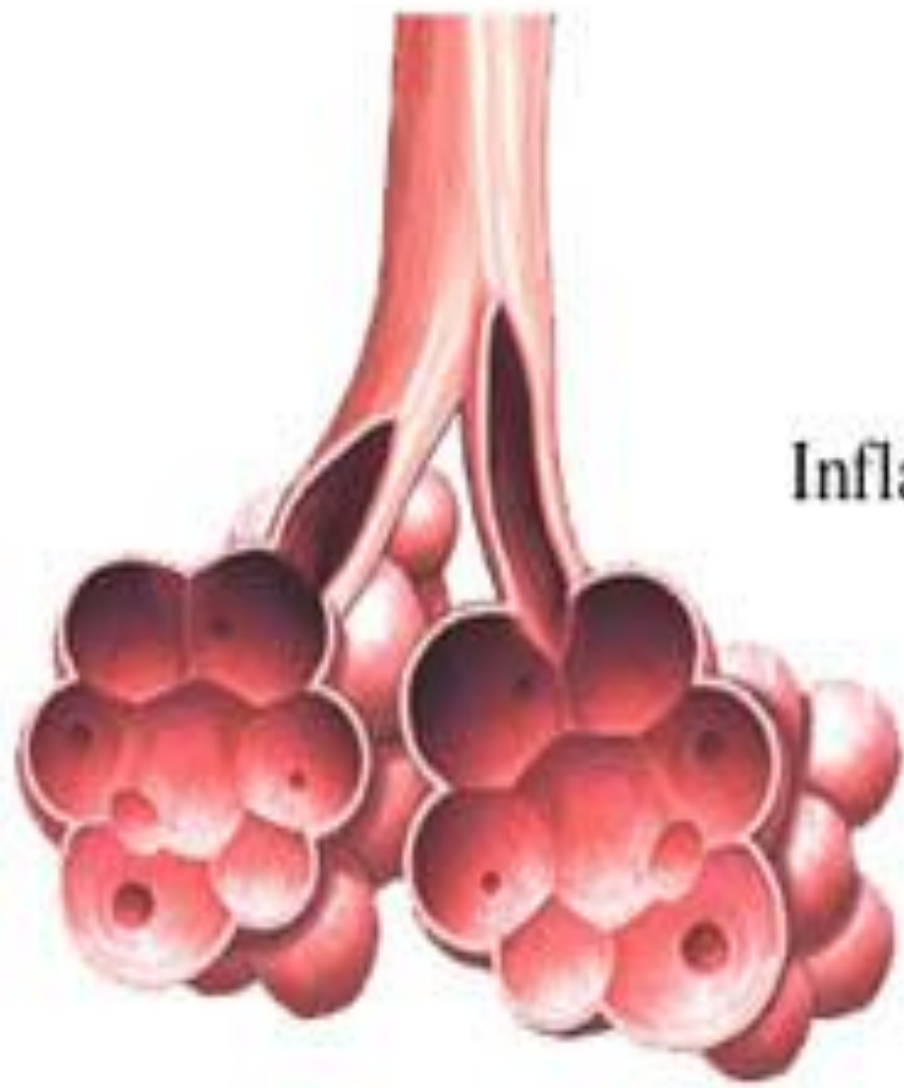
AIRWAY IN BRONCHITIS



CHRONIC BRONCHITIS

Inflammation of the bronchial tubes' mucus membranes with epithelial shedding and excess mucus production.

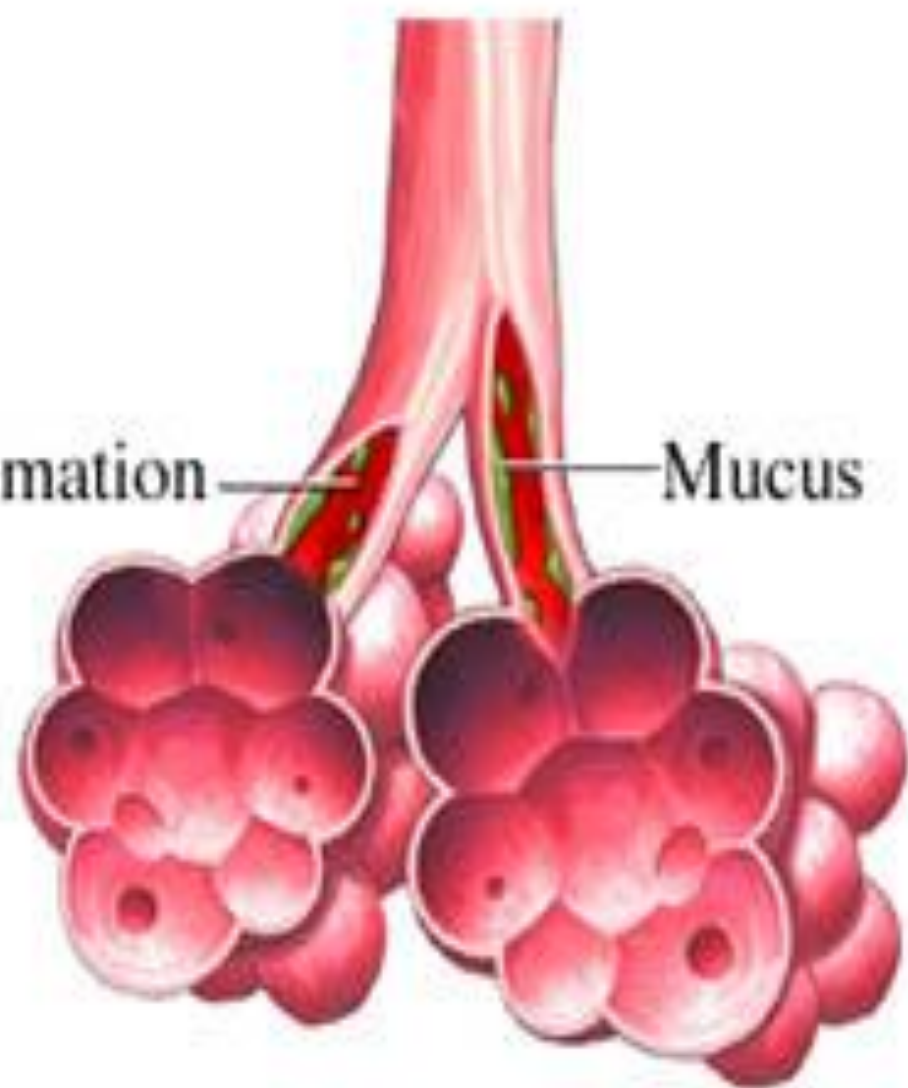
Normal



Chronic bronchitis

Inflammation

Mucus





PATHOLOGICAL CHANGES

Gross

- bronchial wall thickened, hyperemic, edematous, Lumina of bronchi contains mucus plug and purulent exudates.

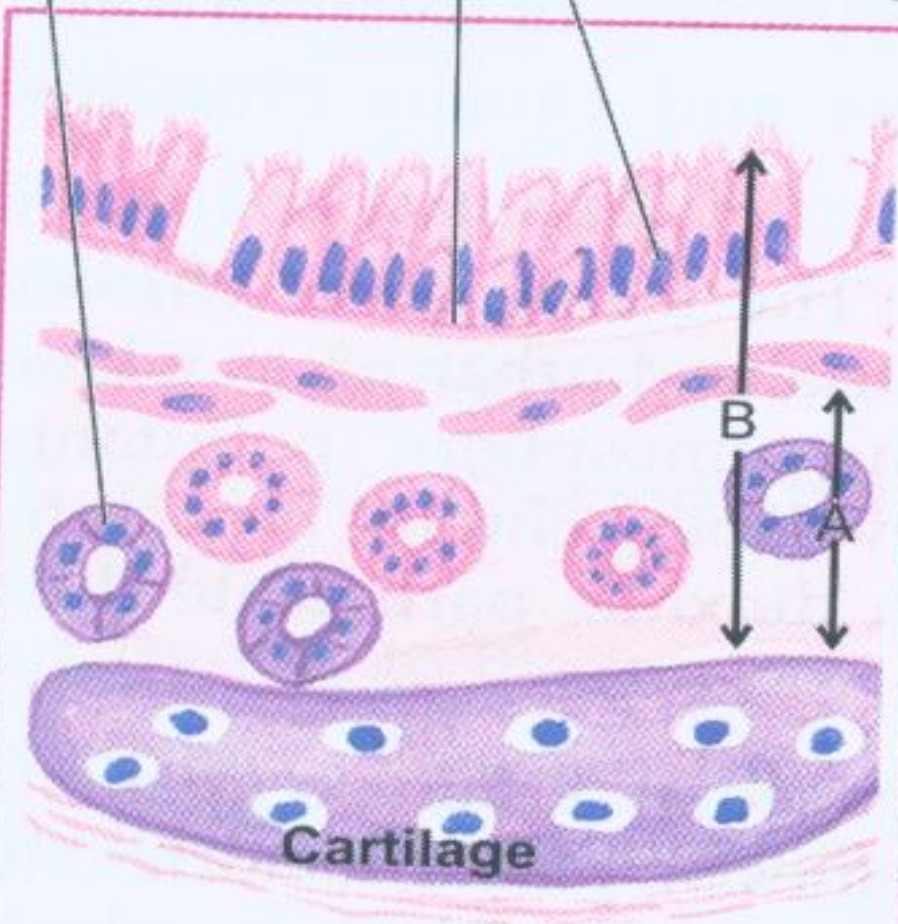
Microscopy

- cartilage containing large airways have hypertrophy and hyperplasia of submucosal mucin glands. Increase in thickness quantitatively assessed by **Reid index**(*the ratio between thickness of submucosal gland to that of bronchial wall*)
- Bronchial epi. may show sq.metaplasia and dysplasia.
- The non-cartilage containing small airways show goblet cell hyperplasia and intraluminal and peribronchial fibrosis.

Basement membrane

Mucous glands

Mucous lining



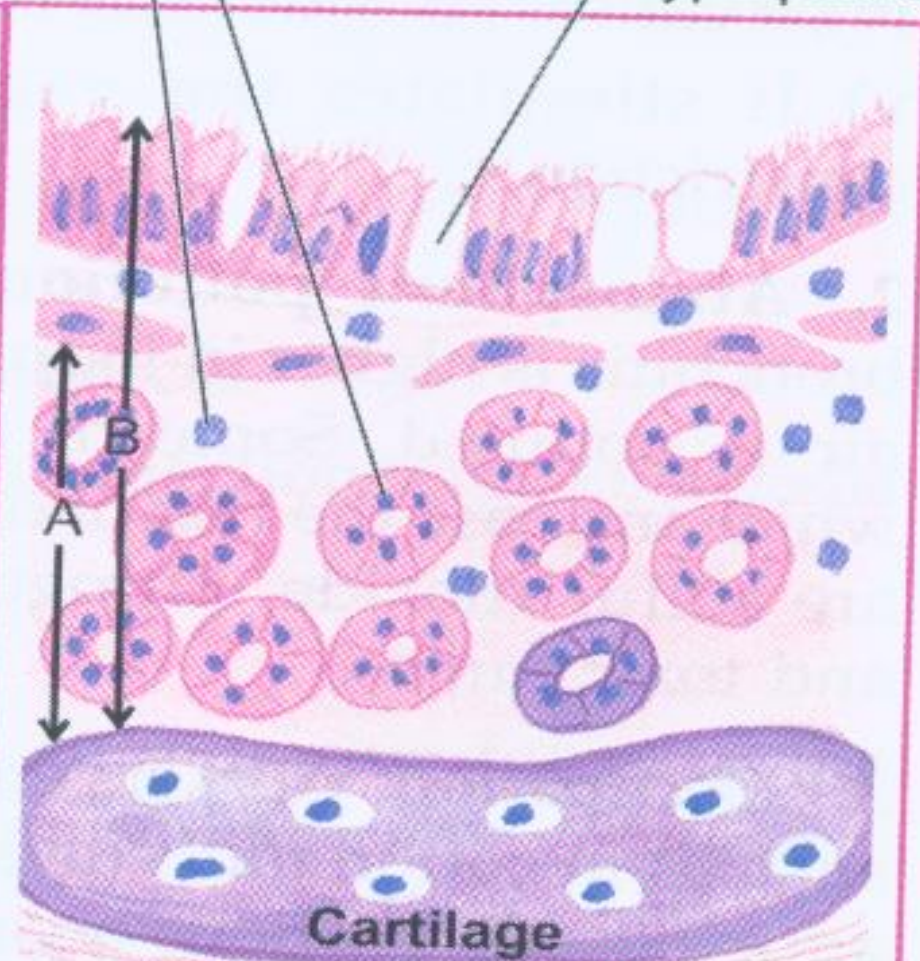
Reid index=Ratio of B:A

NORMAL

Chronic inflammatory cell infiltrate

↑Mucous glands

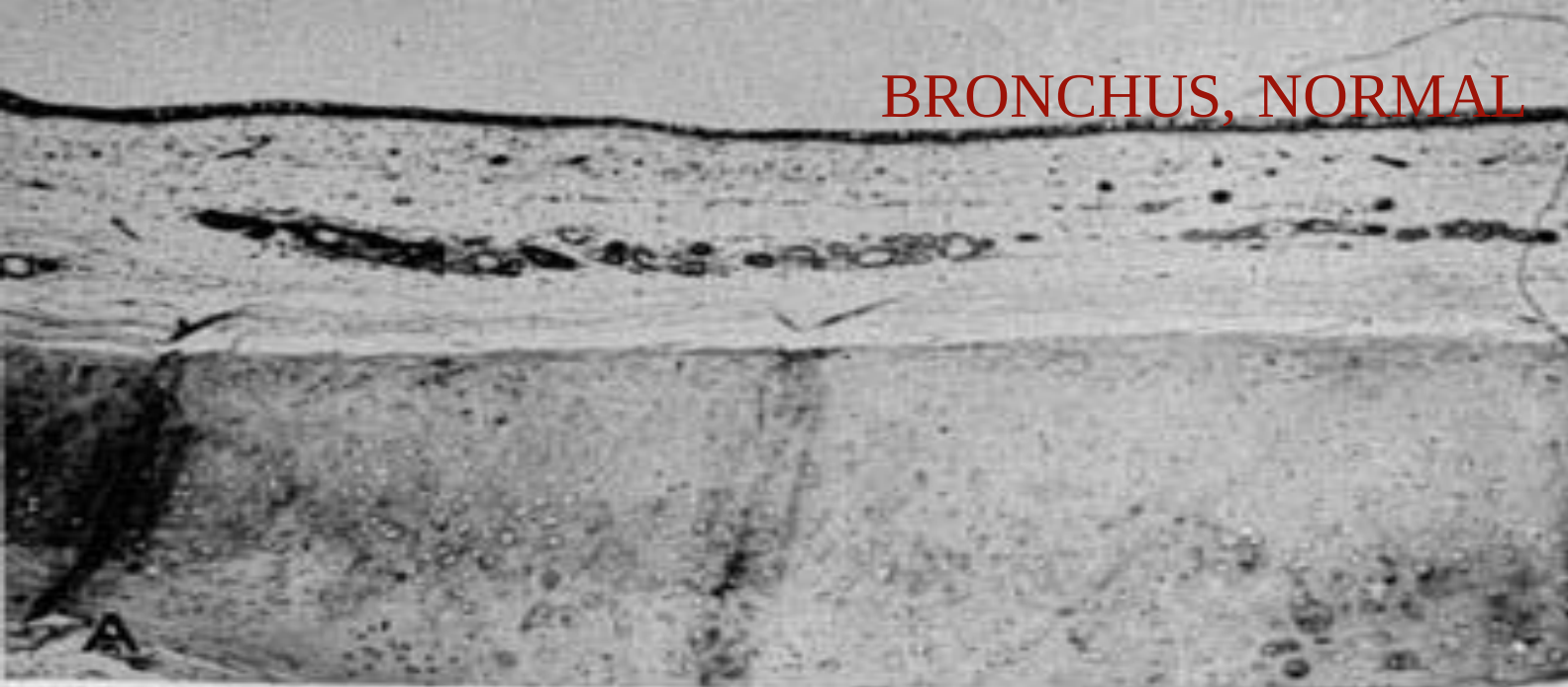
Goblet cell hyperplasia



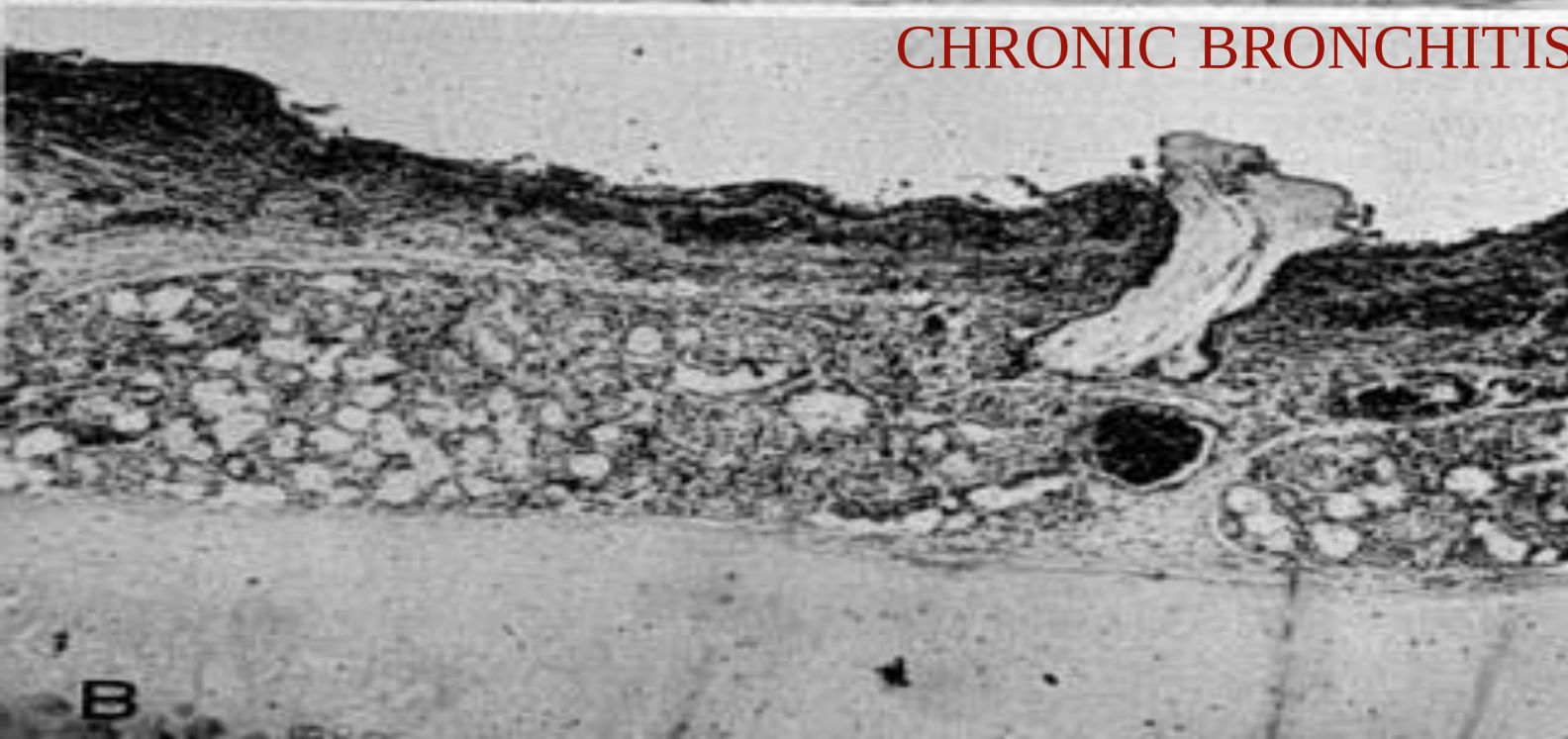
Reid index increased

CHRONIC BRONCHITIS

BRONCHUS, NORMAL

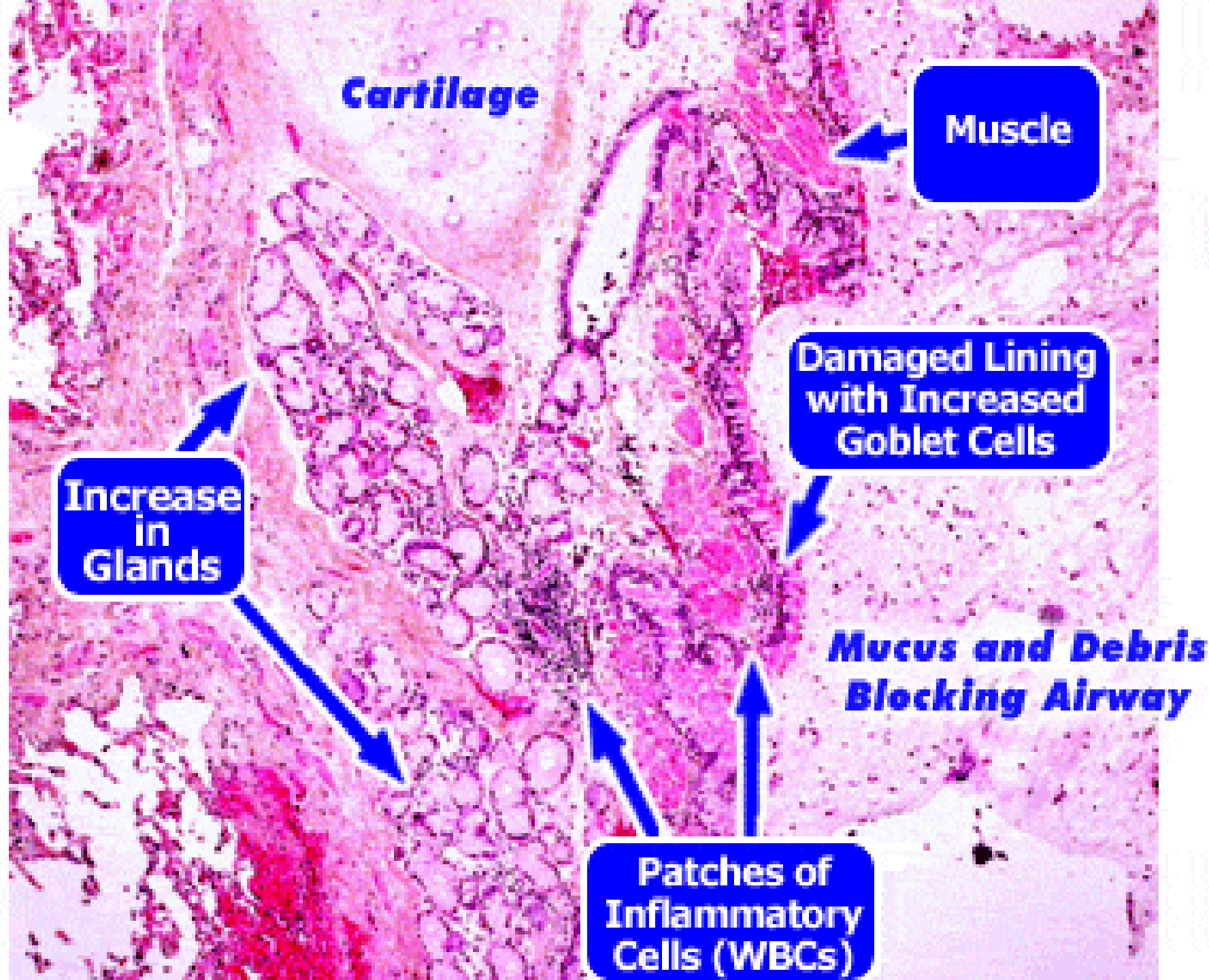


CHRONIC BRONCHITIS

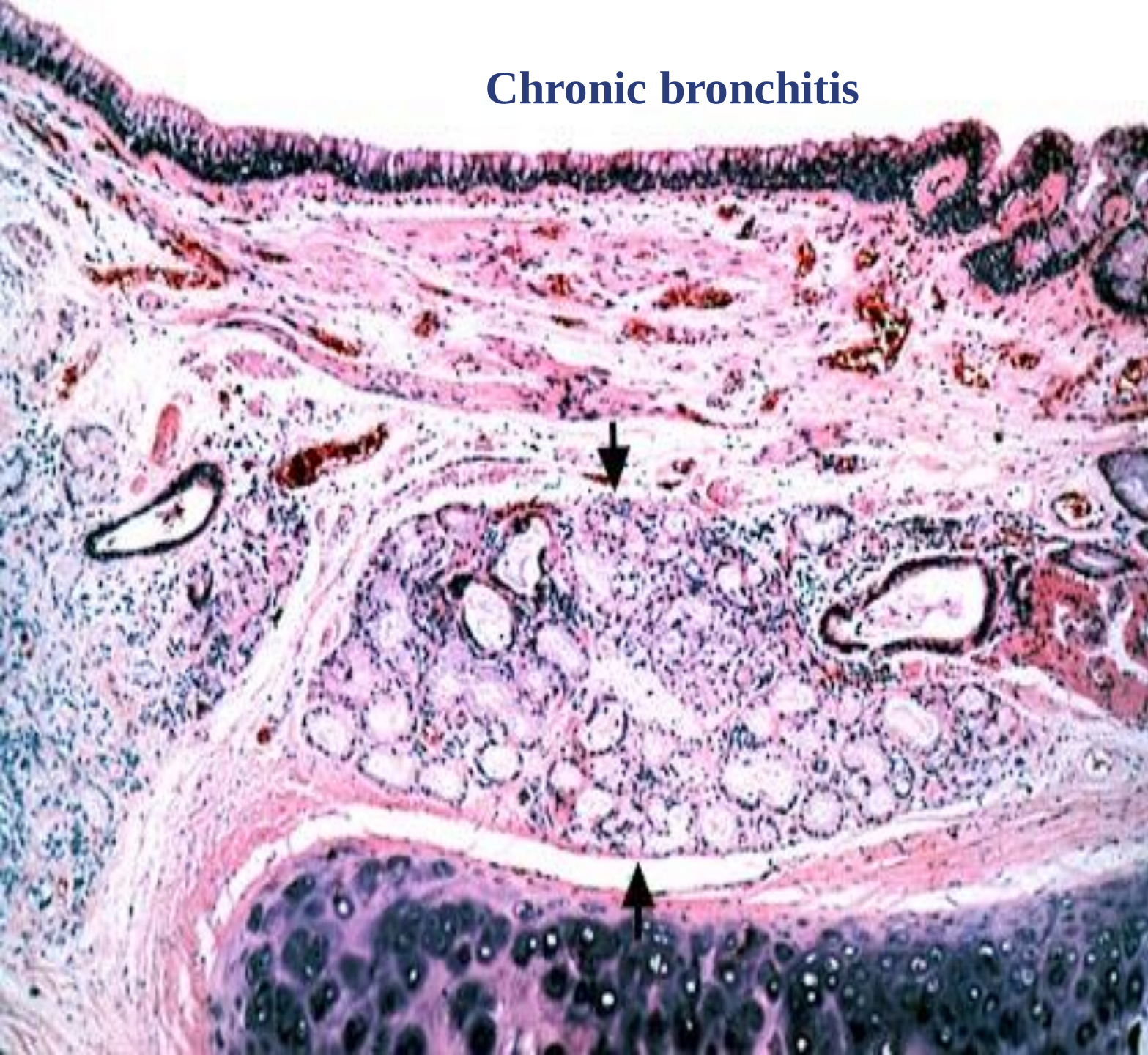


Note the increased thickness of bronchial mucous glands in the submucosa of the image with chronic bronchitis (bottom) compared to the normal bronchus (top image).

Chronic Bronchitis



Chronic bronchitis



Mucous gland hyperplasia, the histologic correlate of chronic bronchitis is defined as a seromucous gland to wall thickness greater than $\frac{1}{3}$. In this case the seromucous gland thickness (arrows) is more than $\frac{1}{2}$ of the distance between the perichondrium and the epithelial basement membrane. Note also the goblet cell hyperplasia in the epithelium. The normal percentage of goblet cells is 20%.

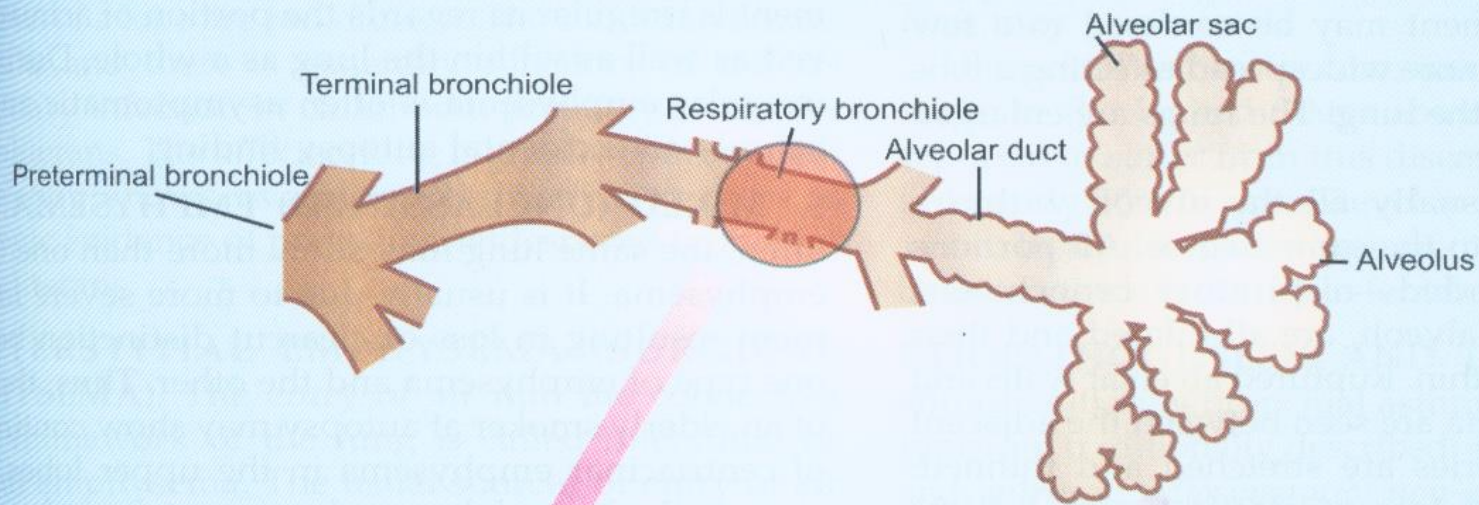
EMPHYSEMA

Definition

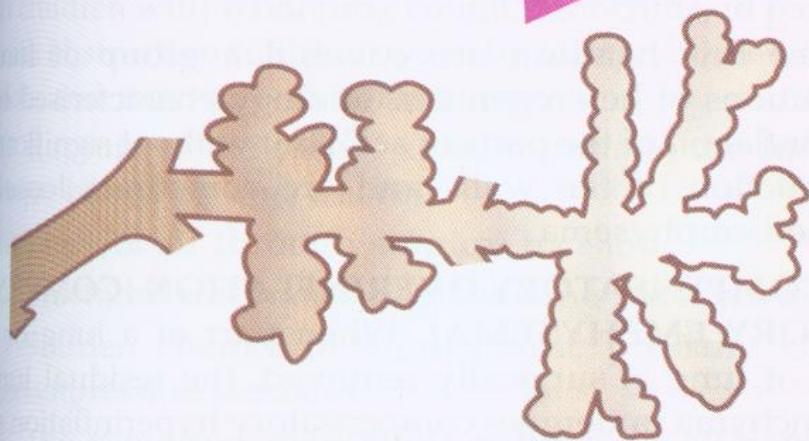
- Combination:-permanent dilatation of air spaces distal to the terminal bronchioles and the destruction of the walls of dilated air space.



- Commonest form of COPD is the combination of chronic bronchitis and pulmonary emphysema.



A, NORMAL ACINUS



B, CENTRIACINAR EMPHYSEMA



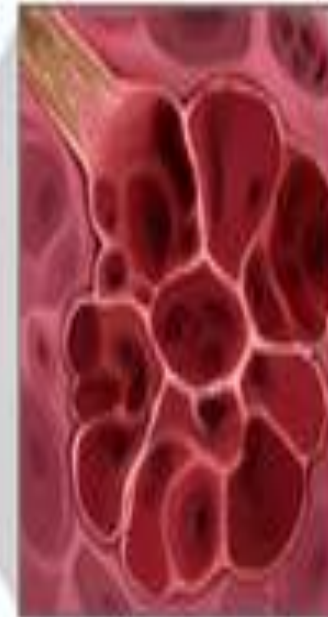
C, PANACINAR (PANLOBULAR) EMPHYSEMA

FIGURE 15.15

The anatomic regions of involvement in an acinus in major forms of emphysema.



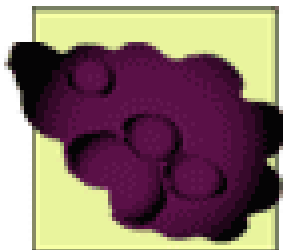
Enlarged view of air sacs (alveoli)



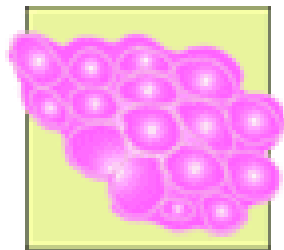
Emphysema:
weakened and
collapsed air
sacs with
excess mucus



Normal
healthy
air sacs



Alveoli with
emphysema

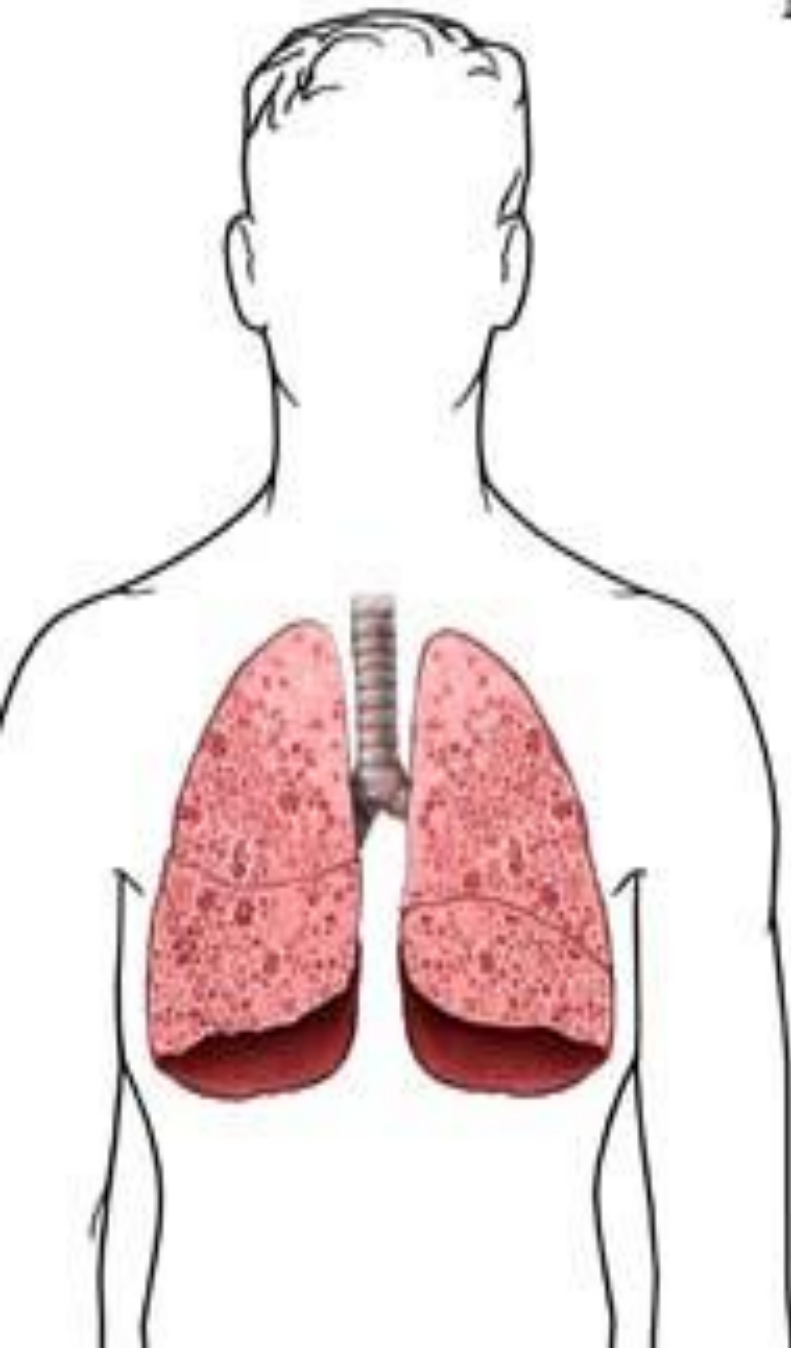


normal alveoli

Normal bronchiole
and alveoli



Emphysema

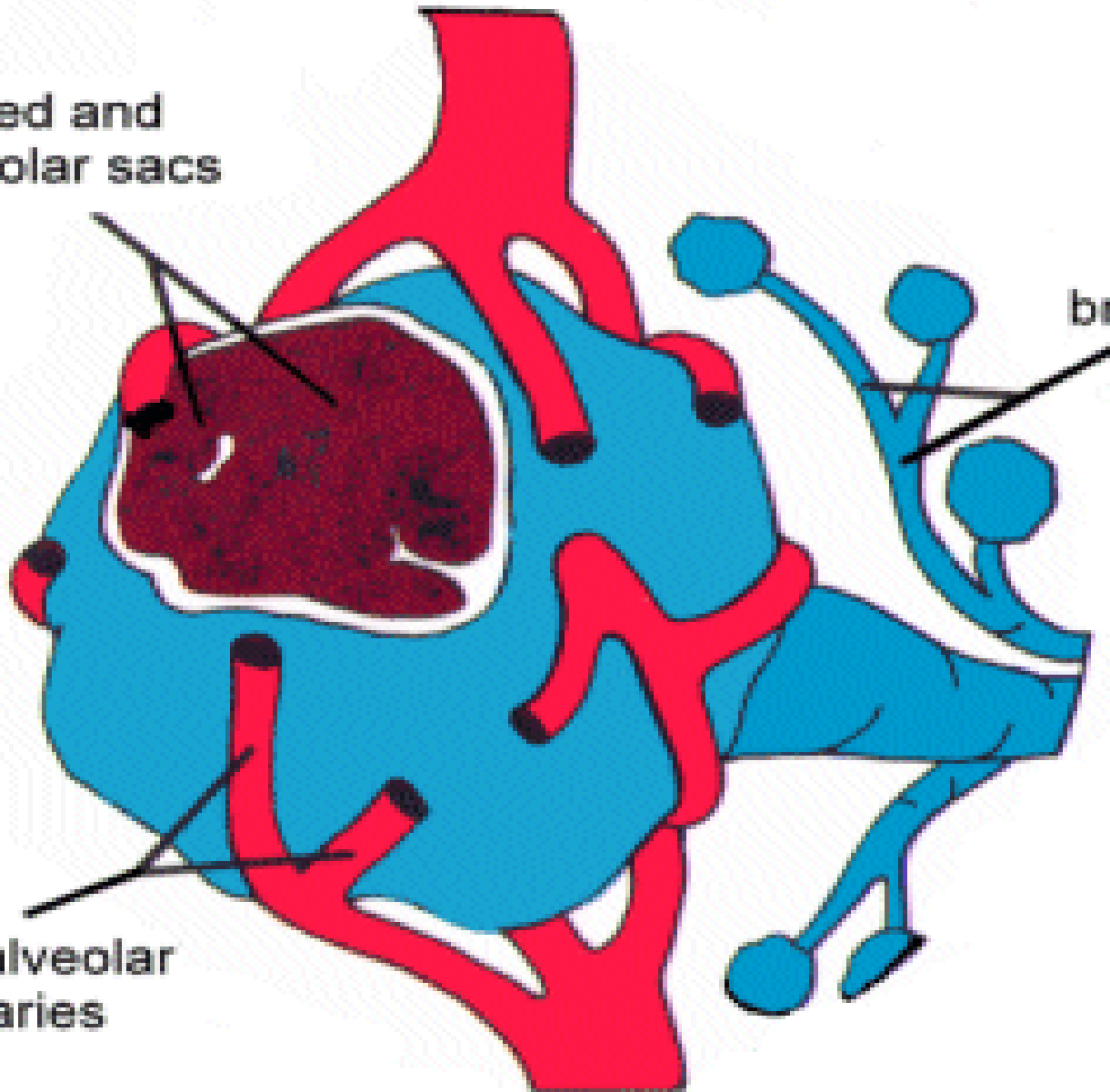


Emphysema

overinflated and
broken alveolar sacs

bronchioles

broken alveolar
capillaries



Etiopathogenesis



"I used to smoke heroin until my emphysema got really bad."

- Tobacco smoking /air pollutant
- Other less imp. – occupational exposure infection
- Familial and genetic influences.
- Destruction of alv. Walls – closely related to deficiency of serum alpha-1 antitrypsin (alpha 1-protease inhibitor) termed as protease-antiprotease hypothesis.

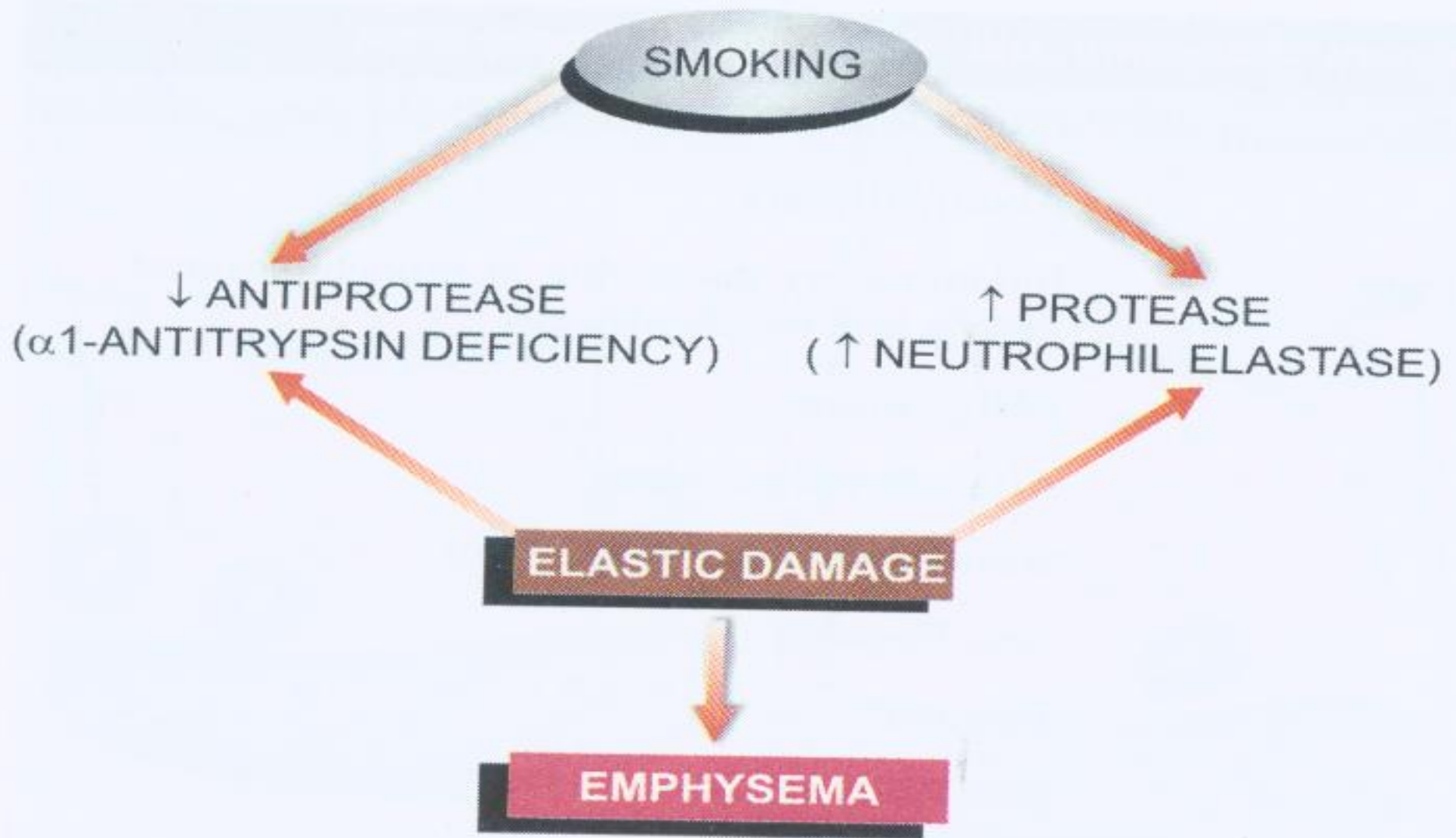


FIGURE 15.13

Pathogenesis of alveolar wall destruction in emphysema by protease-antiprotease mechanism.



Alpha-1-antitrypsin

- Glycoprotein-Located on long arm of chromosome – 14 which codes for Ig light chains.
- alpha 1 glob fraction of plasma protein(electrophoresis)
- Synthesized by liver and distributed in circulating blood, tissue fluid and macrophages
- Function – to inhibit proteases hence name. Proteases derived from neutrophils.
- Neutrophil elastase has capability of digesting lung parenchyma but is inhibited from doing so by alpha-1 antitrypsin.



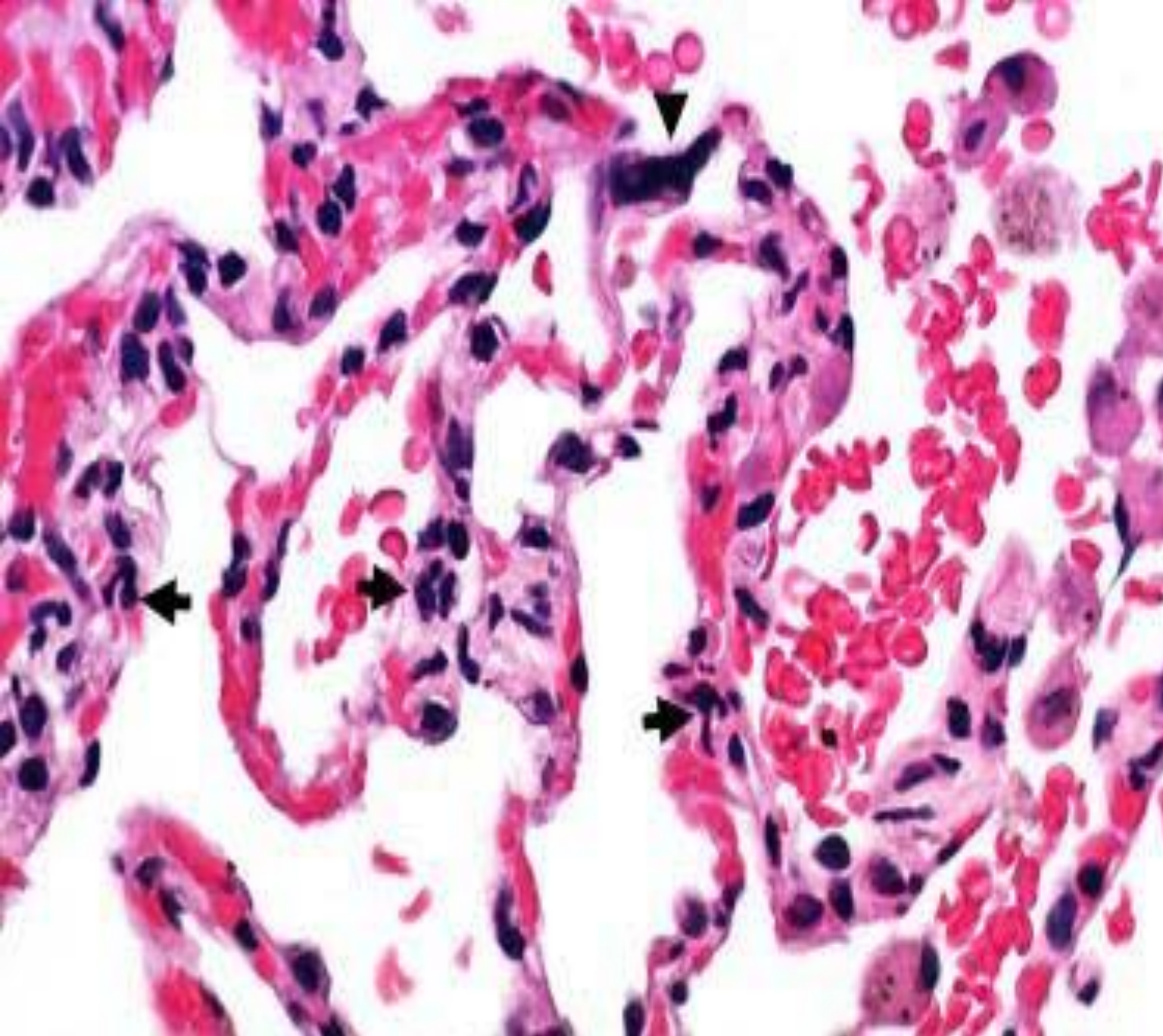
Alpha-1-antitrypsin

- Deficiency of alpha 1 antitrypsin develops in homozygous state due to genetically abn. Locus for protease inhibit(Pi) (N) phenotype of individual in homozy is called Pi22. Heterozygote pattern of P1M2 is not sufficient to produce clinical deficiency, but heterozygot individual who smoke heavily have higher risk of developing emphysema.
- alpha 1 antitrypsin def. Develops in adults cause pul.emp. In smokers as well as non smokers.
- Other organ is liver – develop obst jaundice early in infancy.
- Cirrhosis and hepatoma – adult.



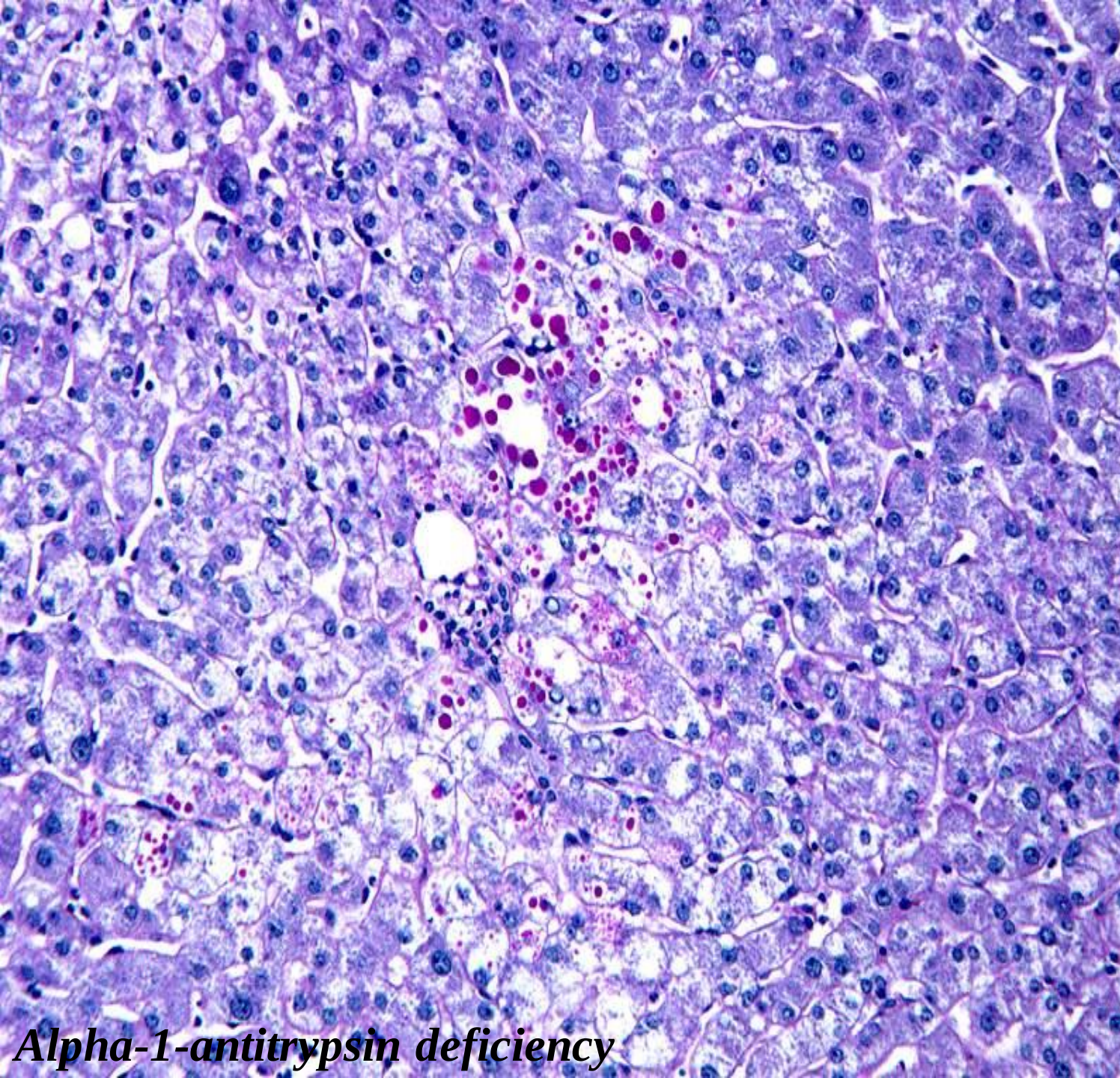
Alpha-1-antitrypsin

- Mechanism – alv. Wall destruct by elastolytic action is based on imbalance between protease and anti protease.
- By decreased anti elastase activity I.e. def. Of alpha 1 ant
- By increase activity of elastase I.e. | PMN inf. In lung causing destruct of wall.
- Enough evidence that smoking promotes.
- Oxidant in smoke has inhibitory influence on alpha 1 ant.
- Smokers have more macrophages, phagocytes and PMN in lungs than nonsmokers.



This photo is taken from a patient with alpha-1-antitrypsin deficiency-related emphysema. It shows an increased number of PMNs in capillaries (arrows and many more) and some alveolar and interstitial blood. These PMNs, if activated, can potentially digest the collagen and elastin of the alveolar wall. Note the megakaryocyte in a capillary (arrowhead at top). Its function is not known, but any contribution of platelets would help to preserve the integrity of the capillary bed.

Pathogenesis of emphysema--proposed



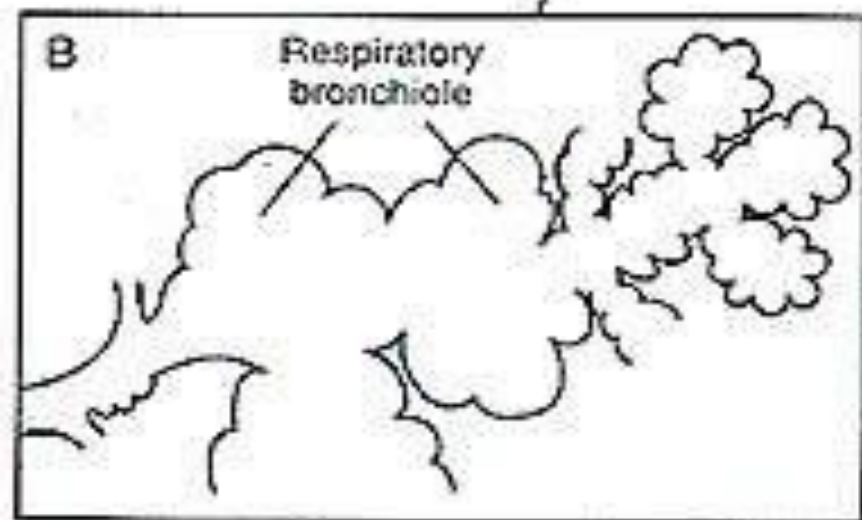
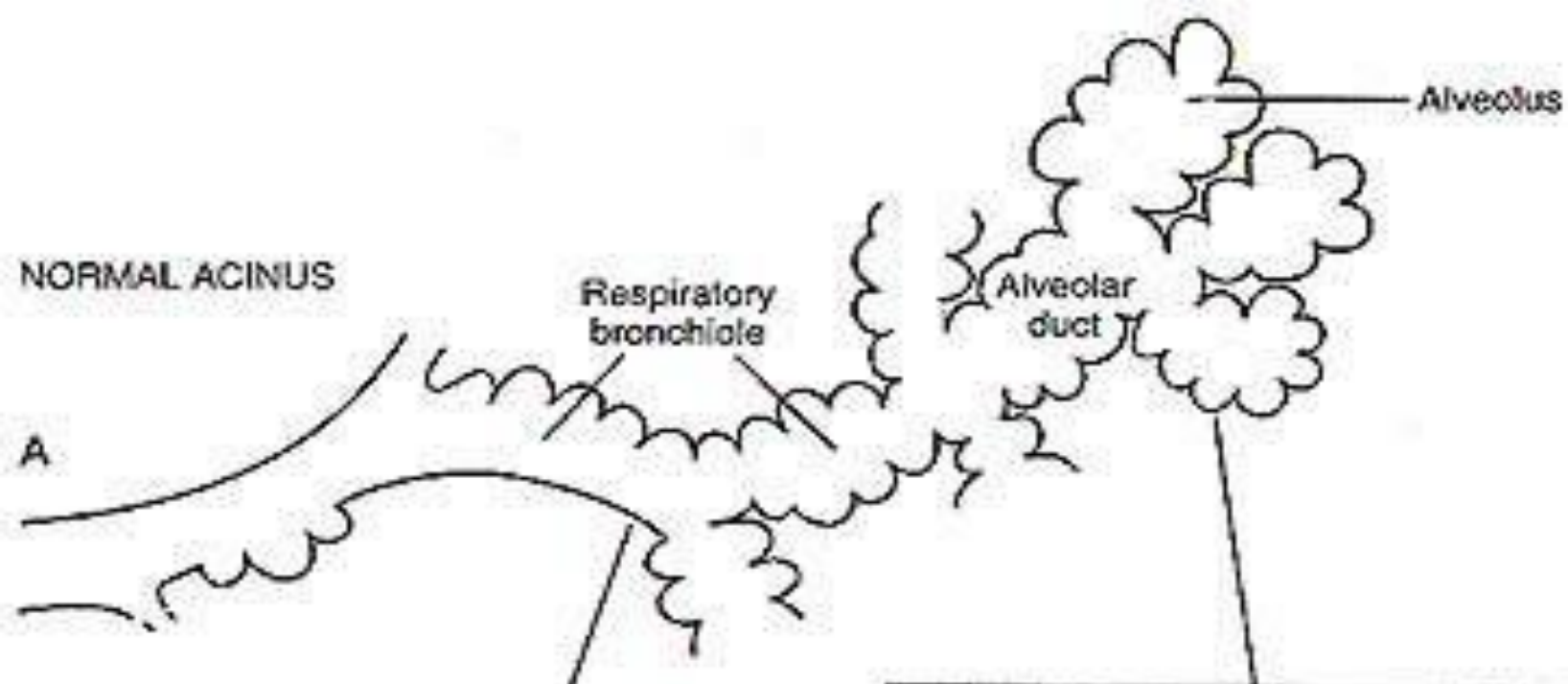
PAS stained section of liver below show red globular inclusions in hepatocytes. These globules are accumulations of alpha-1-antitrypsin. There may be slight increase of fibrous tissue in portal areas.

Alpha-1-antitrypsin deficiency

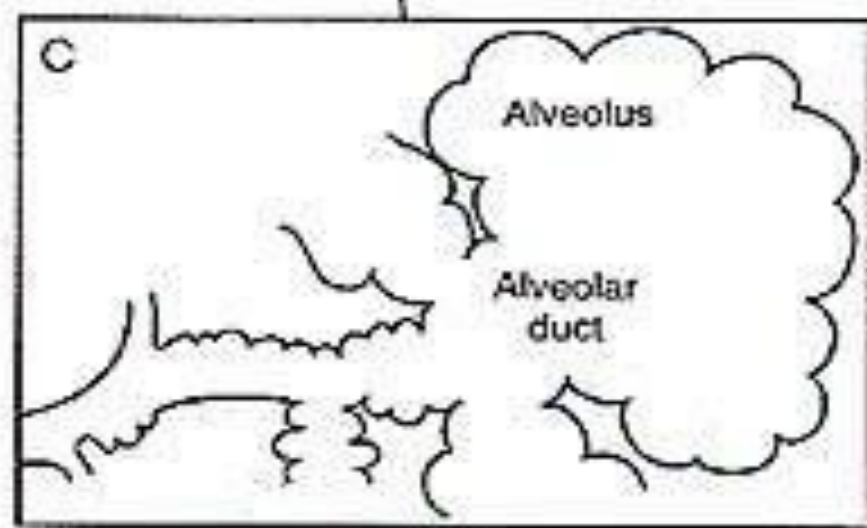


EMPHYSEMA-Classification

- **WHO**-according to *protion of acinars involved*
- I. Centriacinar
- II. Panacinar (panlobular)
- III. Para septal (distal acinar)
- IV. Irregular Para cicatricial
- V. Mixed (unclassified)



Centrilobular emphysema



Panacinar emphysema



PATHOLOGICAL CHANGES

Gross

- Lungs perfused with formalin under pressure in inflated state to grade the severity with naked eye.
- lungs are voluminous, pale with little blood.
- Edges are rounded.
 - Dilatations of air spaces visible with hand lens
- Advanced cases show subpleural bullae and blebs bulging outwards from surface of lungs with rib marking.
- Bullae – air filled cysts-like or bubble-like, Longer than 1 cm in dia. Formed by rupture of airspaces.
- Blebs-due to rupture of alveoli directly into subpleural interstitial tissue common cause of pneumothorax.

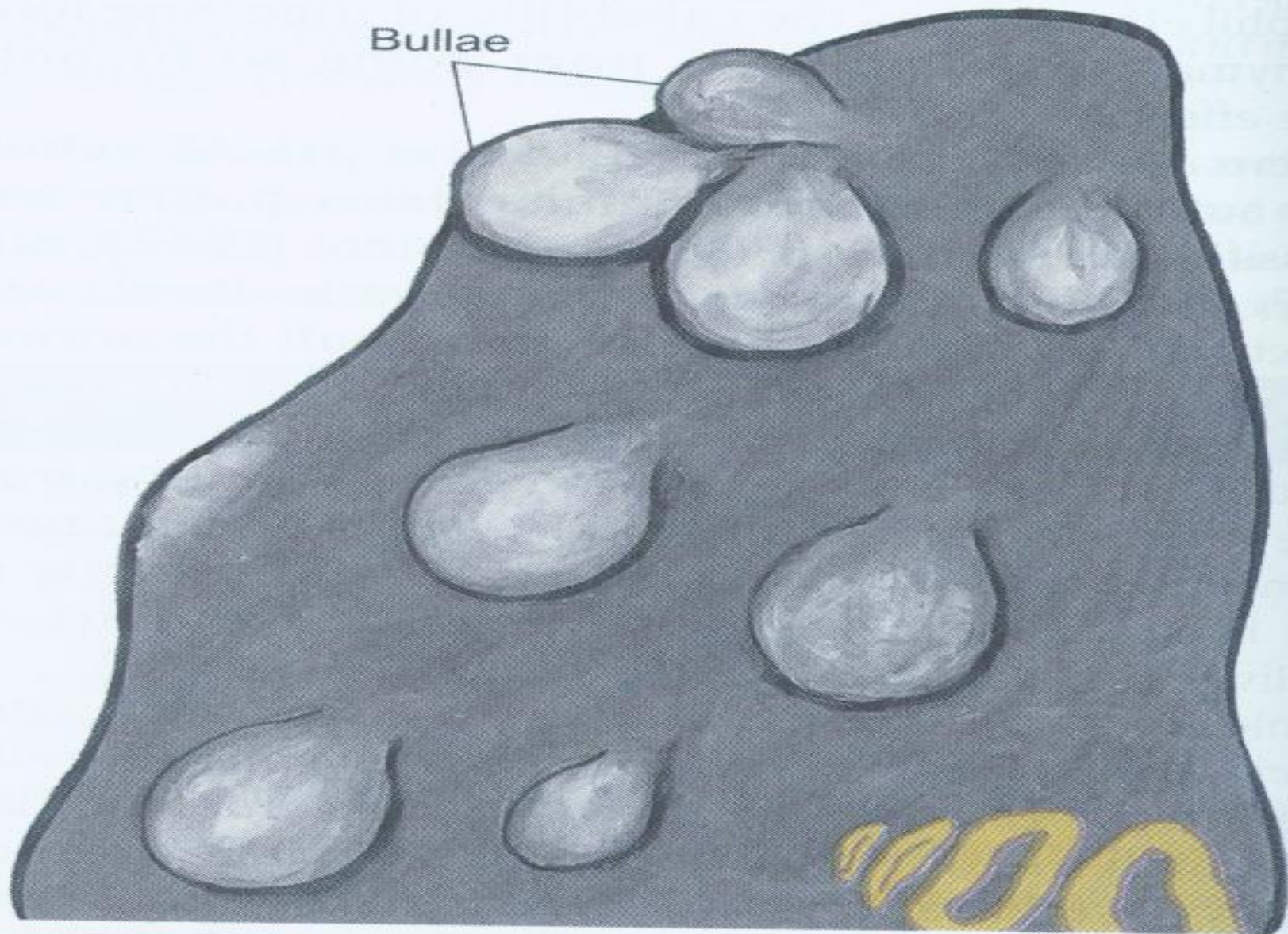


FIGURE 15.14

Bullous emphysema as seen on the external surface of the lung.

Emphysema





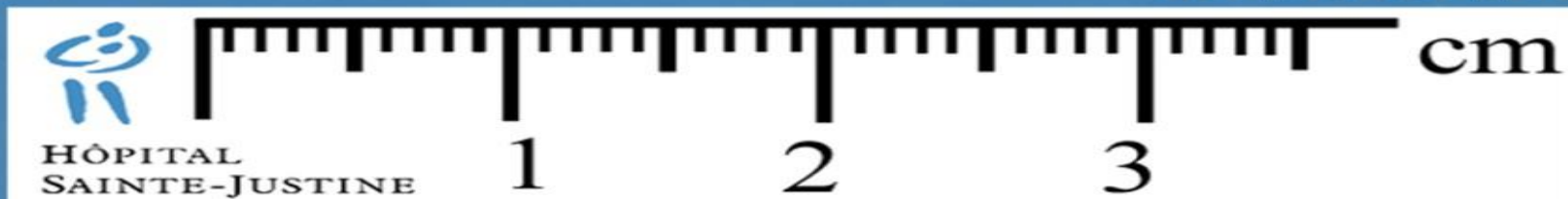
Emphysema is an enlargement of air spaces caused by destruction of alveolar walls. Air spaces greater than one cm are bullae. This photo shows apical bullous disease with relatively little involvement of the rest of the lung.



Congenital lobar emphysema



Normal control





Emphysema



PATHOLOGICAL CHANGES(contd.)

Microscopy(depending upon type of emphysema)

- Dilatation of air spaces
- Destruction of septal walls of part of acini i.e. resp. bron, alv.ducts and alv.sacs
- Changes of bronchitis
 -
- Bullae and blebs show fibrosis
- Chronic inf. of wall.



Alveoli with
emphysema



Microscopic view
of normal alveoli





Clinical features

- Long h/o slowly increasing severe excretional dyspnoea
- Pt. Is quite distressed with obvious use of accessory muscles of respiration
- Chest baned shaped and hyper resonant
- Cough ass with scanty mucoid sputum
- Pink puffers pt. Remains well oxygenated have tachypnoea.
- Wt. Loss is common
- Rt. Heart failure (cor pulmonale) and hypercapneic resp. failure-terminal event
- Xray chest small heart with hyperinflated lungs.

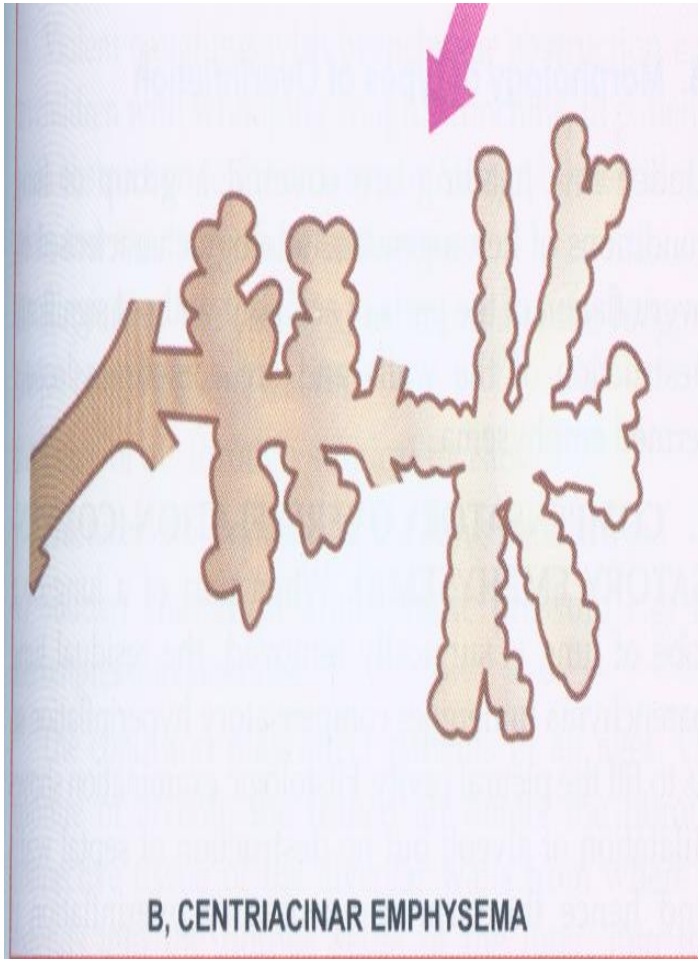


Individual Types

Centriacinar (centrilobular)

- Most common
- Involvement of resp. br. i.e. the central or proximal part of acinus.
- Co-exists with chr. Br.
- Occurs predom. In smoker and in coal miners pneumonia.

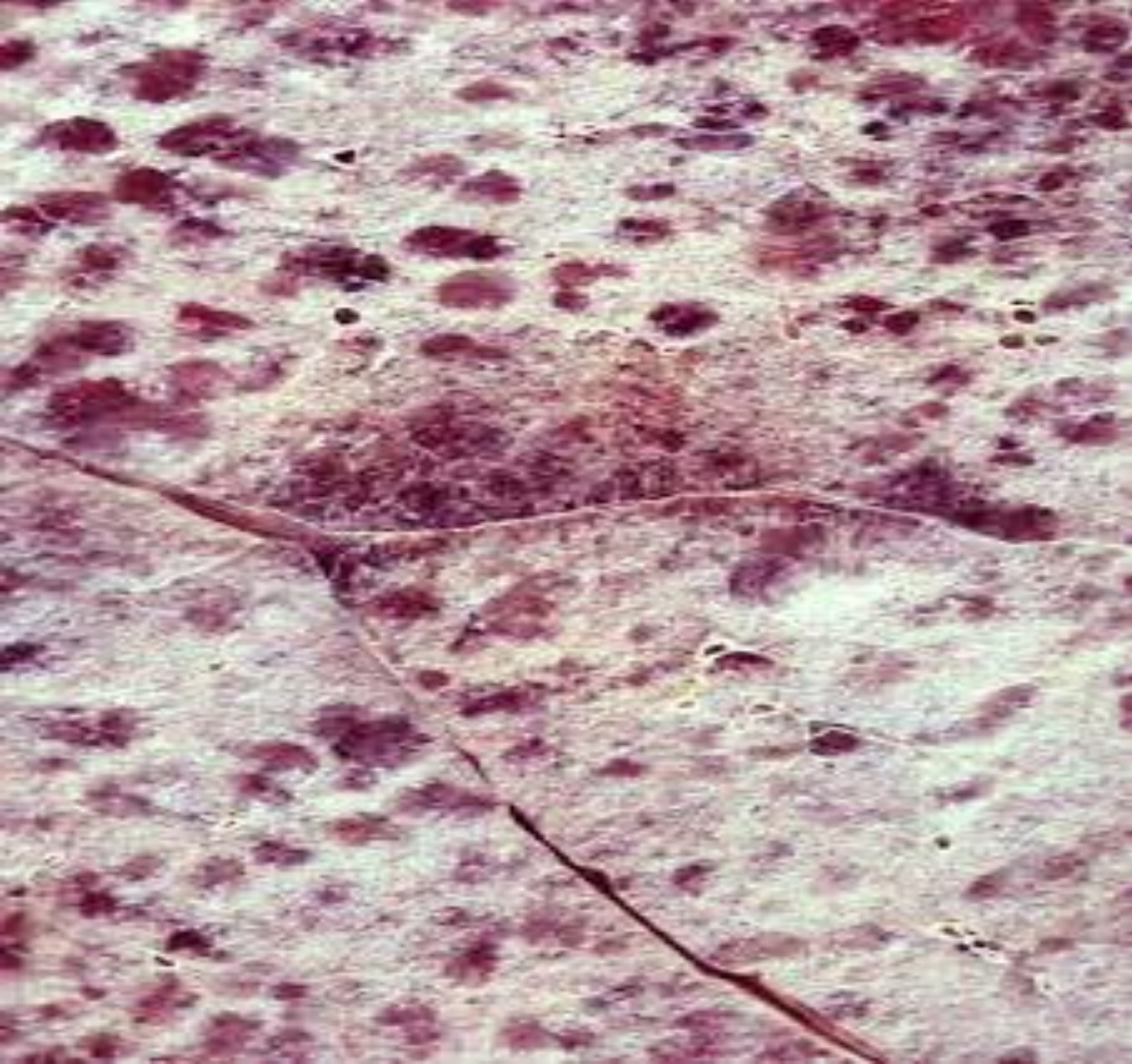
Centriacinar(contd.)



Gross – Upper lobes – common and severe.

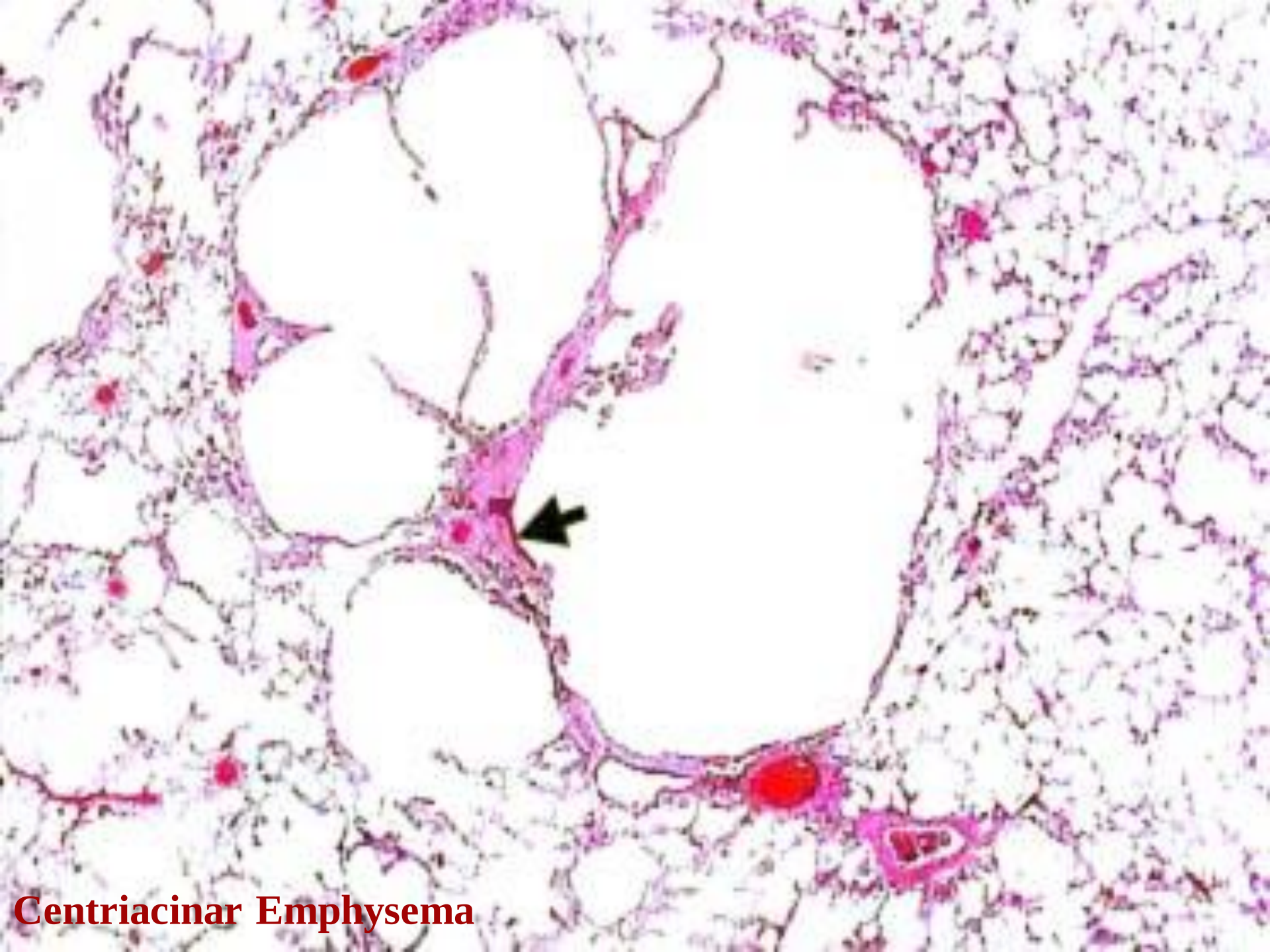
C/S distended air spaces in the center of lobules surrounded by (N) lung parenchyma. Lobules separated by fine fibrous tissue septae. Large amount of black pig.

Micro-distention & destruction of RB in centre of lobules – surrounding is (N) lung alveoli. Terminal bronchiole show chronic infl. and narrowing.

A low-magnification photomicrograph of lung tissue stained with hematoxylin and eosin (H&E). The image shows a large, irregularly shaped air space (alveolus) in the lower half, which is relatively clear, indicating distal acinar emphysema. The surrounding lung parenchyma is densely packed with smaller alveoli. In the upper half, there are several small, dark, circular holes (bronchioles) surrounded by a thickened wall of alveolar tissue, characteristic of centriacinar emphysema. A prominent, dark, branching structure, likely a blood vessel or bronchiole, runs diagonally across the lower half of the image.

Centriacinar emphysema/ distal acinar emphysema

- This lung shows centriacinar emphysema with holes located around the terminal bronchioles and preservation of more distal air spaces. The parenchyma along the major fissure shows some distal acinar emphysema. Panacinar emphysema involves the entire acinus.



Centriacinar Emphysema



Panacinar(Panlobular)

- All portions of acinus affected
- Ass. with alpha-1 antitrypsin def.
- In middle aged smokers .



Panacinar (contd.)

Gross-Lower zones of lungs in contrast to centrilobular

- may be confined to a few lobules or part of a lobe of lung.
- Lungs are enlarged and overinflated.

Micro.- all portions of acini are distended.

- RB, Al. duct, alv. Dilated walls are stretched and thin.
- Ruptured alv. Walls and spurs of broken septa are seen bet the adjacent alveoli.
- Capill. Are stretched and thin
- Sp. Stain – loss of elastic tissue
- Infl. Changes – absent



Paraseptal(Distal-acinar)

- Involves distal part of acinus and proximal part is normal
- localised along pleura and perilobular septa.
- Involvement is seen adjacent to areas of fibrosis and atelectasis.
- Involv. Upp. Part of lung > than lower
- Causes spontaneous pneumothorax in young.
- **Grossly**, subpleural portion of lung show airfilled cystic 0.5 to 2 cm in dia



Irregular (Para-cicatricial)

- Surrounding scar from any cause.
- Irregular as regard the portion of acinus involved as well as within lung as a whole.
- During life- asymptomatic
- Incidental finding at autopsy.



Mixed(unclassified)

- Same lung show more than one type.
- Lungs of elderly smokers may show continuation of centriacinar emp in upp. Lobes, panacinar in lower lobes and paraseptal in subpleural region

TABLE 15.5: Contrasting Salient Features of 'Predominant Bronchitis' and 'Predominant Emphysema'.

FEATURE	PREDOMINANT BRONCHITIS	PREDOMINANT EMPHYSEMA
<i>Age at diagnosis</i>	About 50 years	About 60 years
<i>Underlying pathology</i>	Hypertrophy of mucus-producing cells	Inflammatory narrowing of bronchioles and destruction of septal walls
<i>Dyspnoea</i>	Late, mild	Early, severe
<i>Cough</i>	Before dyspnoea starts	After dyspnoea starts
<i>Sputum</i>	Copious, purulent	Scanty, mucoid, less frequent
<i>Bronchial infections</i>	More frequent	Less frequent
<i>Respiratory insufficiency</i>	Repeated	Terminal
<i>Cyanosis</i>	Common ('blue-bloaters')	Rare ('pink-puffers')
<i>Lung capacity</i>	Normal	Increased (barrel-chest)
<i>Blood gas values</i>	$\downarrow$ pO ₂ , $\downarrow$ pCO ₂ , no compensatory hyperventilation	pO ₂ and pCO ₂ usually within normal limits due to compensatory hyperventilation
<i>Cor pulmonale</i>	Frequent	Rare and terminal
<i>Chest X-ray</i>	Large heart, prominent vessels	Small heart, hyperinflated lungs



Thank you.