PATHOLOGY LAB

Maram Abdaljaleel, MD

PATHOGENESIS

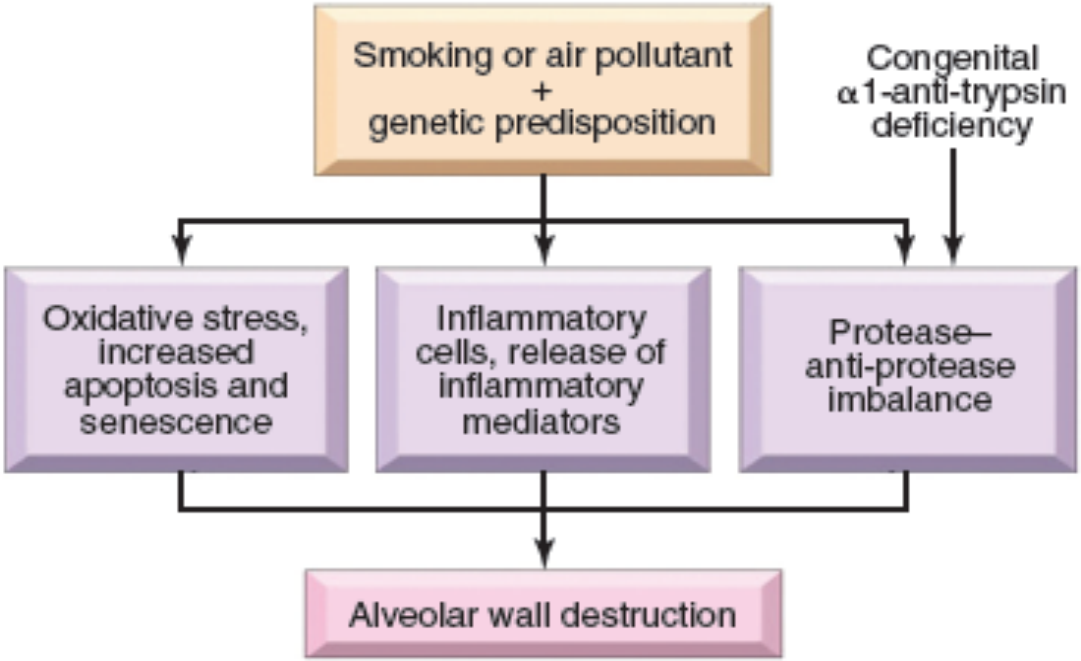
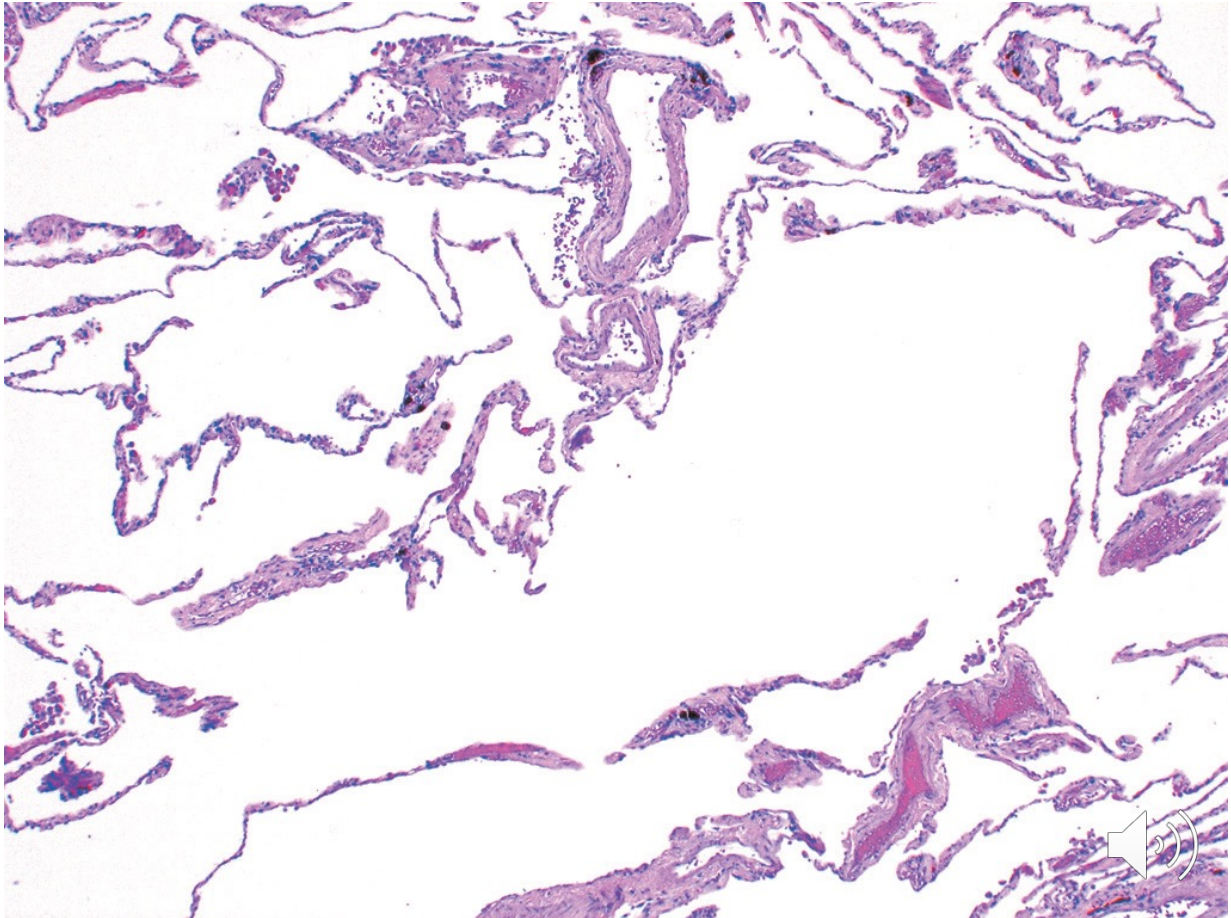


Fig. 13.6 Pathogenesis of emphysema. See text for details.

,ROBBINS BASIC PATHOLOGY, 10TH EDITION



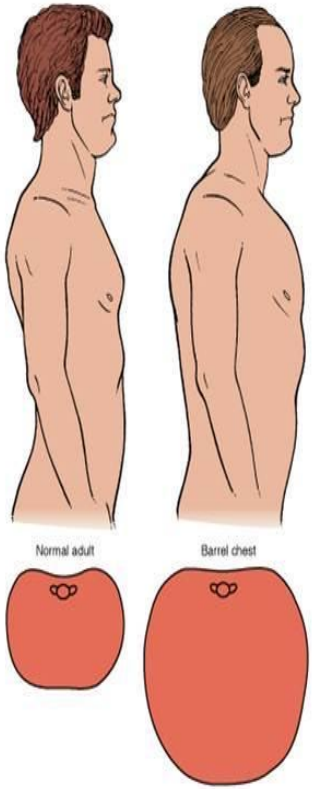


Figure 25-31 Profile and anteroposterior diameter of normal adult chest and barrel chest.

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

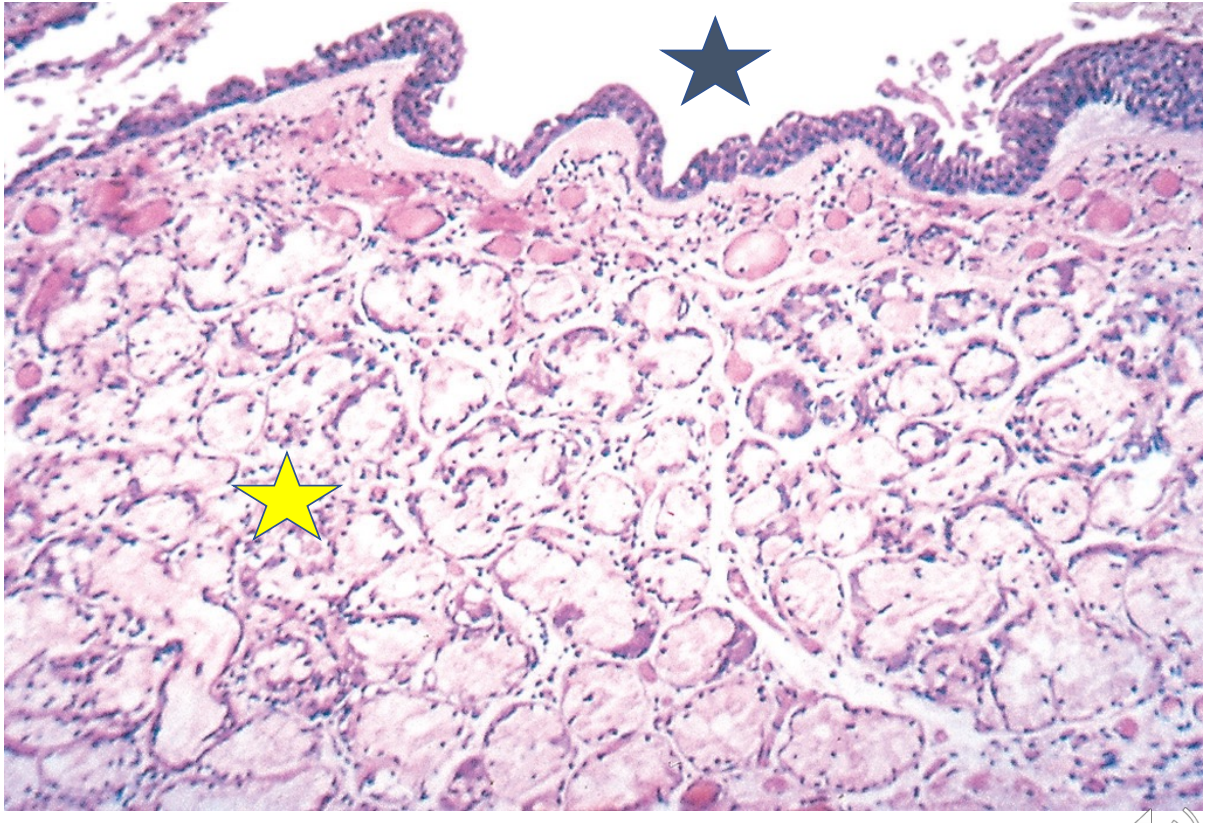
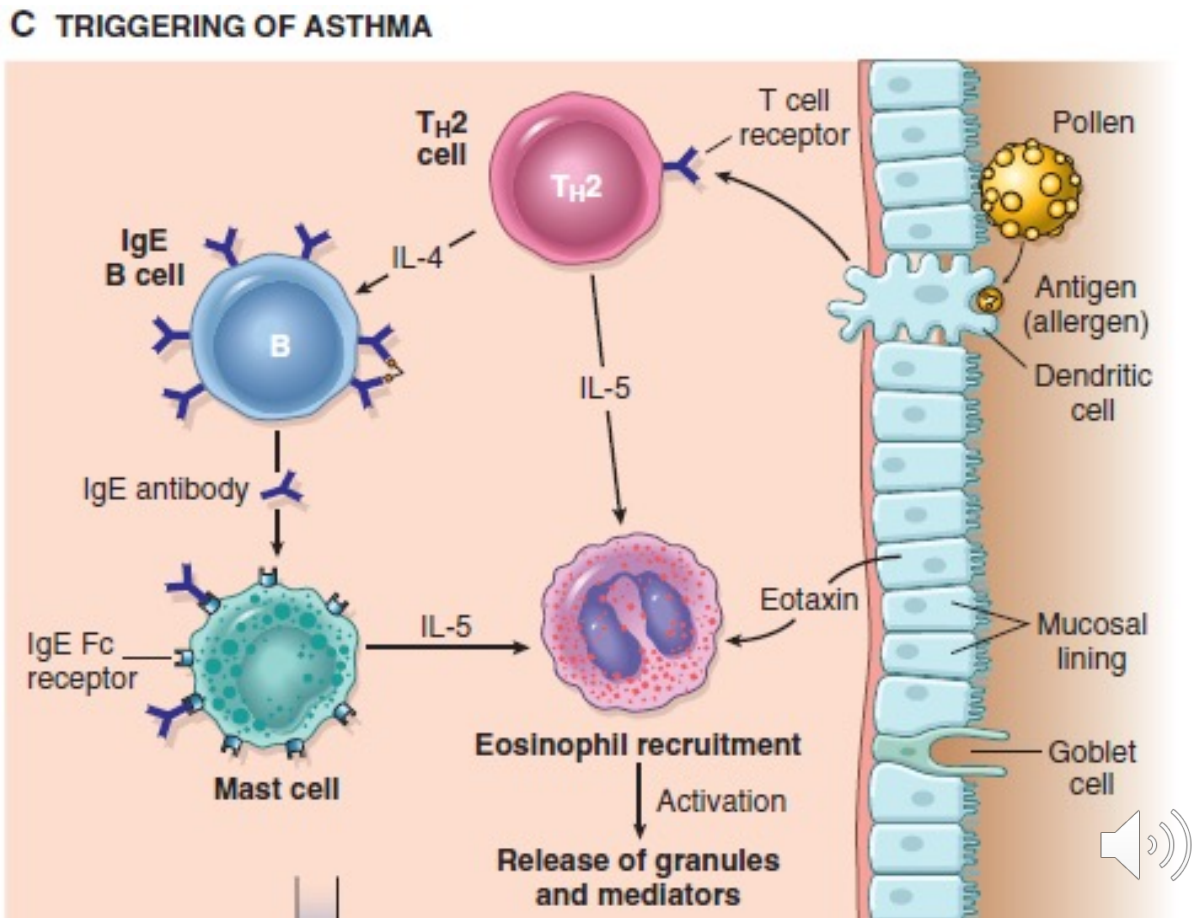
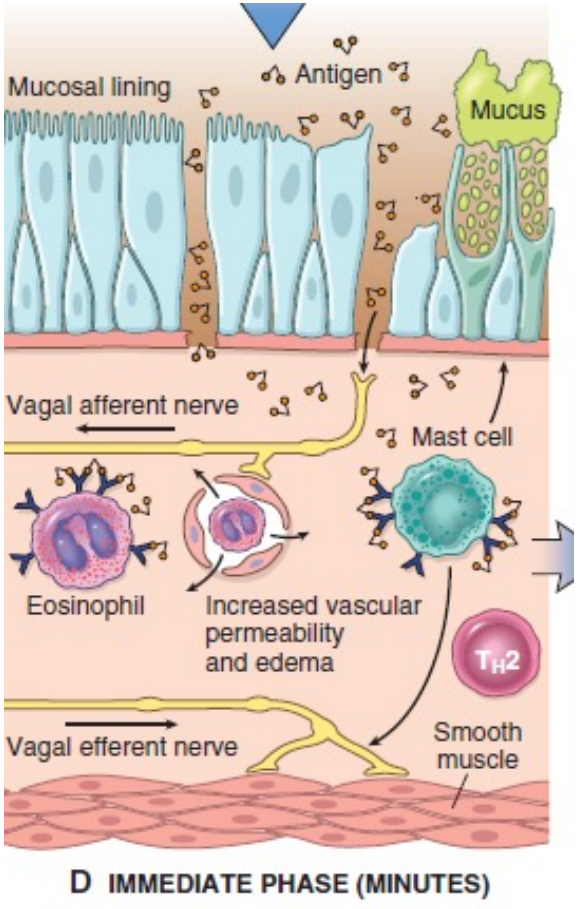


Fig. 13.9 Chronic bronchitis. The lumen of the bronchus is above. Note the marked thickening of t
mucous gland layer (approximately twice-normal) and squamous metaplasia of lung epithelium. *(From the
Teaching Collection of the Department of Pathology, University of Texas, Southwestern Medical School,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


C TRIGGERING OF ASTHMA
T_H2 cell
T cell receptor
Pollen
IgE B cell
IL-4
Antigen (allergen)
Dendritic cell
IL-5
IgE antibody
Eotaxin
IgE Fc receptor
IL-5
Mucosal lining
Eosinophil recruitment
Goblet cell
Activation
Mast cell
Release of granules and mediators

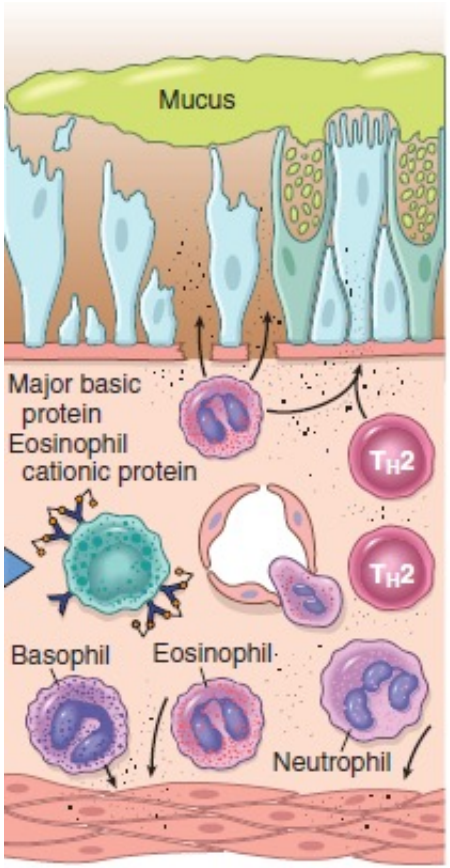


D IMMEDIATE PHASE (MINUTES)

on re-exposure to antigen (ag)→ immediate reaction

triggered by Ag-induced cross-linking of IgE bound to Fc receptors on mast cells.

mast cells release preformed mediators that directly and via neuronal reflexes induce:
bronchospasm,
increased vascular permeability,
mucus production
recruitment of leukocytes

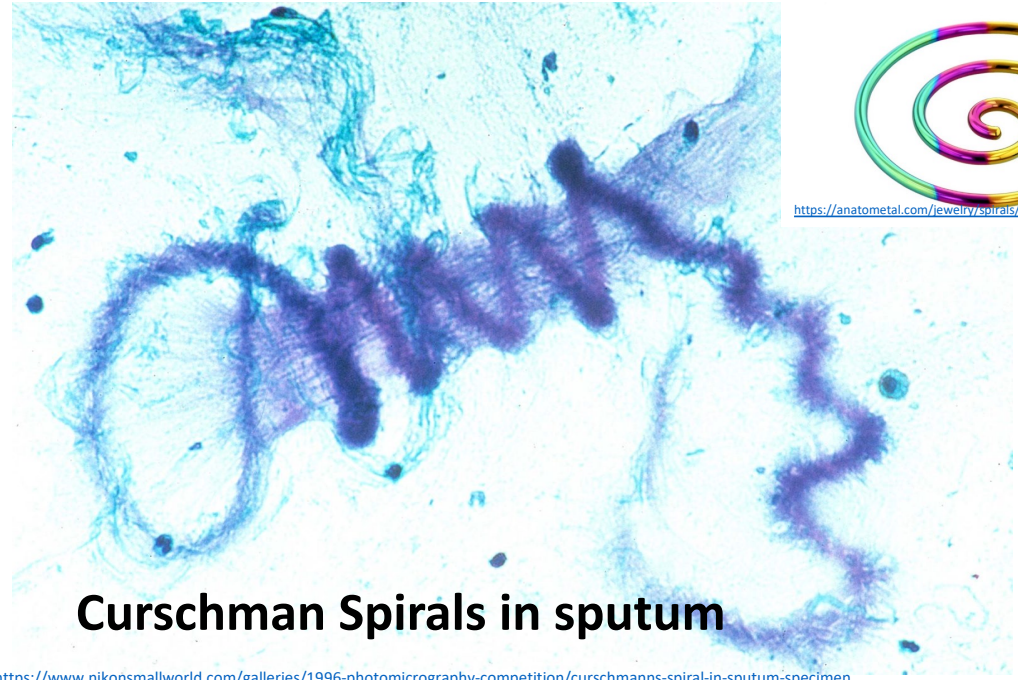


E LATE PHASE (HOURS)

Leukocytes recruited to the site of reaction (neutrophils, eosinophils, and basophils; lymphocytes and monocytes) → release mediators → initiate the late phase of asthma.

eosinophils release major basic protein and eosinophil cationic protein that cause damage to the epithelium

MORPHOLOGY



Curschman Spirals in sputum

https://www.nikonsmallworld.com/galleries/1996-photomicrography-competition/curschmanns-spiral-in-sputum-specimen

https://anatometal.com/jewelry/spirals/

Figure 13.5 ROBBINS BASIC PATHOLOGY, 10TH EDITION

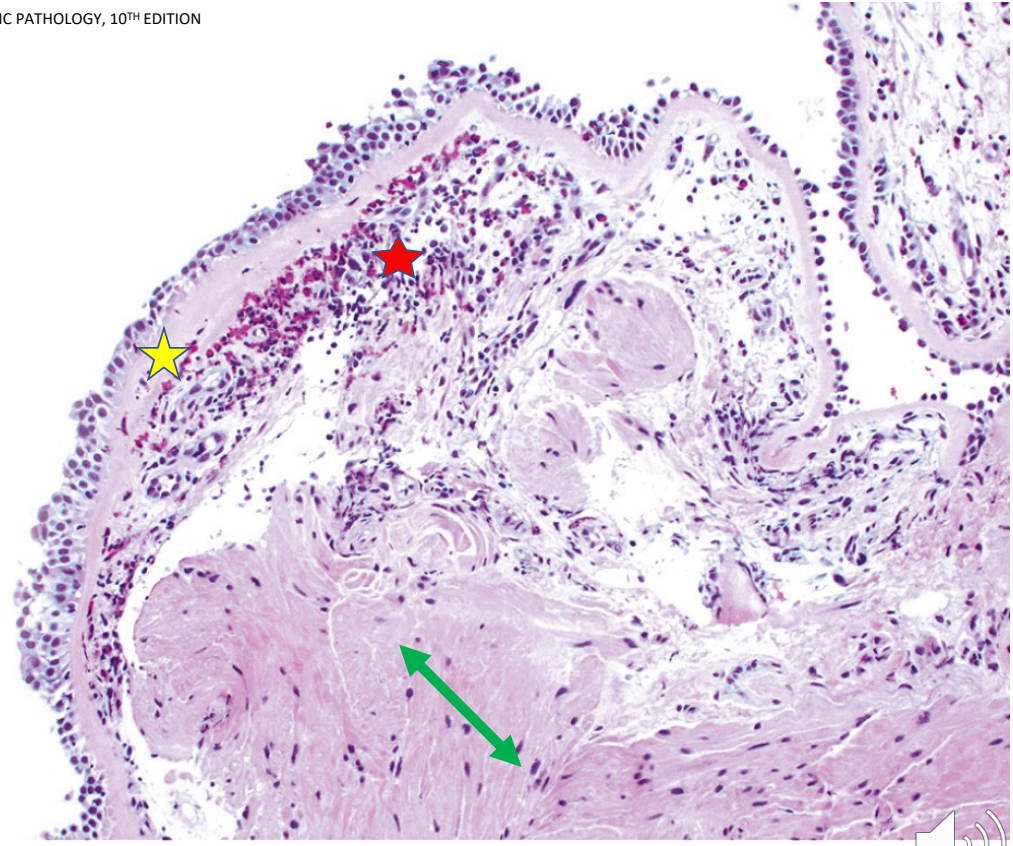
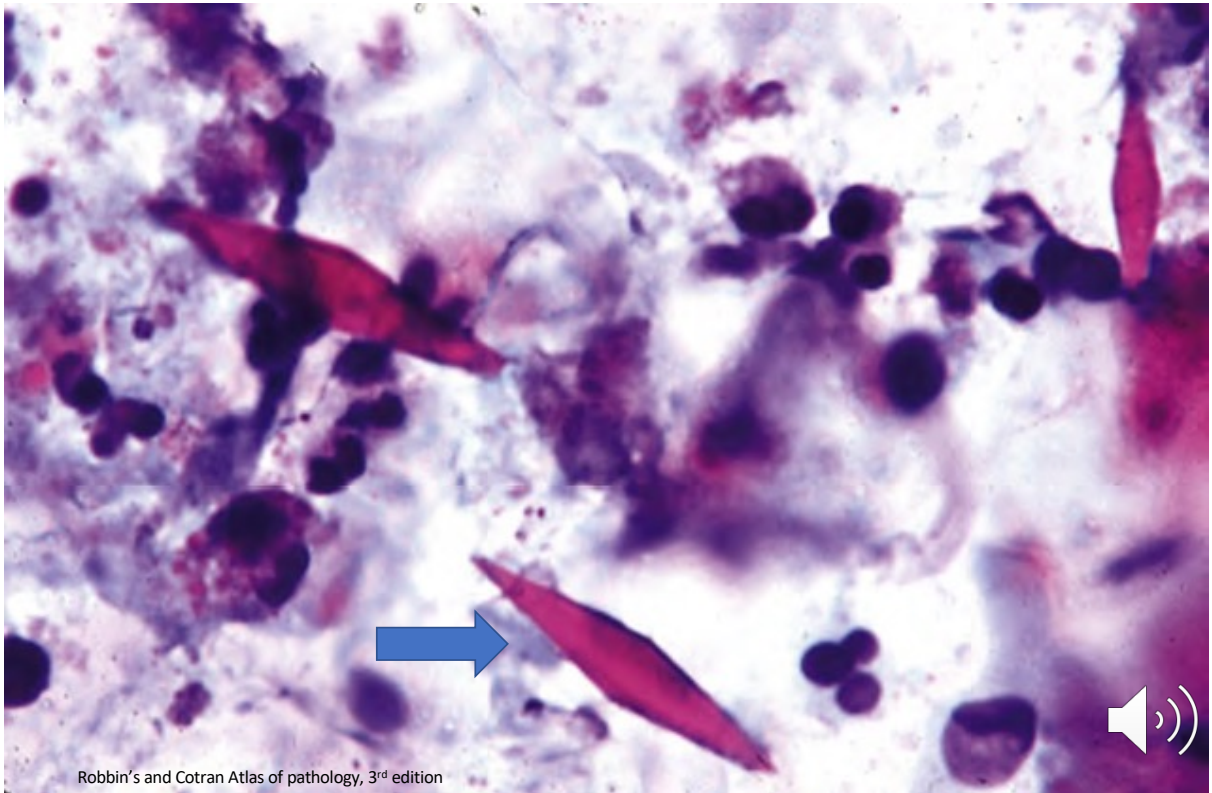
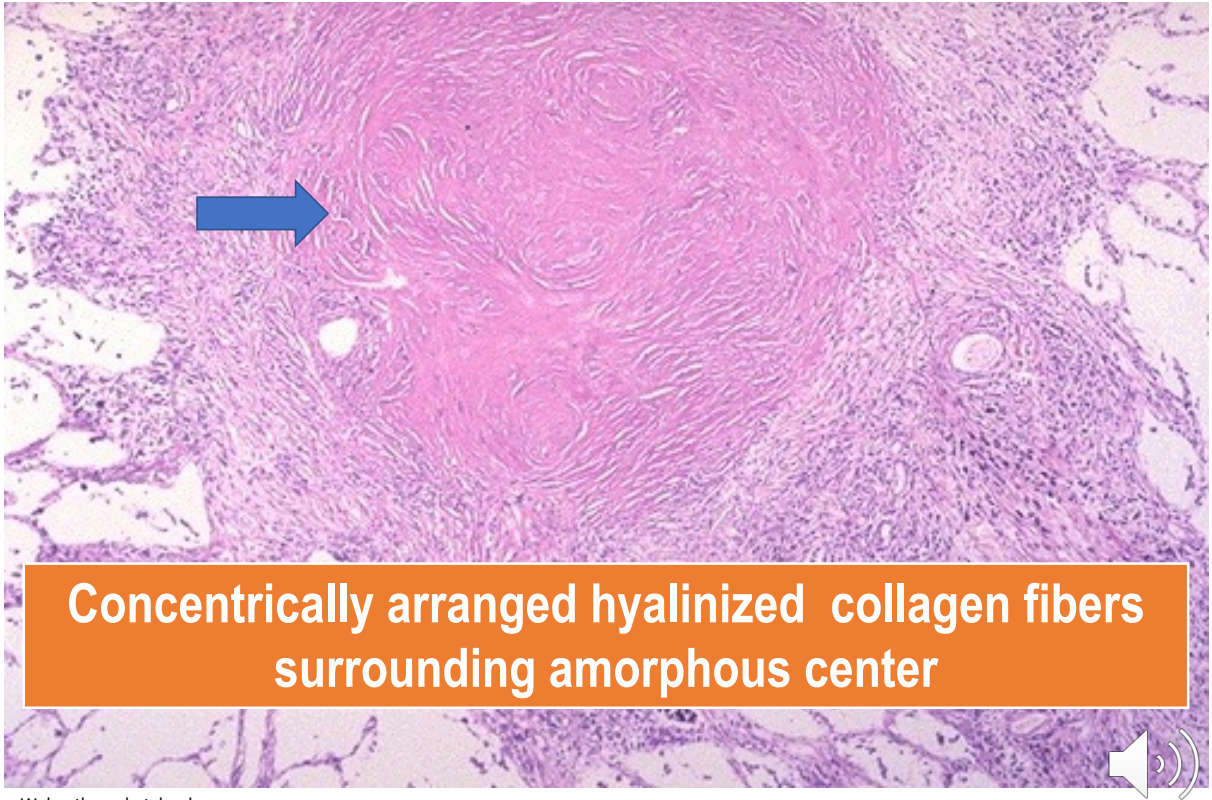


Fig. 13.11 Bronchial biopsy specimen from an asthmatic patient showing sub basement membrane fibrosis, eosinophilic inflammation, and smooth muscle hyperplasia

- Charcot-Leyden crystals: crystalloids made up of the eosinophil protein galectin-10

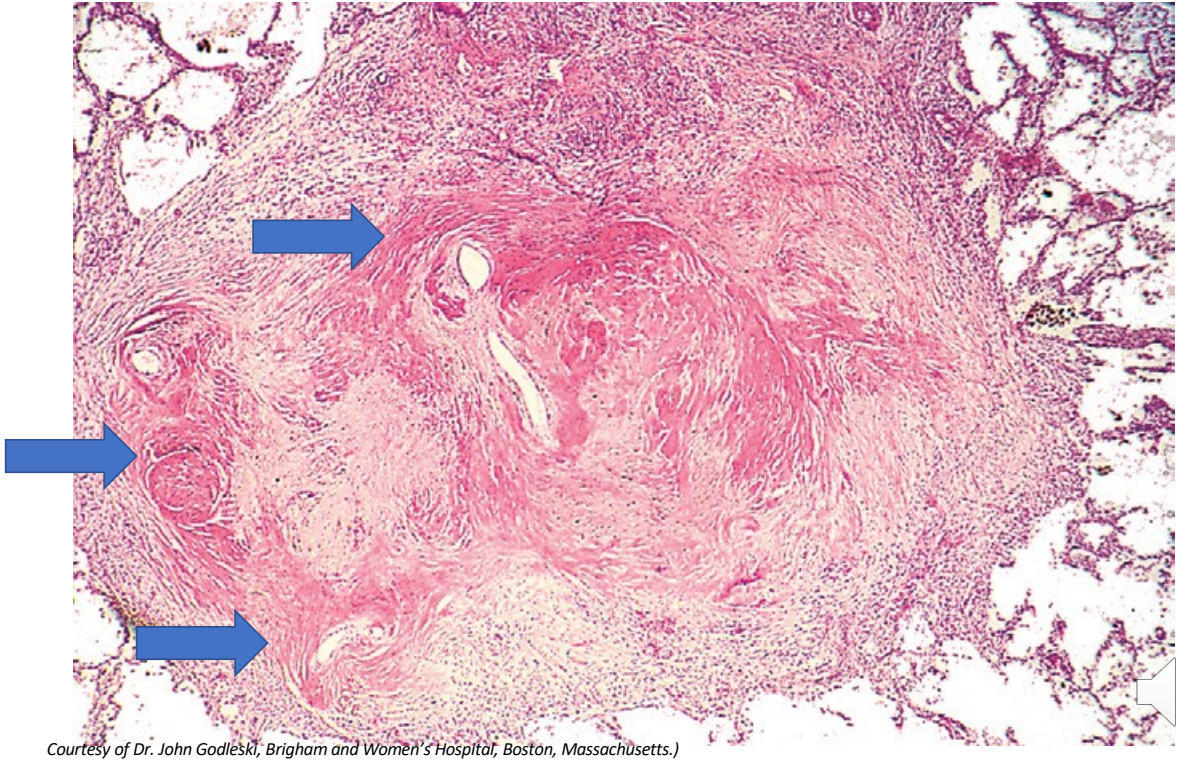


silicotic nodule

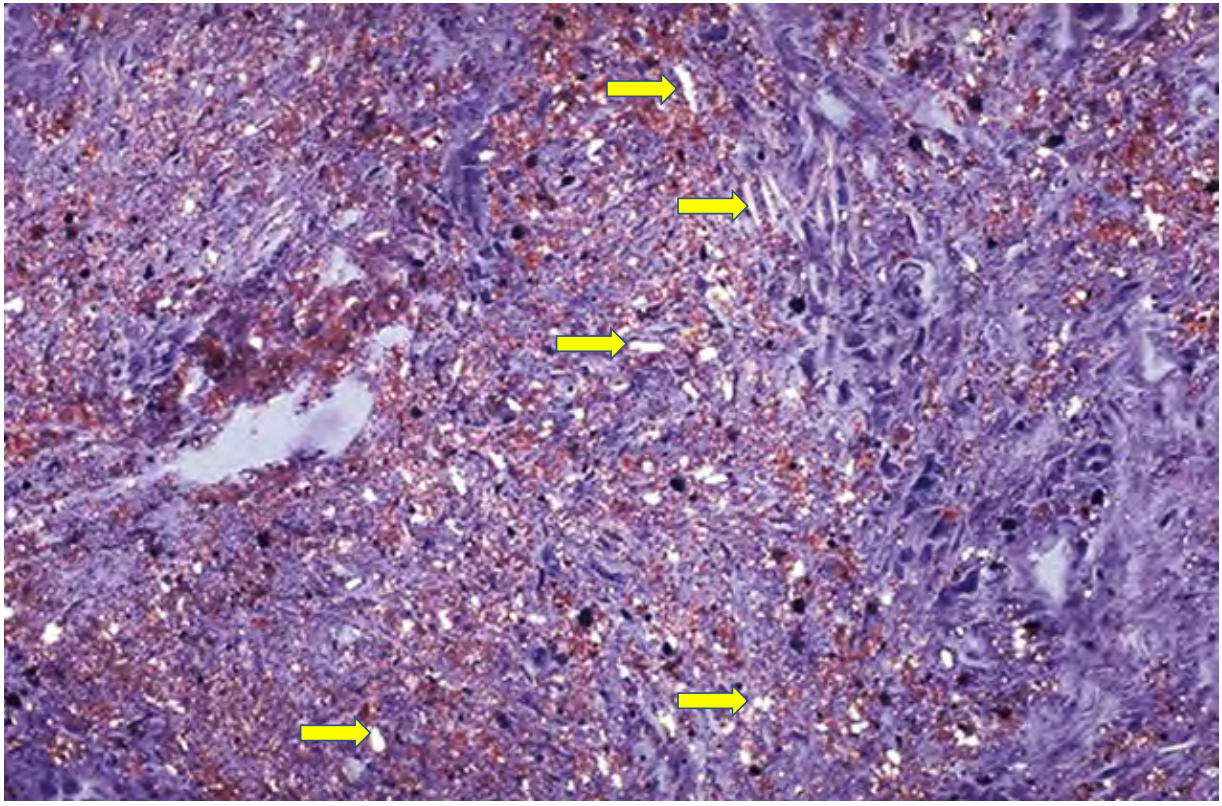


Webpath.med.utah.edu

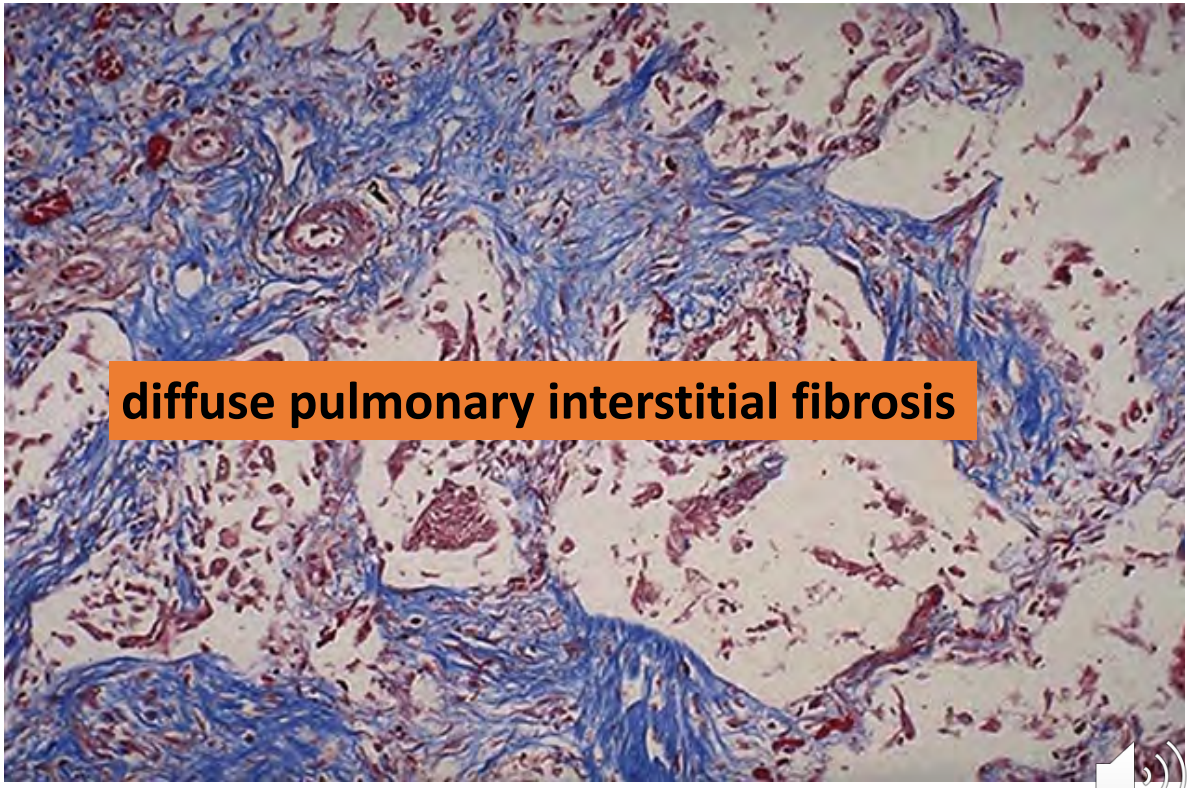
Several coalescent collagenous silicotic nodules



Courtesy of Dr. John Godleski, Brigham and Women's Hospital, Boston, Massachusetts.)

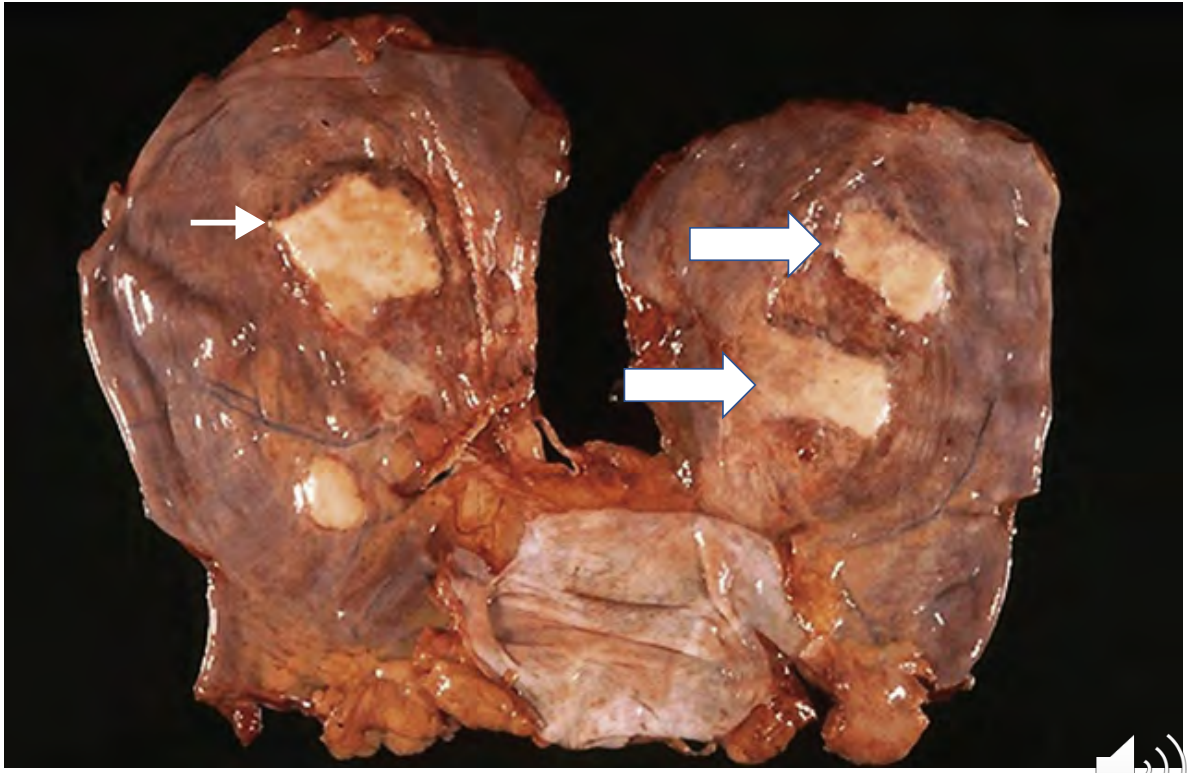


Silica cystals

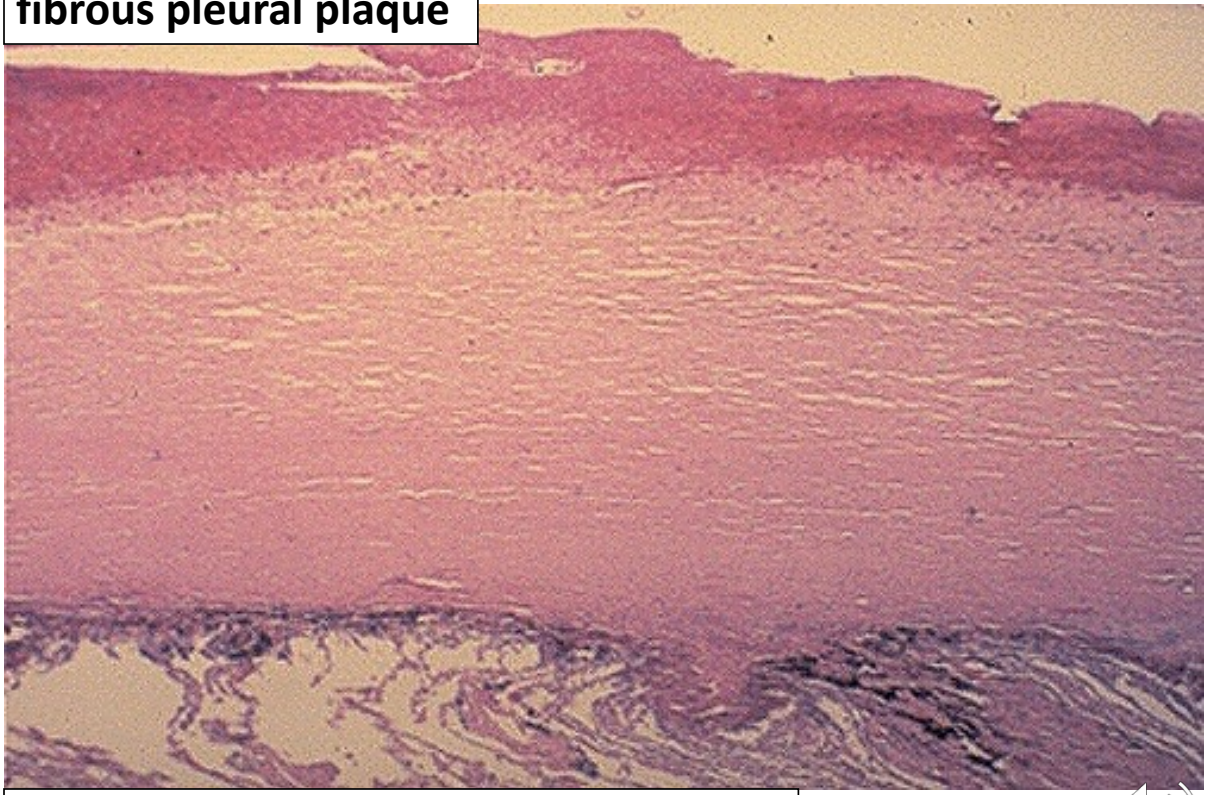

diffuse pulmonary interstitial fibrosis

Asbestos body with beading and knobbed ends

Robbin's Basic pathology, 10 th edition

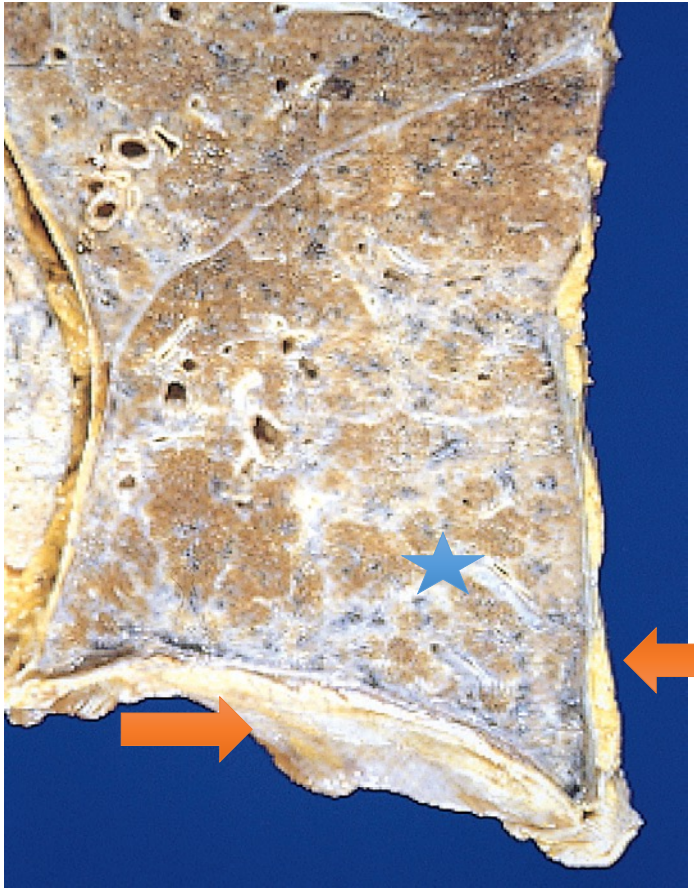


fibrous pleural plaque

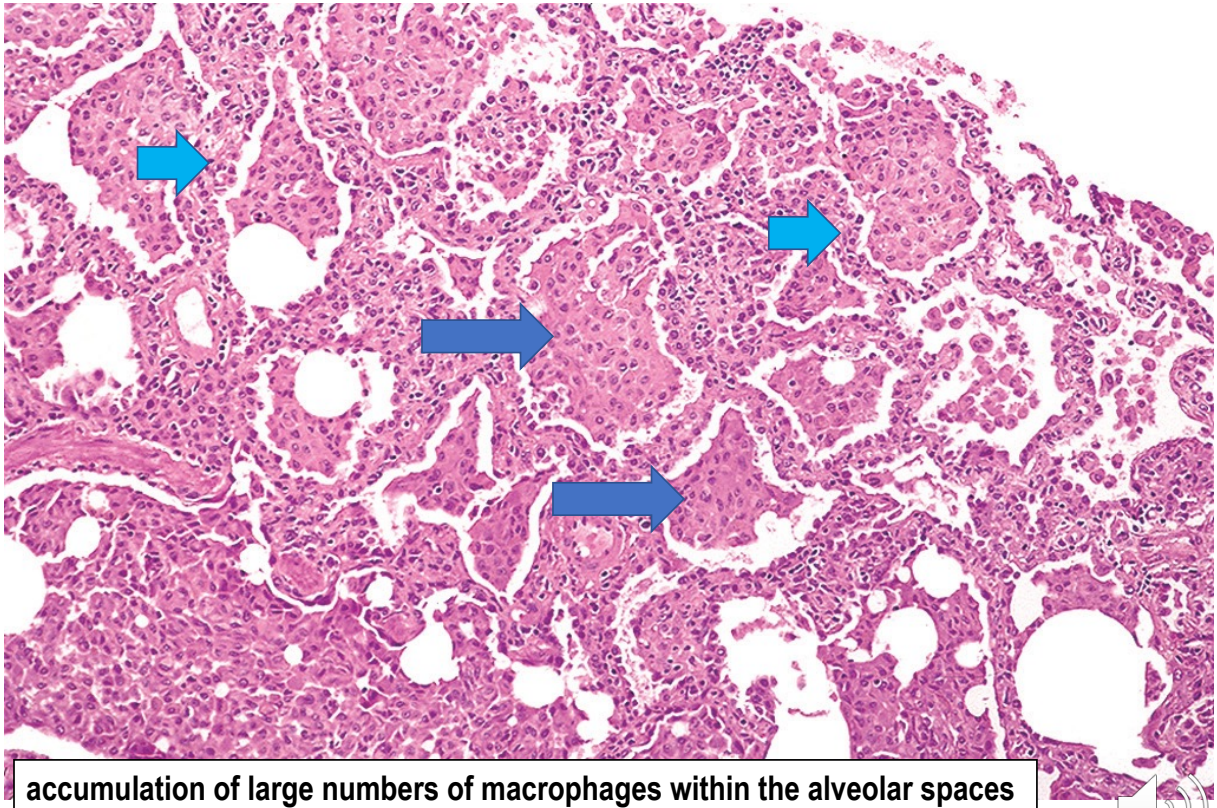


dense laminated layers of collagen (pink)

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



Robbin’s Basic pathology, 10 th edition

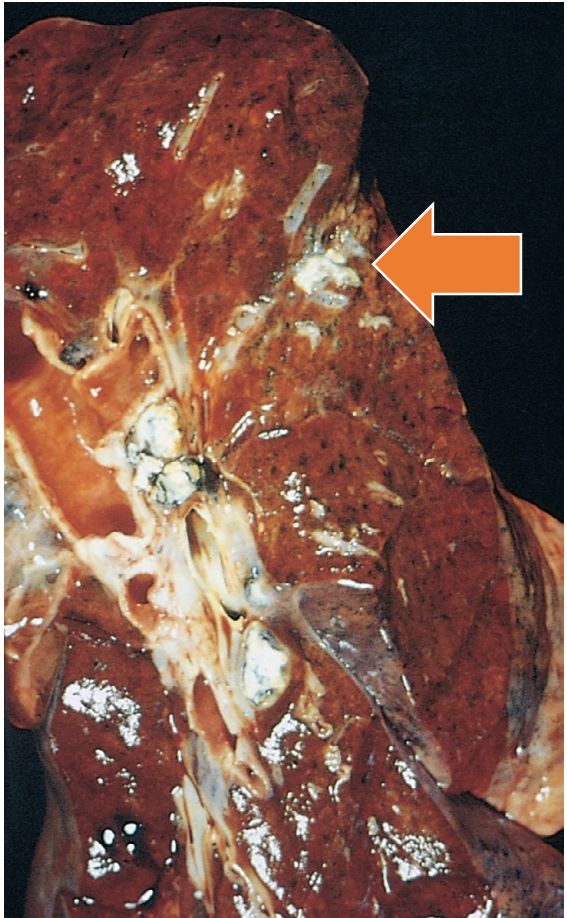


accumulation of large numbers of macrophages within the alveolar spaces

only slight fibrous thickening of the alveolar walls.

MORPHOLOGY, grossly:

- **Ghon focus.**
  - ✓a 1-cm to 1.5-cm area of gray-white inflammatory consolidation emerges during the development of sensitization
  - ✓In majority of cases→ central caseous necrosis.

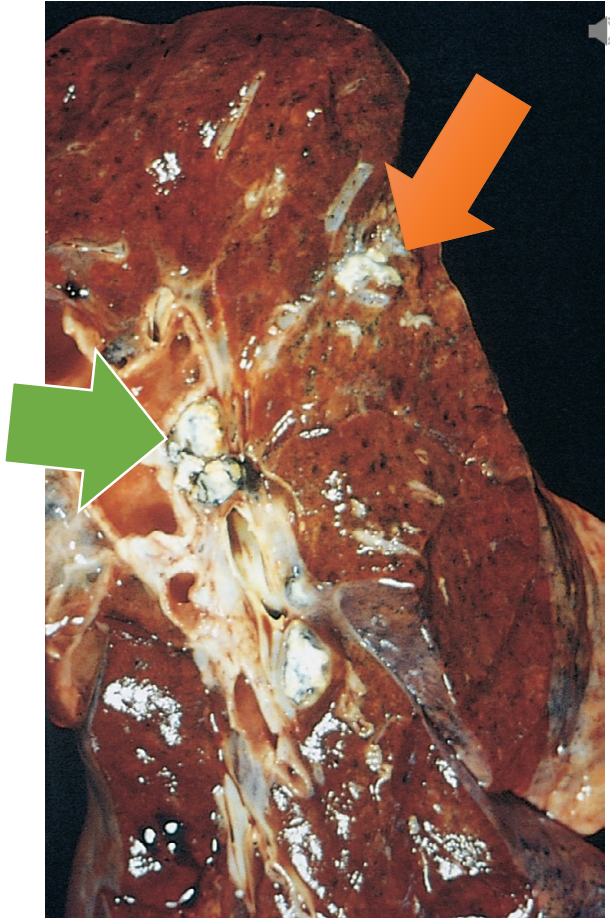


Robbins and Cotran pathologic basis of disease, 10th edition

MORPHOLOGY, grossly:

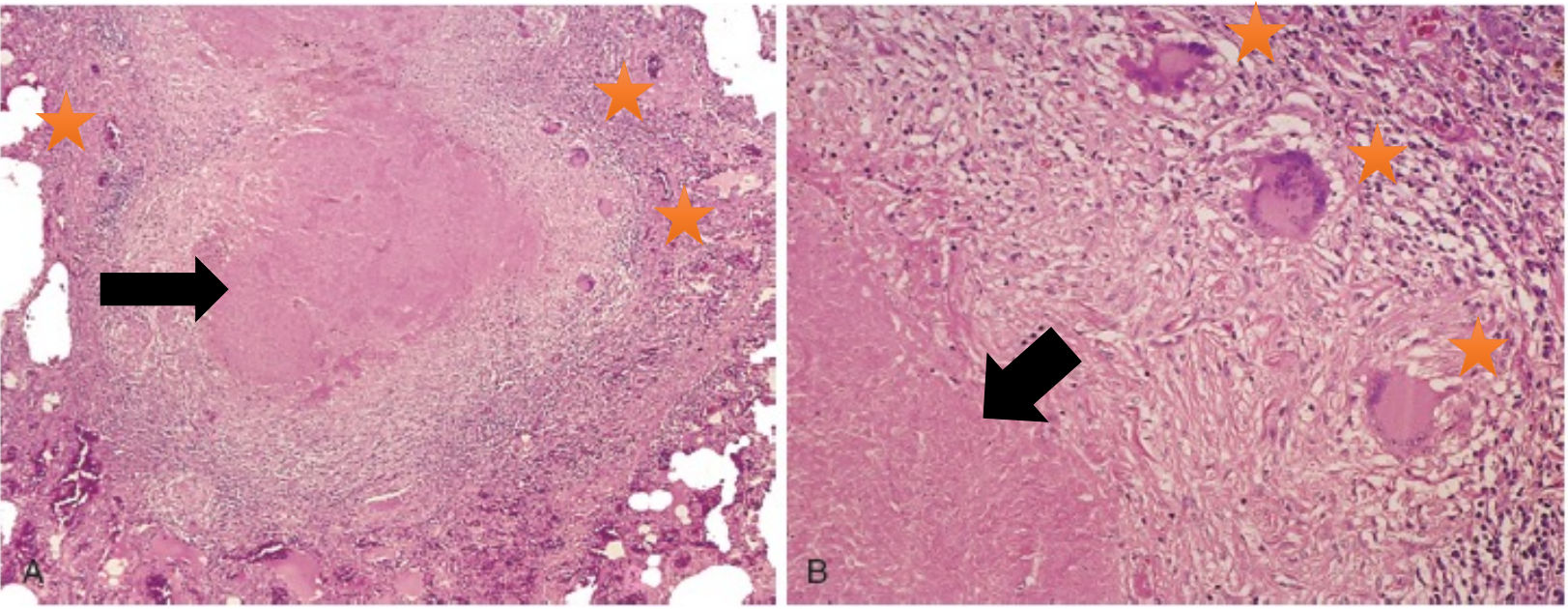
- Tubercle bacilli, free or within phagocytes, travel via the lymphatic vessels to regional lymph nodes.

- **Ghon complex** :This combination of parenchymal and nodal lesions



Robbins and Cotran pathologic basis of disease, 10h edition

MORPHOLOGY, microscopic:



tubercle

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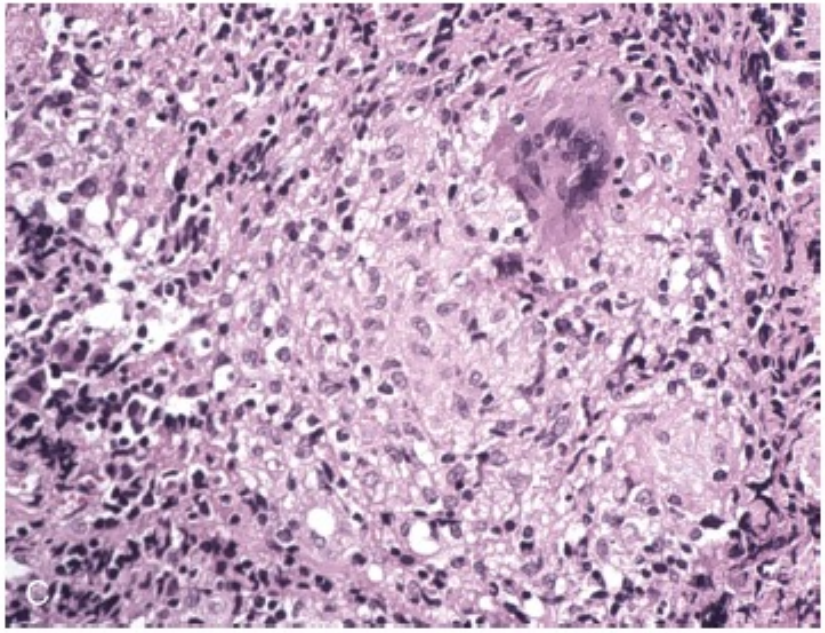

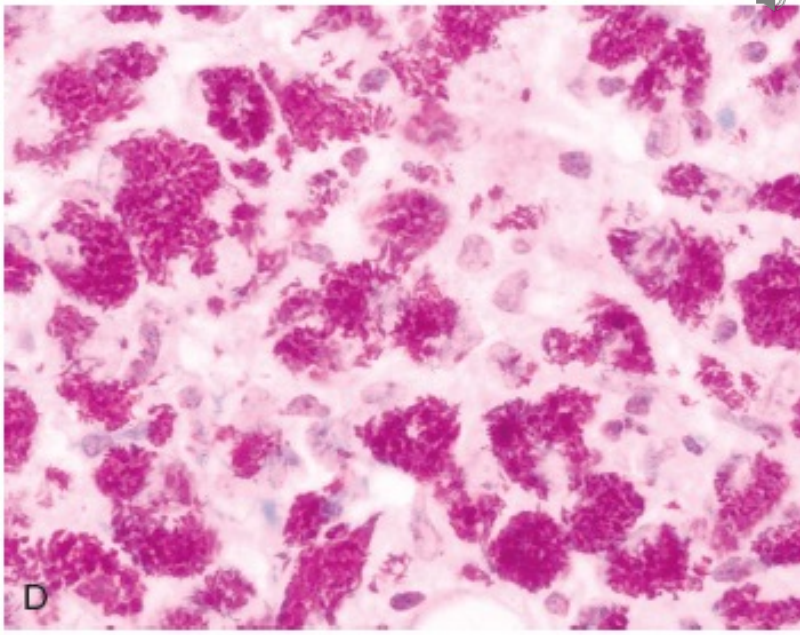

tubercular granulomas without central caseation

ZN stain→ sheets of macrophages packed with mycobacteria

irrespective of the presence or absence of caseous necrosis special stains for acid-fast organism

Robbins and Cotran pathologic basis of disease, 10h edition

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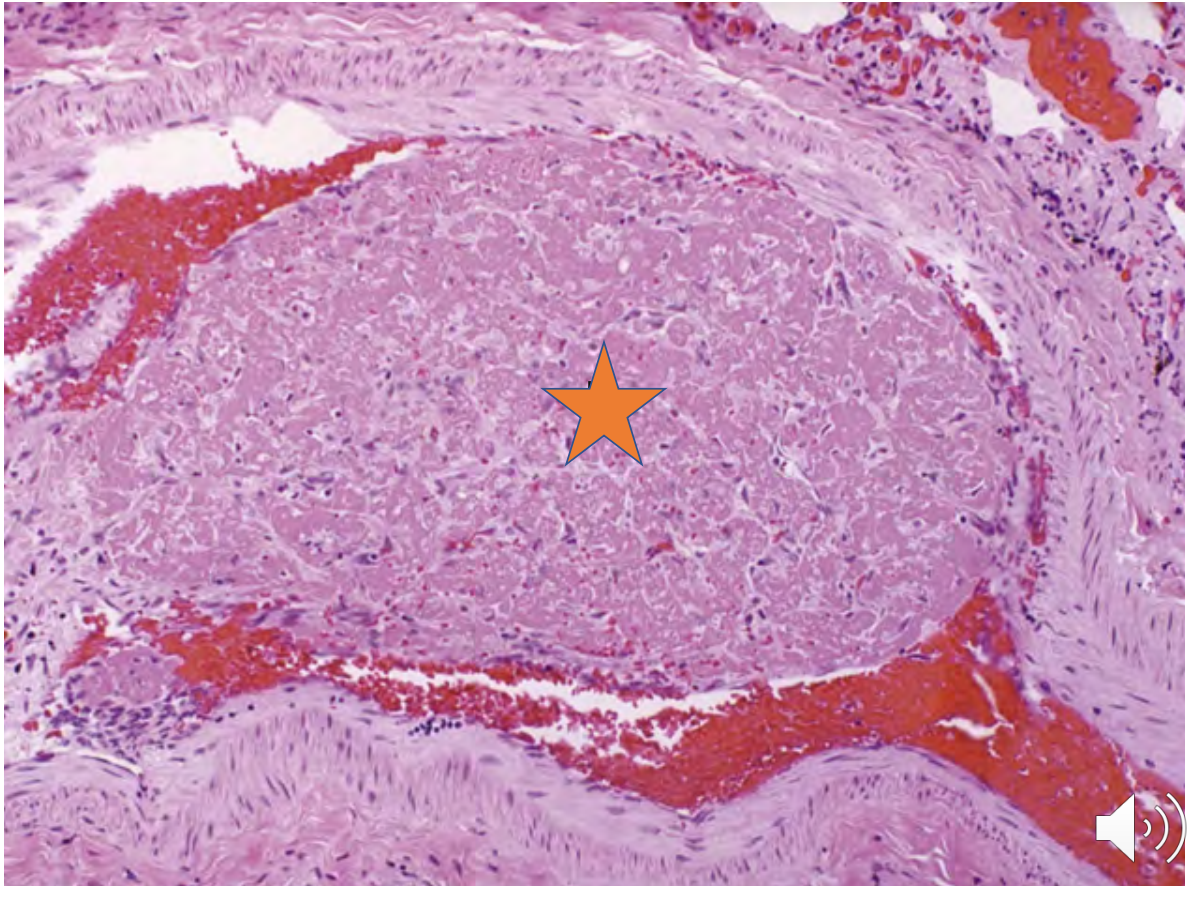

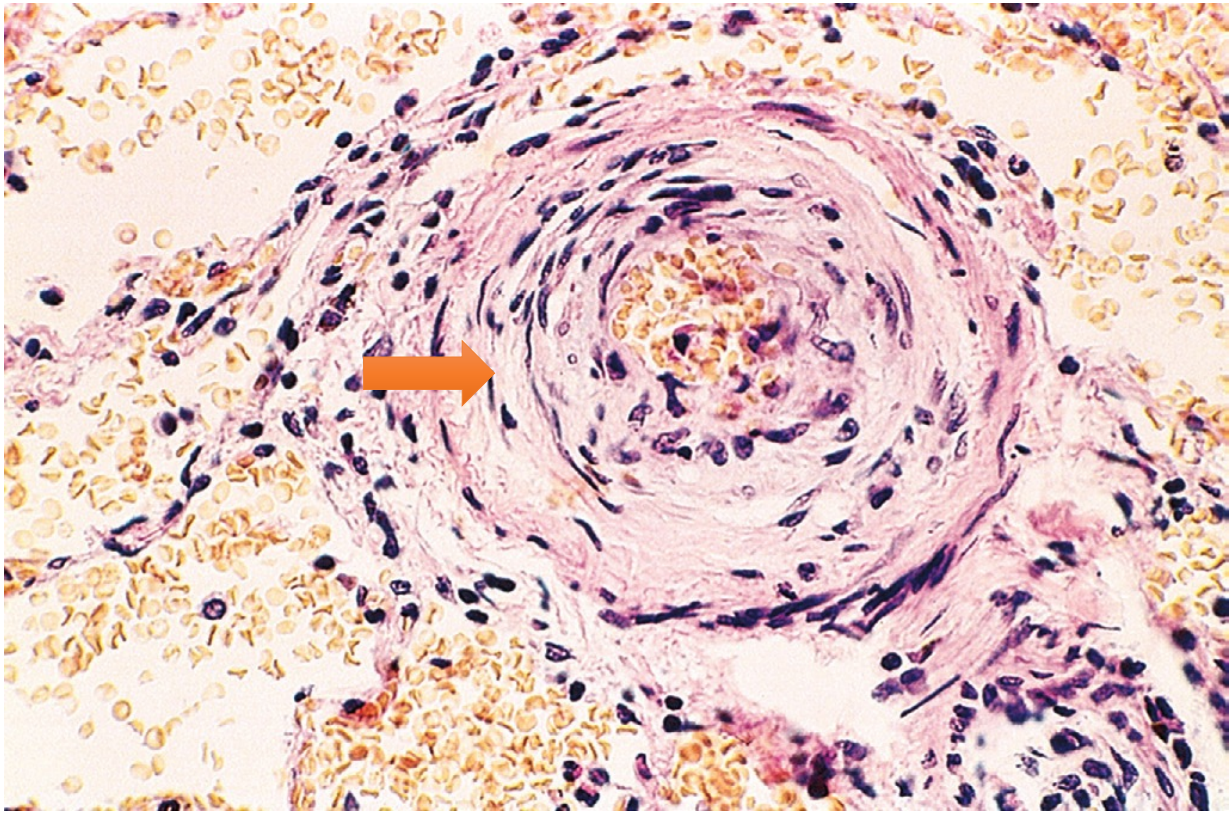

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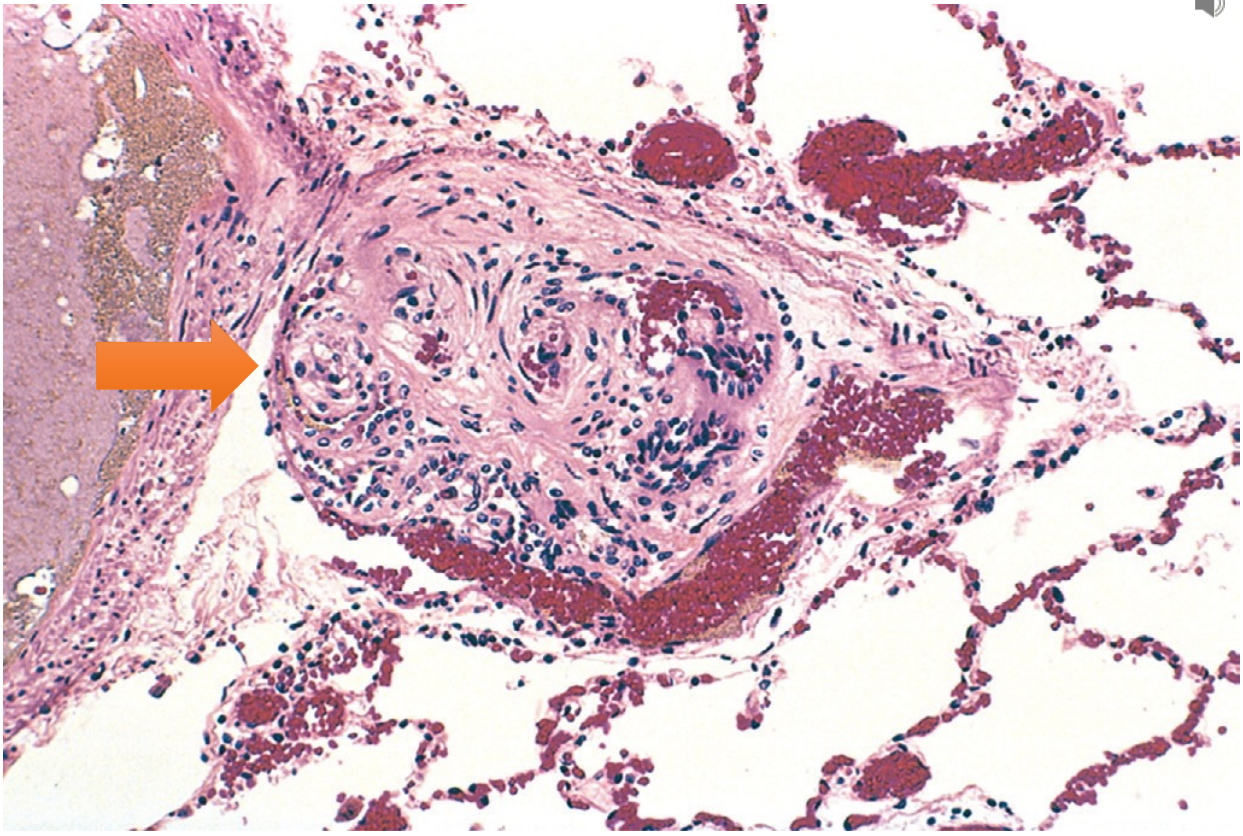

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Good luck!