Pathology of the lung

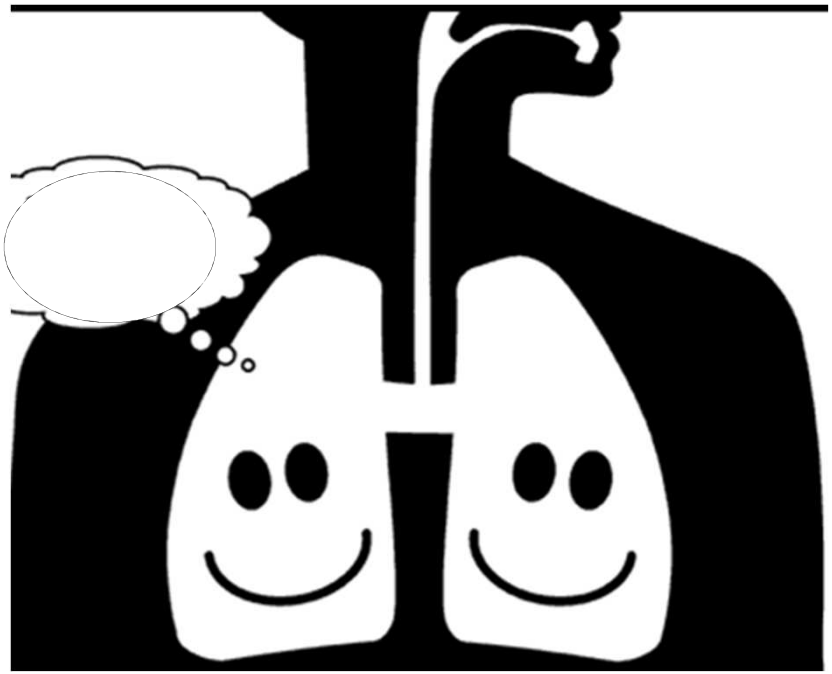
Developmental abnormalities

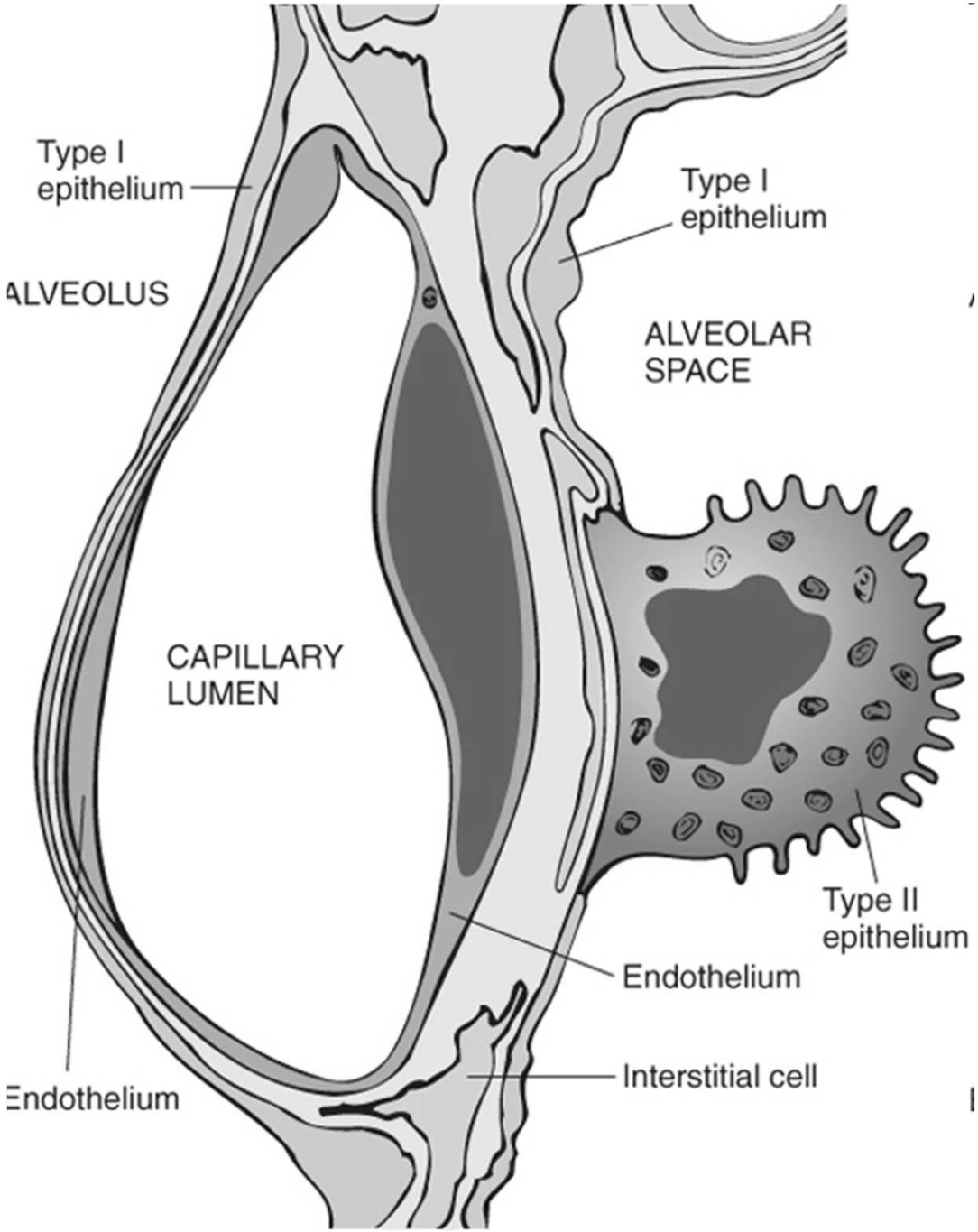
Atelectasis (collapse)

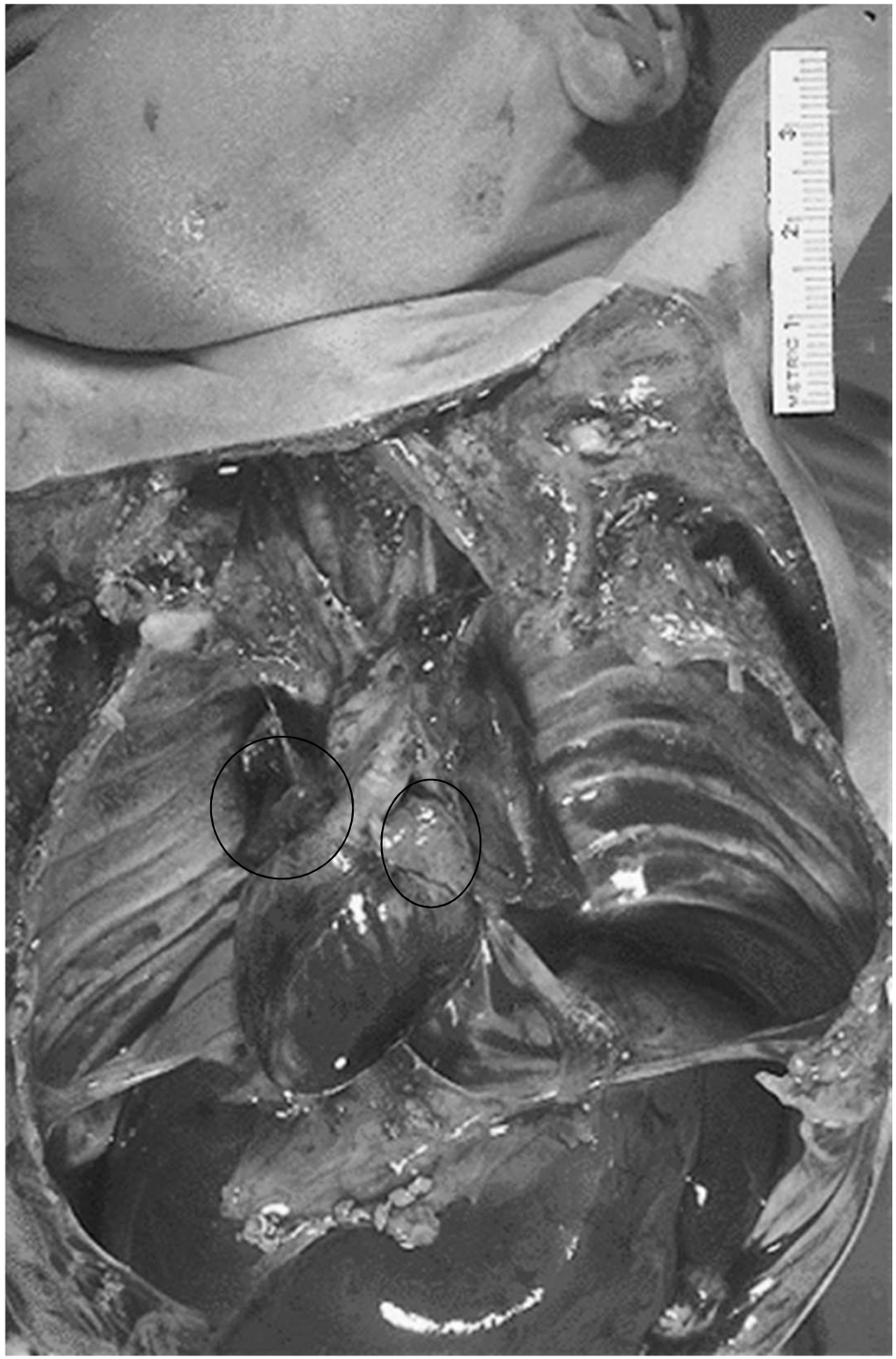
Circulatory disorders

Obstructive lung disease (COPD)

Restrictive "



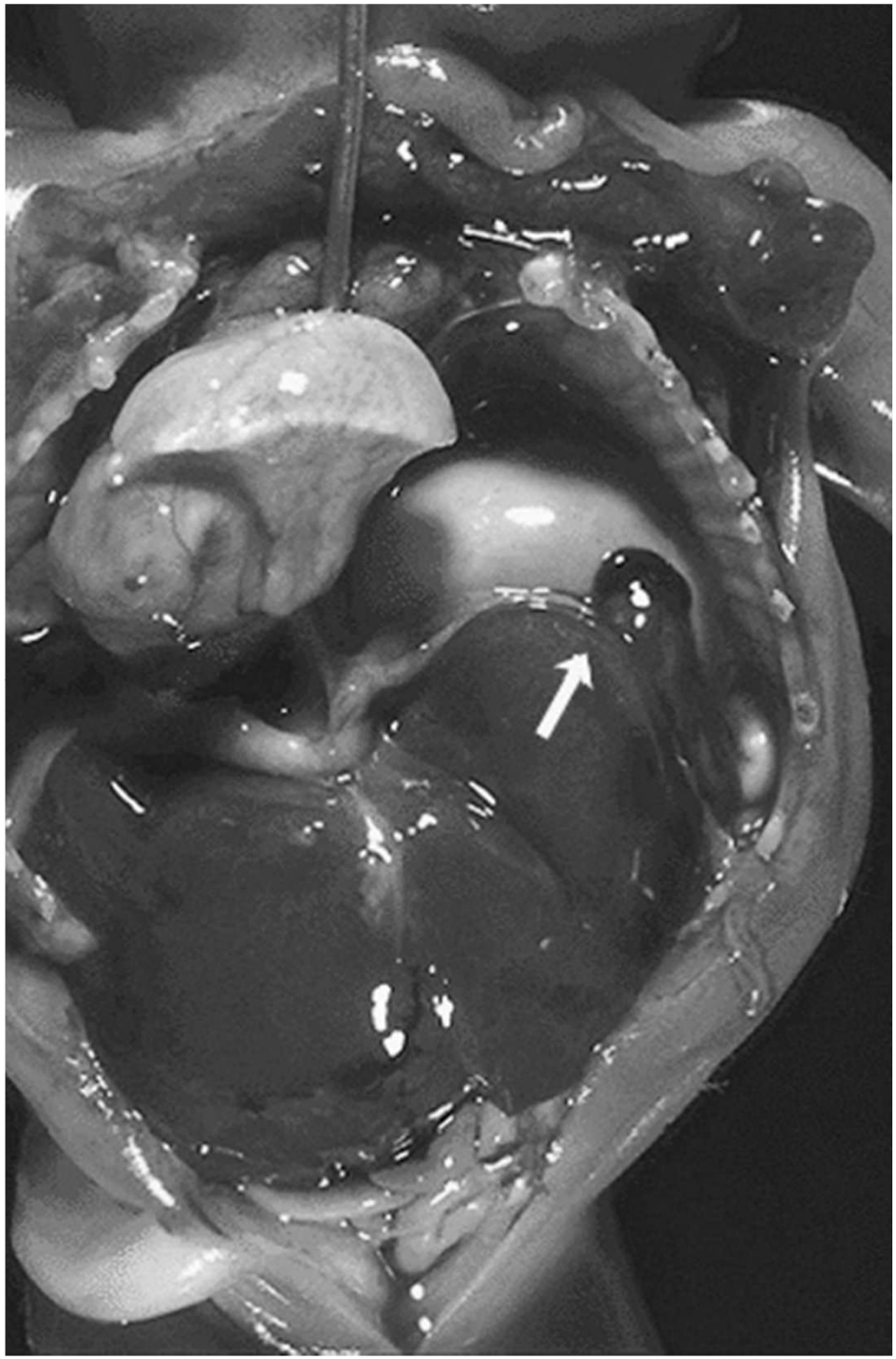




Pulmonary hypoplasia

consequence of oligohydramnion :

extremely small lungs on each side of the heart

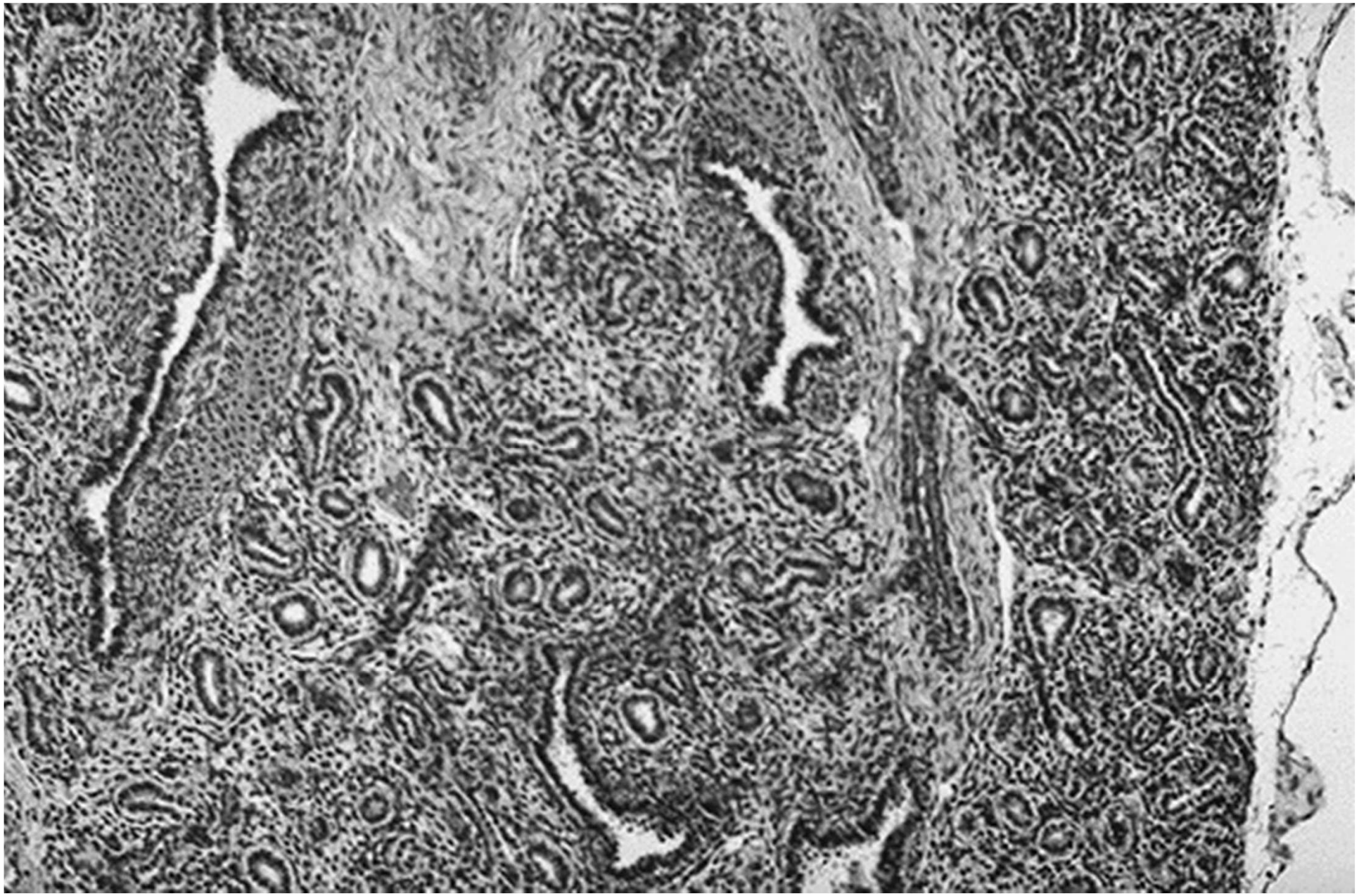


The diaphragmatic dome is missing on the left

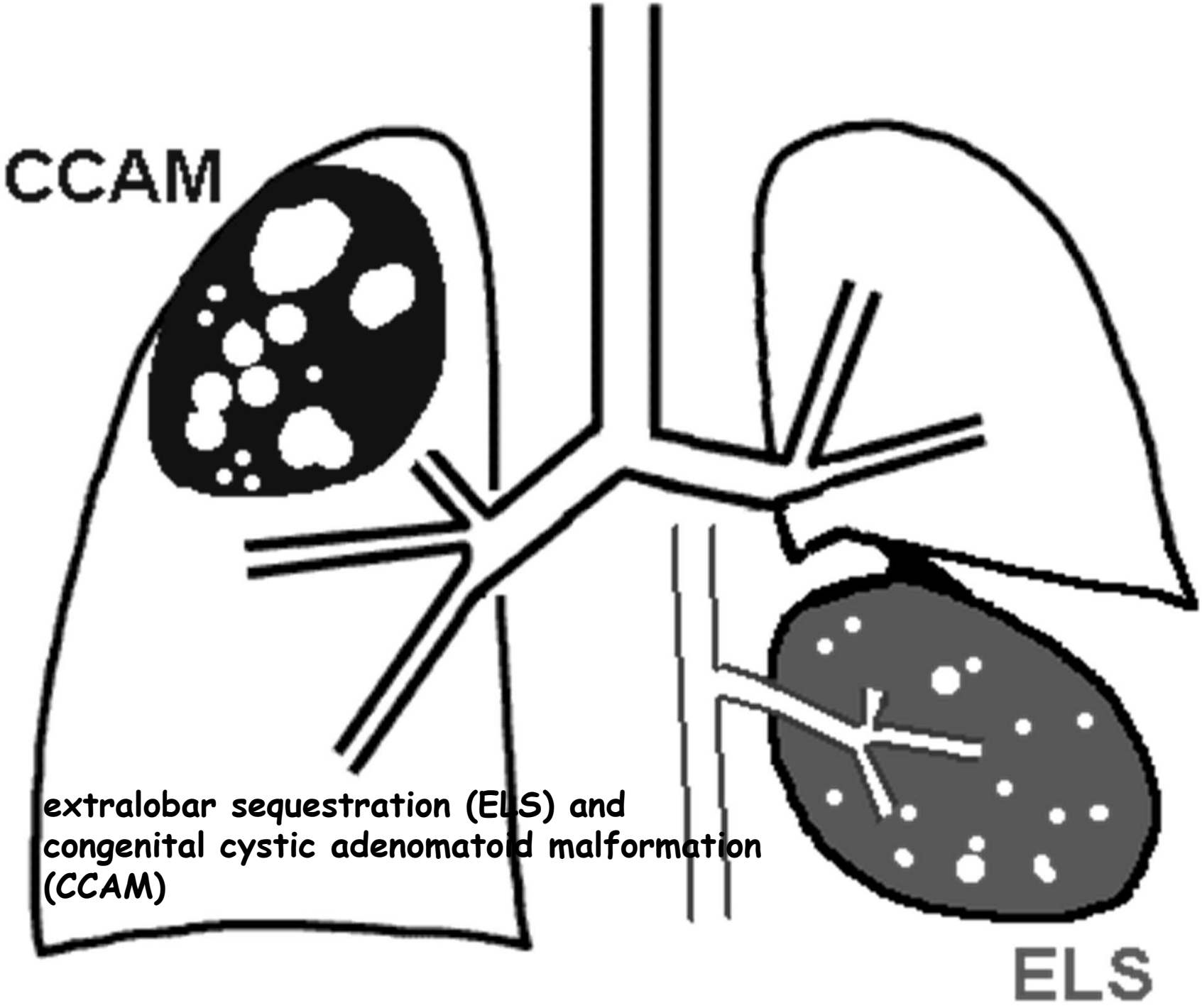
herniation of the abdominal contents into the chest cavity

left lung displaced by the stomach - dark spleen (at the white arrow) -

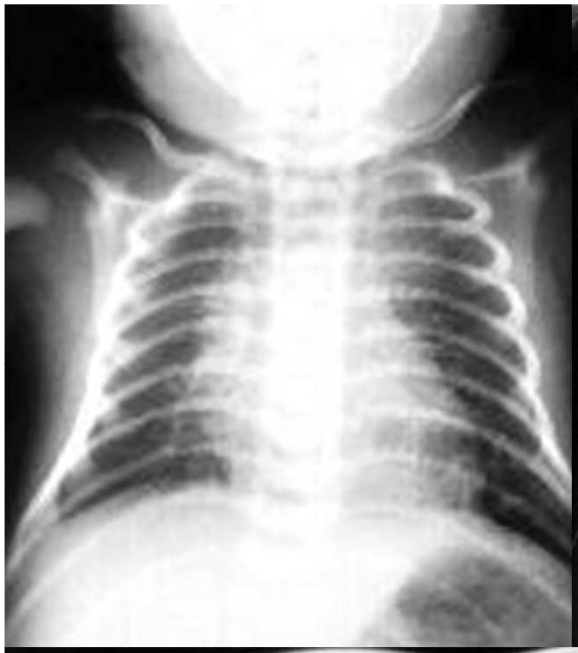
left lobe of liver extending upward.

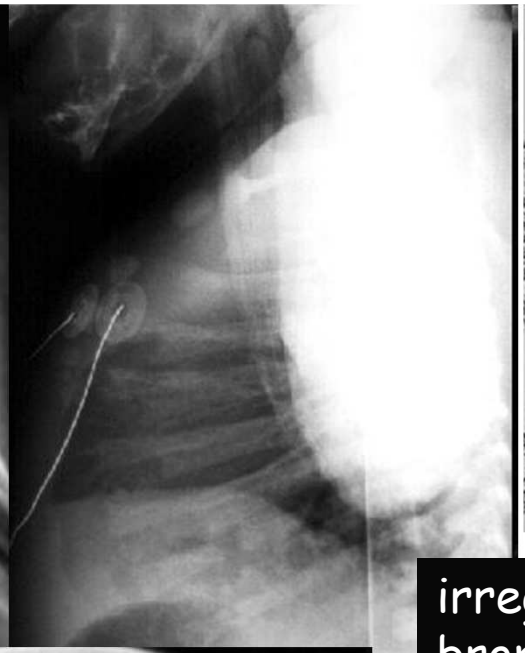


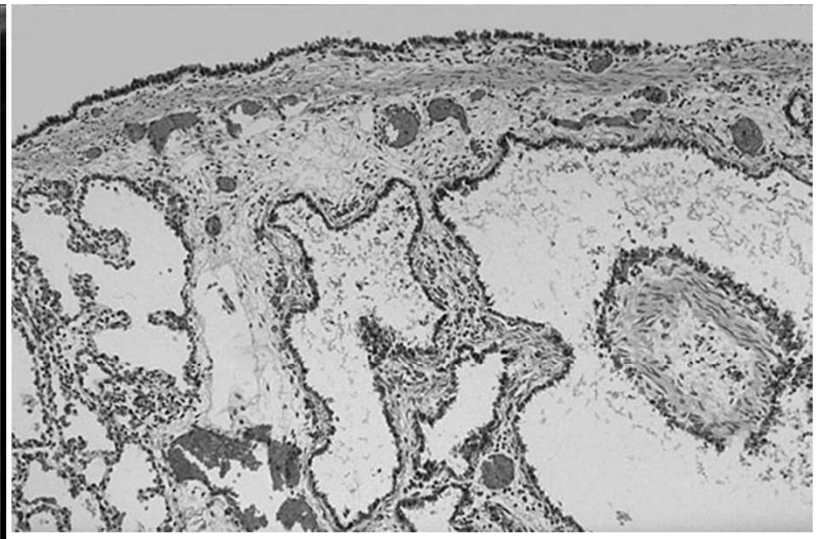
No alveolar development, only tubular bronchioles incapable of significant gas exchange - oligohydramnion and pulmonary hypoplasia



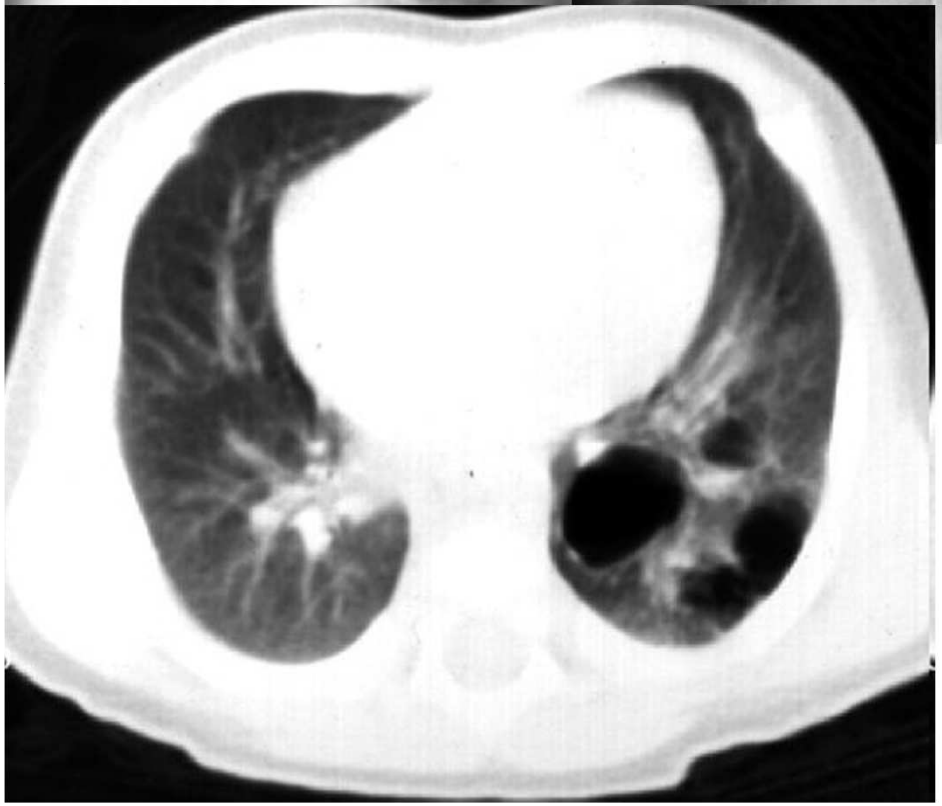
extralobar sequestration (ELS) and congenital cystic adenomatoid malformation (CCAM)

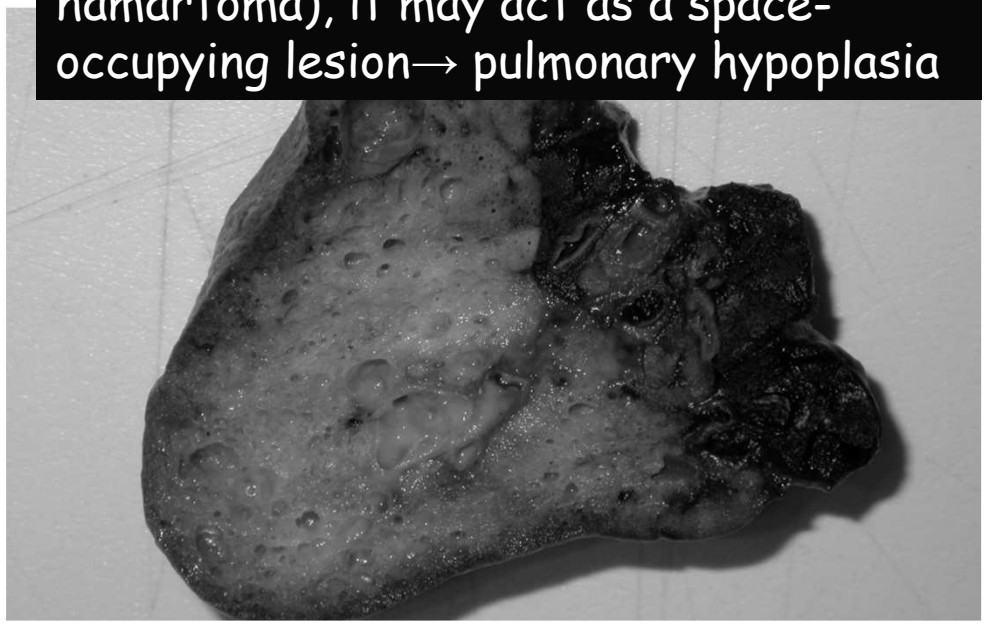


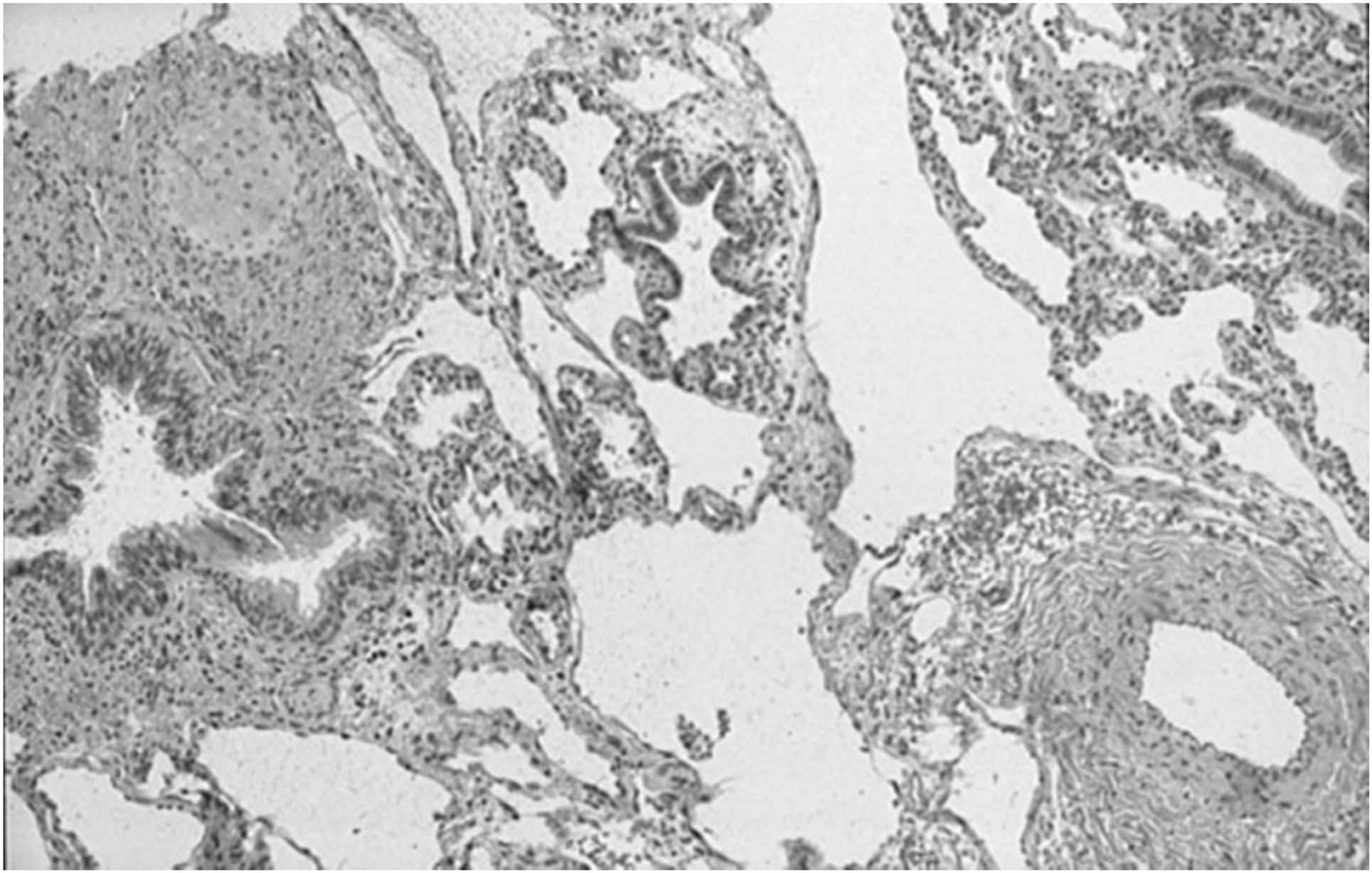




irregular cystic spaces lined by bronchial epithelium - congenital cystic adenomatoid malformation (CCAM) - the lesion is benign (much like a hamartoma), it may act as a space-occupying lesion→ pulmonary hypoplasia







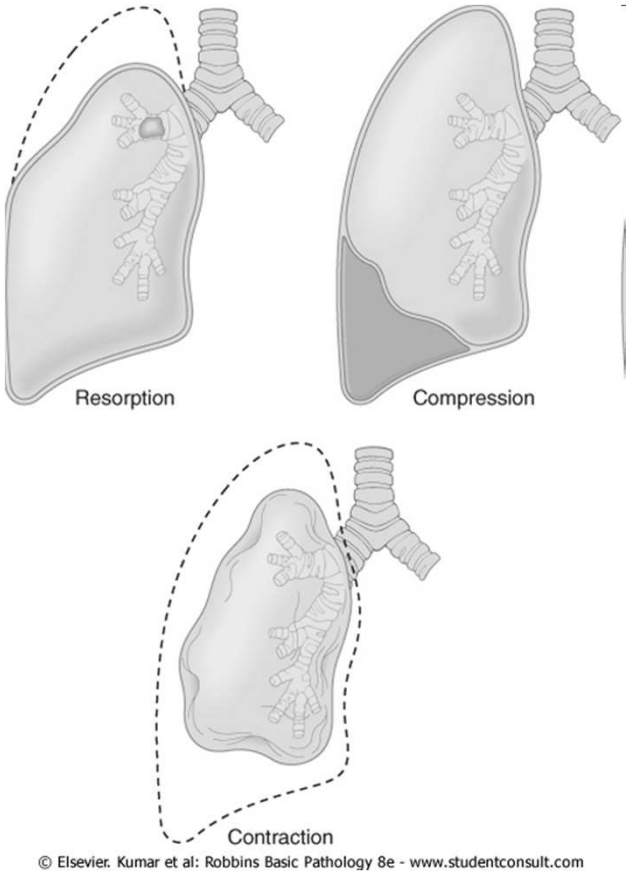
pulmonary extralobar sequestration (ELS) - irregular bronchi as well as dilated distal air spaces - the vascular arterial supply is systemic, not from the pulmonary artery - this portion of lung does not function

Atelectasis (collapse)

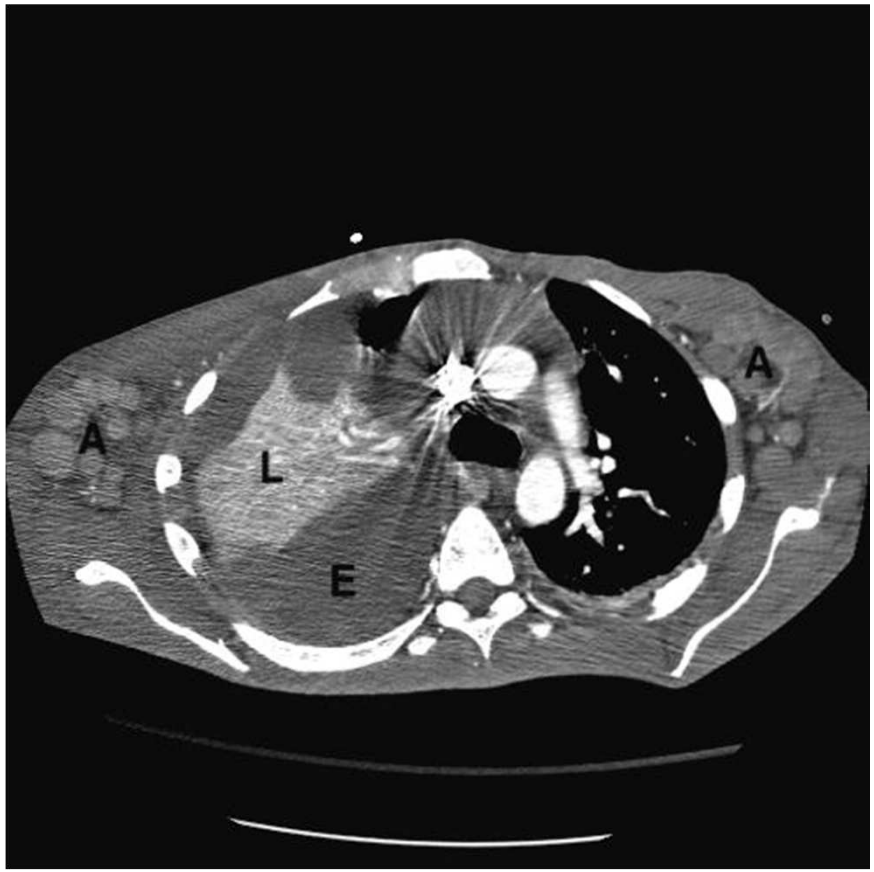
Incomplete expansion of the lungs (neonatal)

Collapse

- resorption (obstruction)
- compression
- contraction



Unsaturated blood flows directly from pulmonary arteries to pulmonary veins



Massive pleural effusion. The computed tomograpy scan of the chest of a patient with massive pleural effusion is shown. The collapsed lung is labeled as L. The pleural effusion is labeled as E. Extensive axillary lymphadenopathy (labeled A) is also present.

Atelectasis (collapse)

Incomplete expansion of the lungs
(neonatal due to IRDS)

Collapse

- resorption (obstruction)
  - Intrabronchial
    - COPD
    - intrabronchial tumor
    - foreign bodies
  - Extrabronchial
    - lymph nodes
    - aneurysms

Atelectasis (collapse)

Compression

fluid

- blood
- exsudate
- lymph
- air (pneumothorax)

intraabdominal pressure elevation

Contraction

usually irreversible

localised or general fibrotic lesions of the lung or pleura

Microatelectasia / Dystelectasia

Weeker lung expansion due to

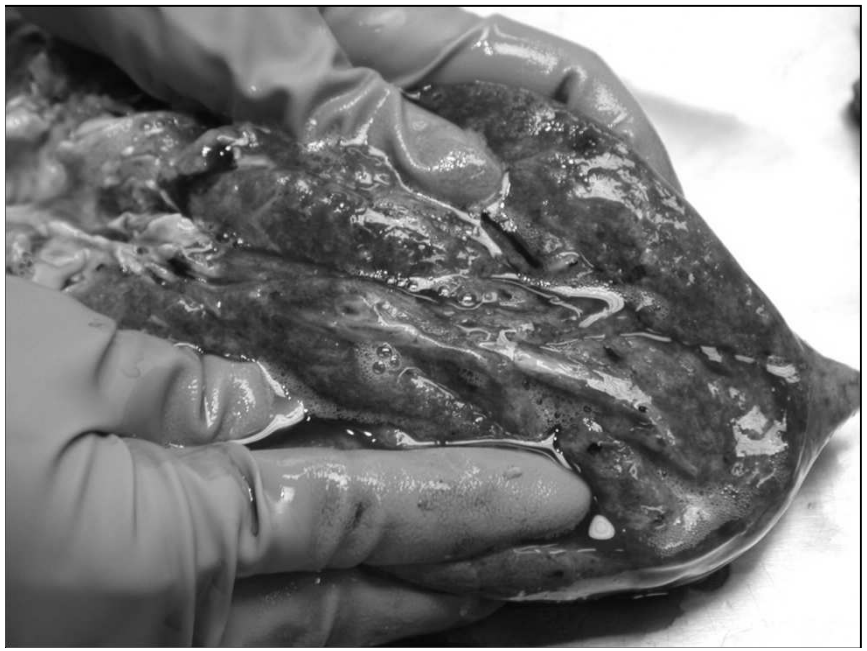
- RDS
- Surgical intervention
- Interstitial inflammation

Circulatory disorders

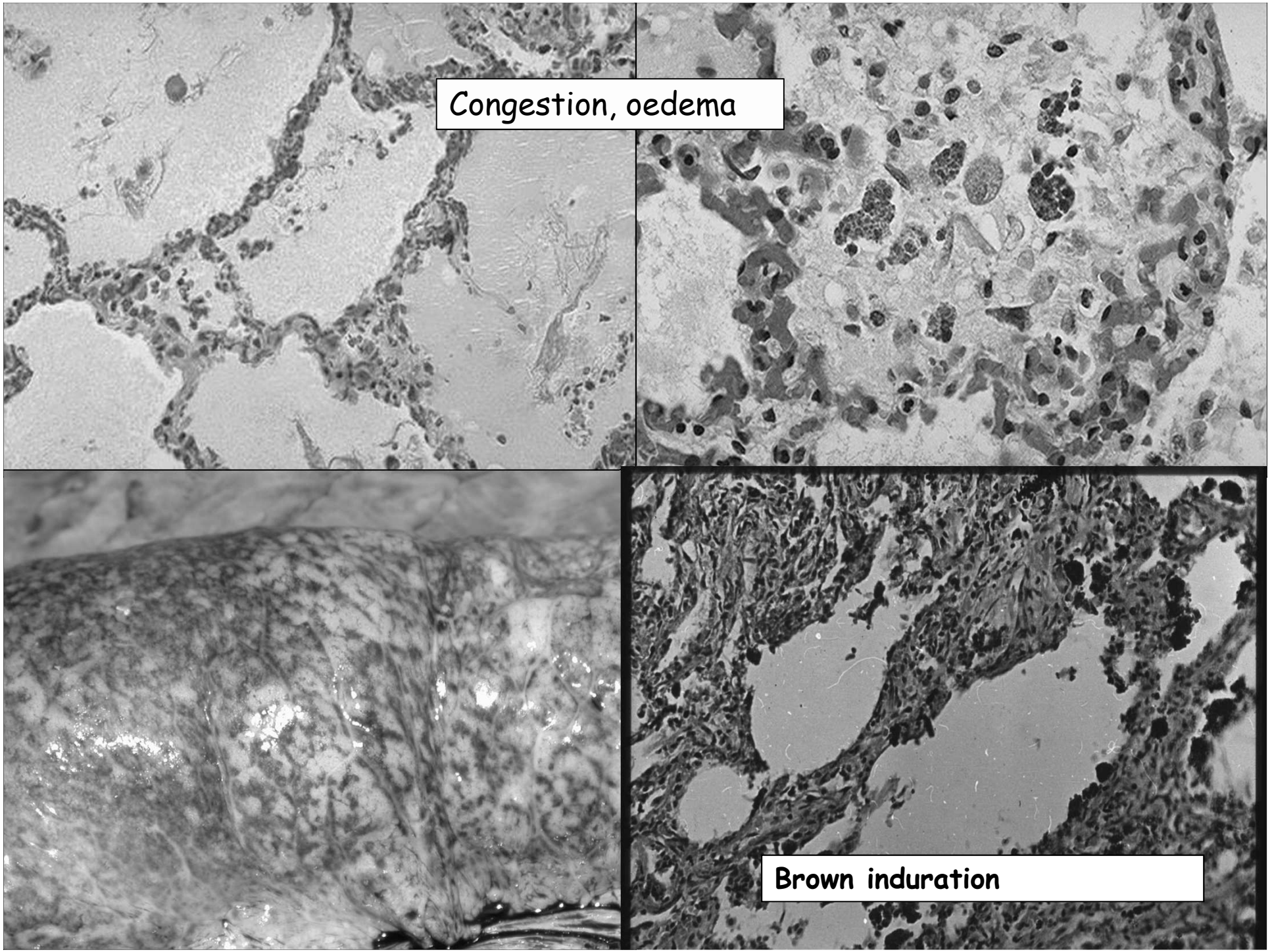
Edema

Cardiac – left ventricular failure

- acute



- chronic (heart failure cells, brown induration)


Congestion, oedema
Brown induration

Circulatory disorders

Edema

DAD (diffuse alveolar damage)

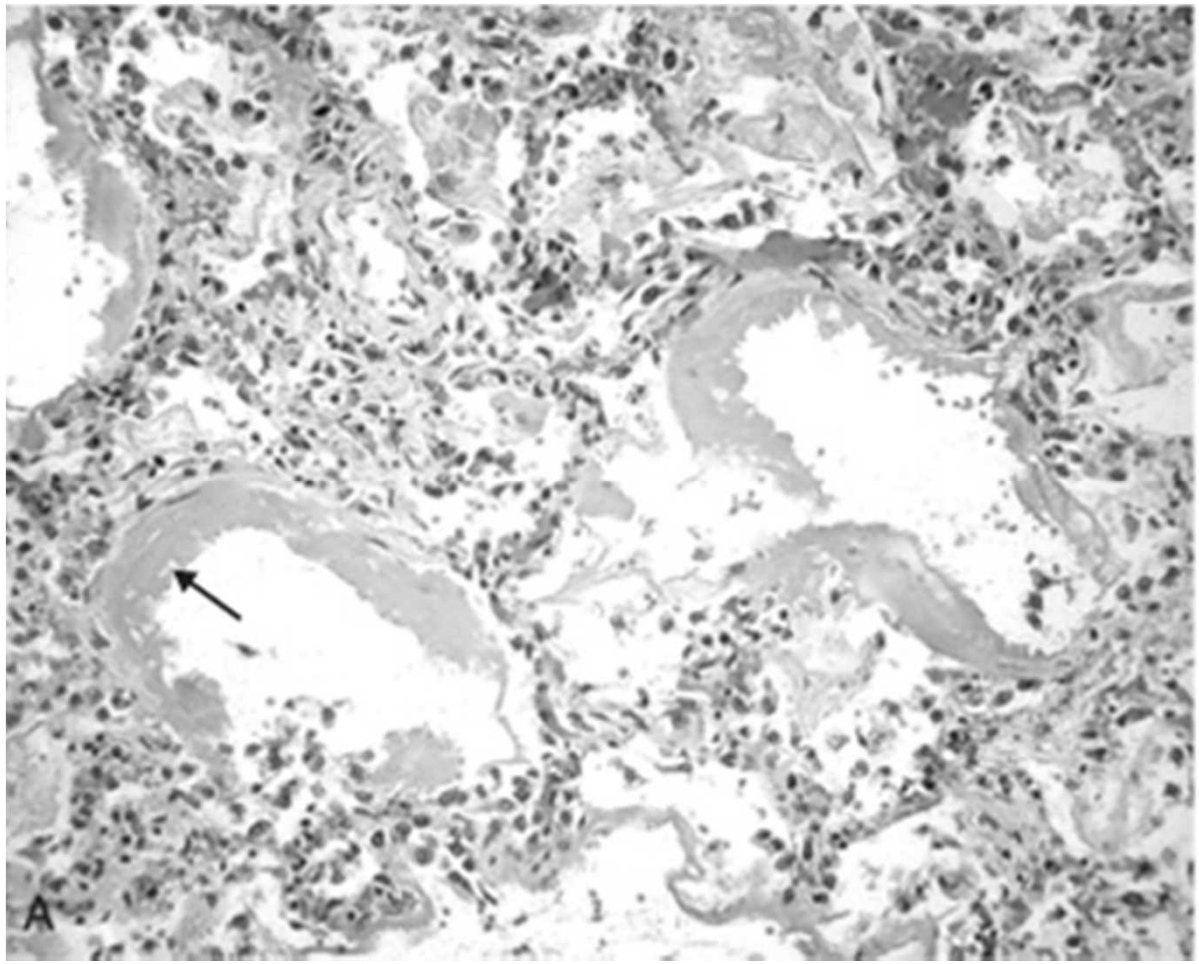
- inhalation of toxic gases
- sepsis
- pulmonary infections
- gastric aspiration
- all types of physical injuries (mechanical, burns)
- fat embolism
- drug overdoses
- haematologic disorders (transfusions, DIC)
- pancreatitis
- uremia
- cardiopulmonary bypass
- hypersensitivity reactions

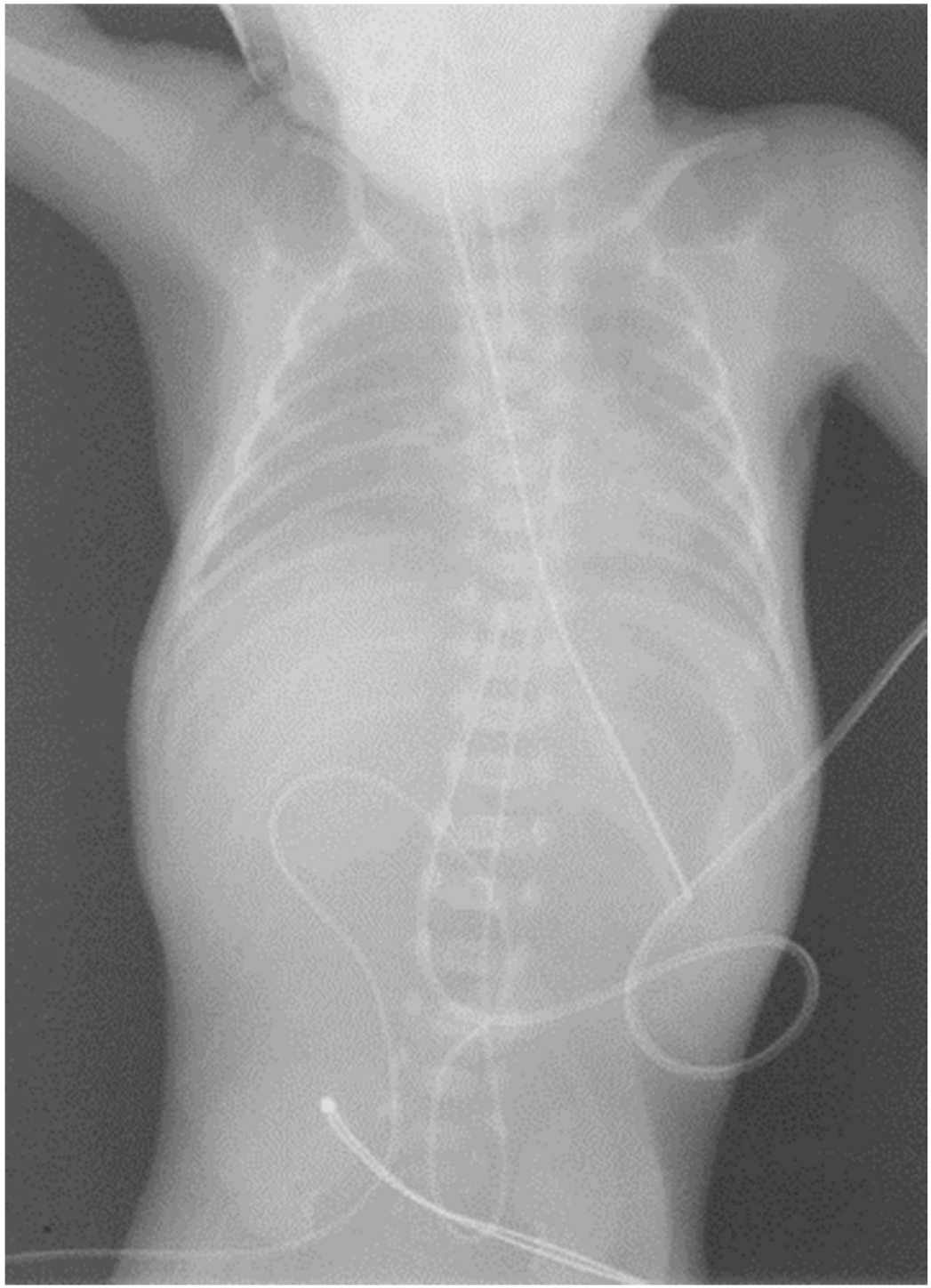
Circulatory disorders

Edema

DAD (diffuse alveolar damage)

ARDS



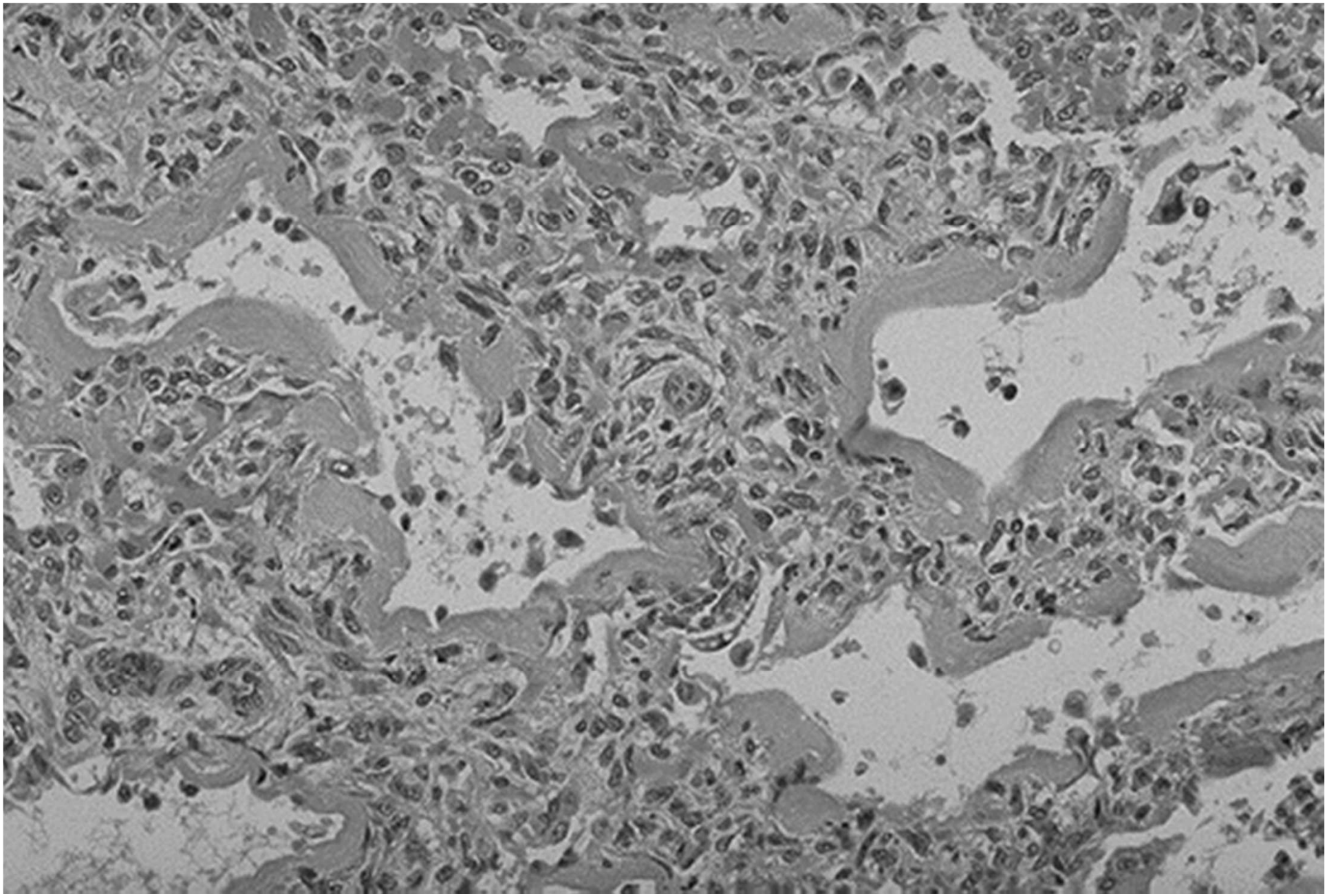


Chest radiograph of a premature newborn at 4 days of life

opacification of all lung fields as a consequence of hyaline membrane disease

the lungs of a premature baby are immature and

insufficient surfactant is produced to provide for appropriate **expansion**



Hyaline membrane disease due to prematurity and lack of surfactant production - thick pink membranes lining the alveolar spaces

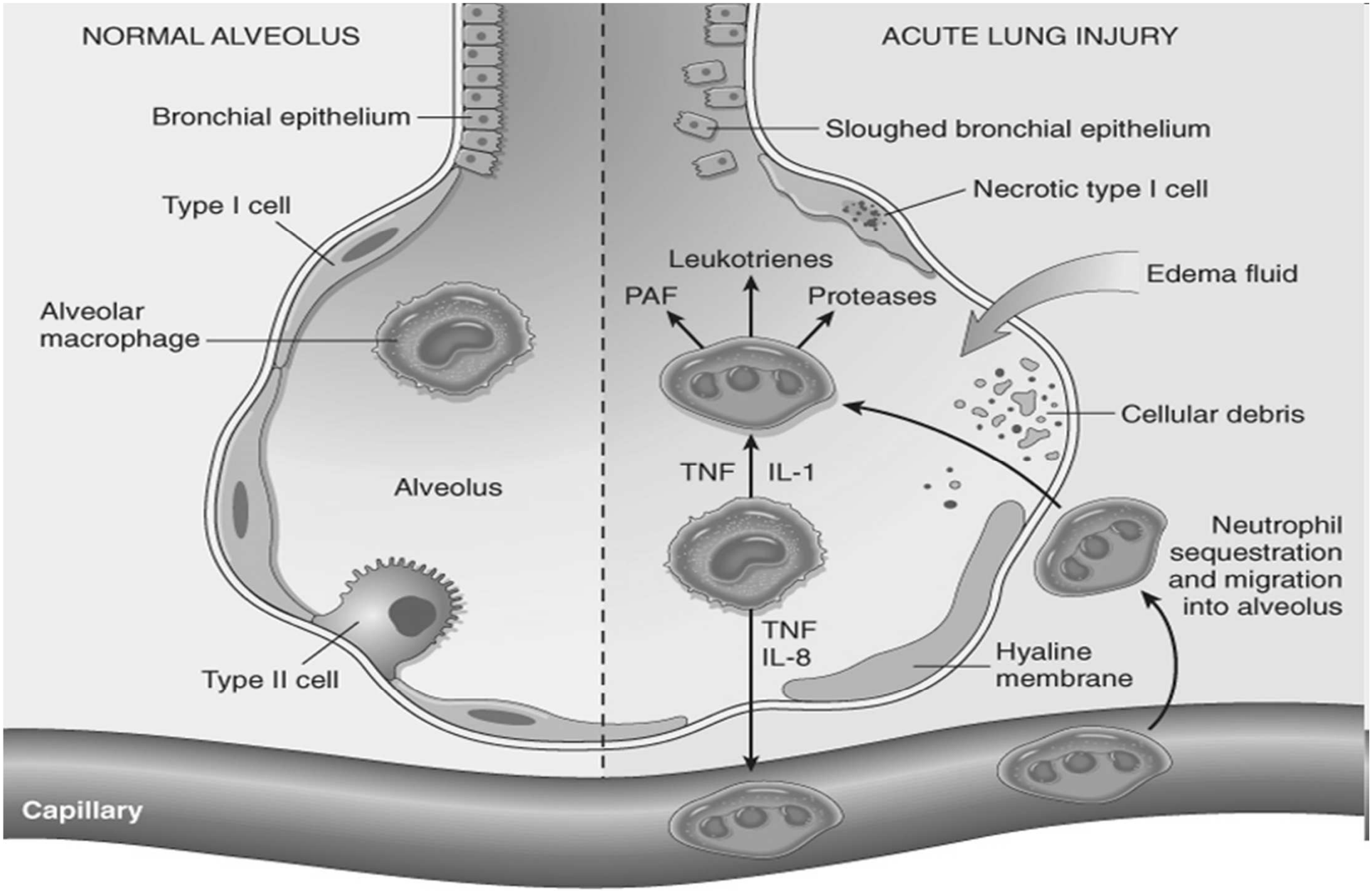


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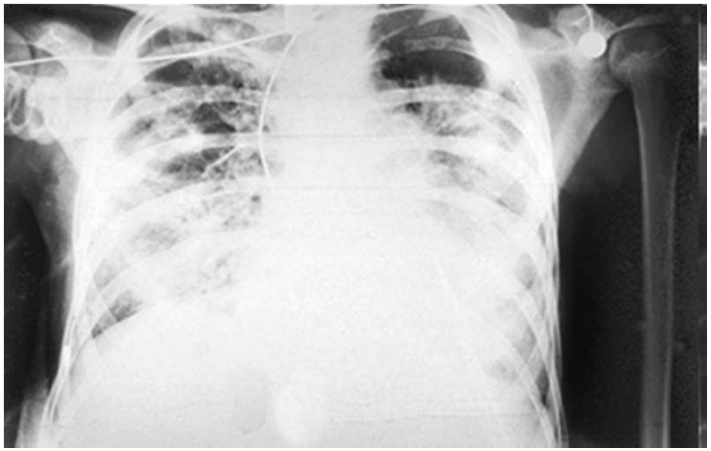
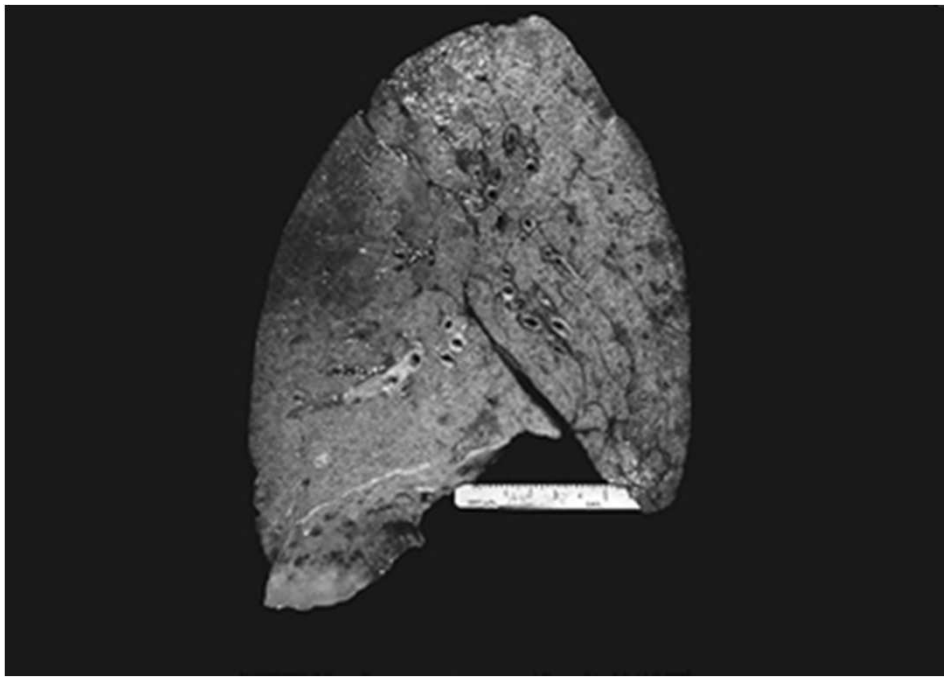
Figure 13-3 The normal alveolus *(left)* compared with the injured alveolus in the early phase of acute lung injury and the acute respiratory distress syndrome. Under the influence of proinflammatory cytokines such as IL-8, IL-1, and TNF (released by macrophages), neutrophils initially undergo sequestration in the pulmonary microvasculature, followed by margination and egress into the alveolar space, where they undergo activation. Activated neutrophils release a variety of factors such as leukotrienes, oxidants, proteases, and platelet-activating factor (PAF), which contribute to local tissue damage, accumulation of edema fluid in the airspaces, surfactant inactivation, and hyaline membrane formation. Subsequently, the release of macrophage-derived fibrogenic cytokines such as transforming growth factor β (TGF-β) and platelet-derived growth factor (PGDF) stimulate fibroblast growth and collagen deposition associated with the healing phase of injury.

ARDS



extensive, diffuse, bilateral pulmonary infiltrates.

ARDS

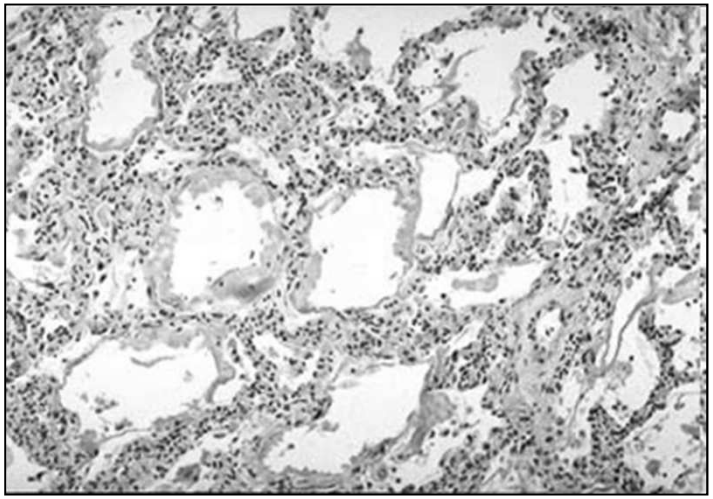


lungs are **heavy** (due to accumulation of fluid),

firm, red, and boggy,

keep their shape on the table

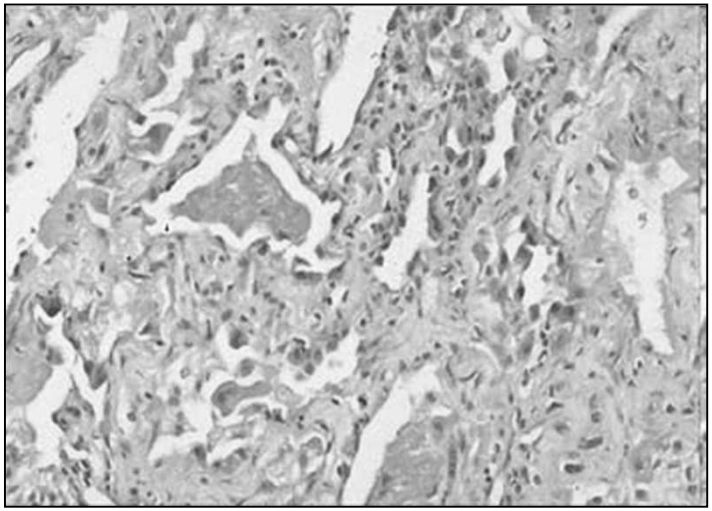
ARDS



ARDS, exsudative stage

Hyaline membranes,

Interstitial inflammation



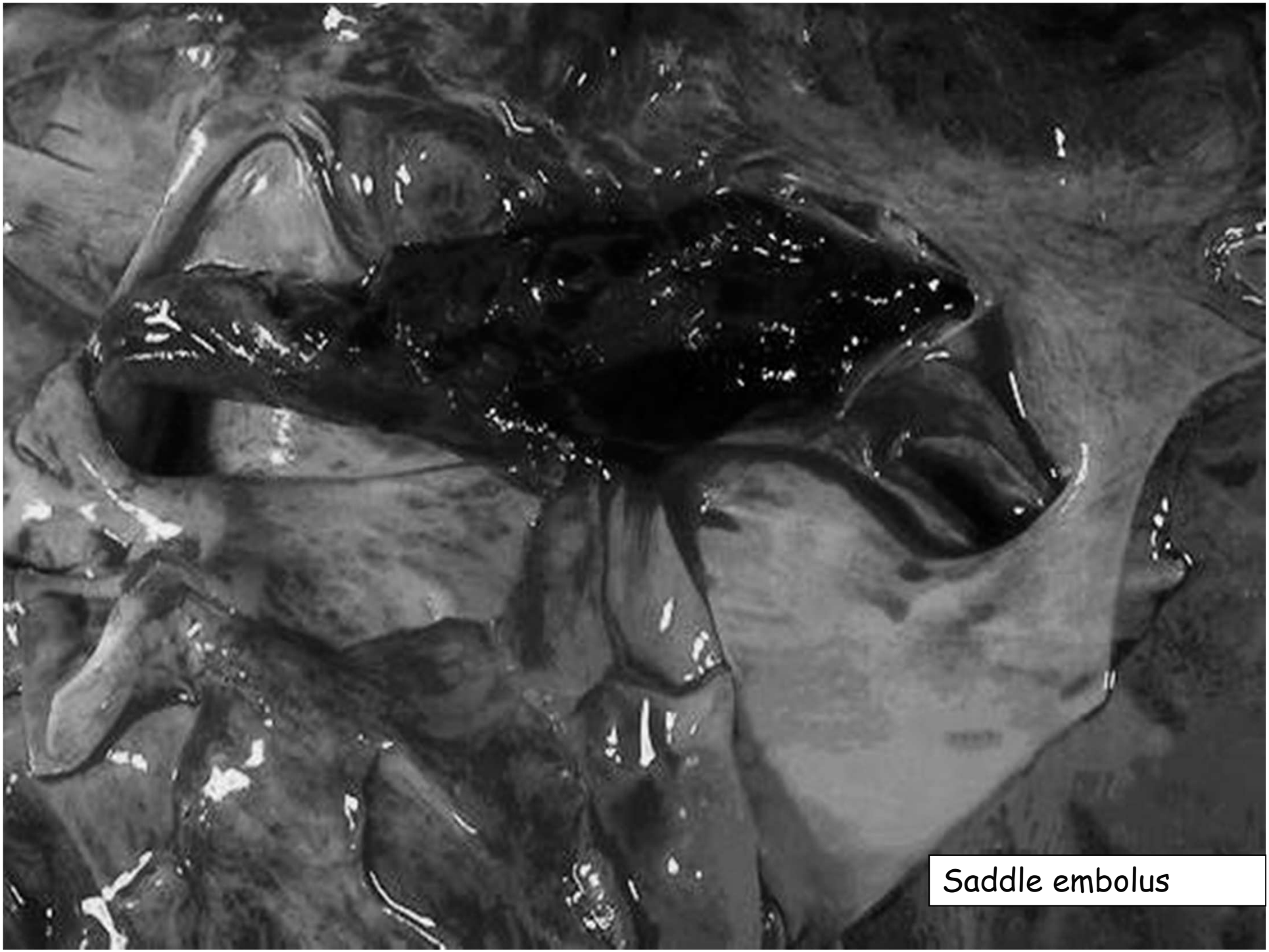
ARDS, proliferative stage

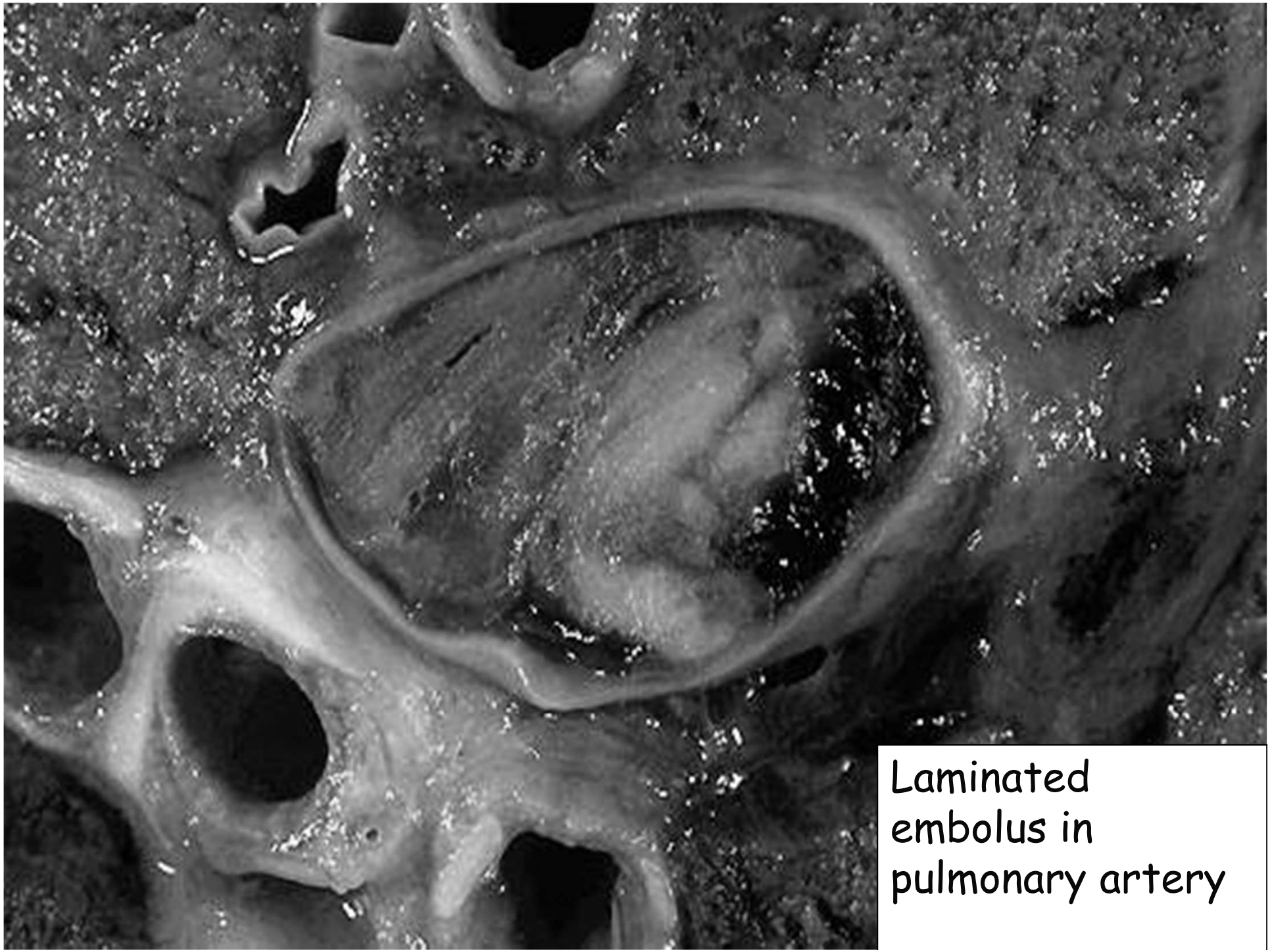
Thickened septa

Hyaline membranes get organized

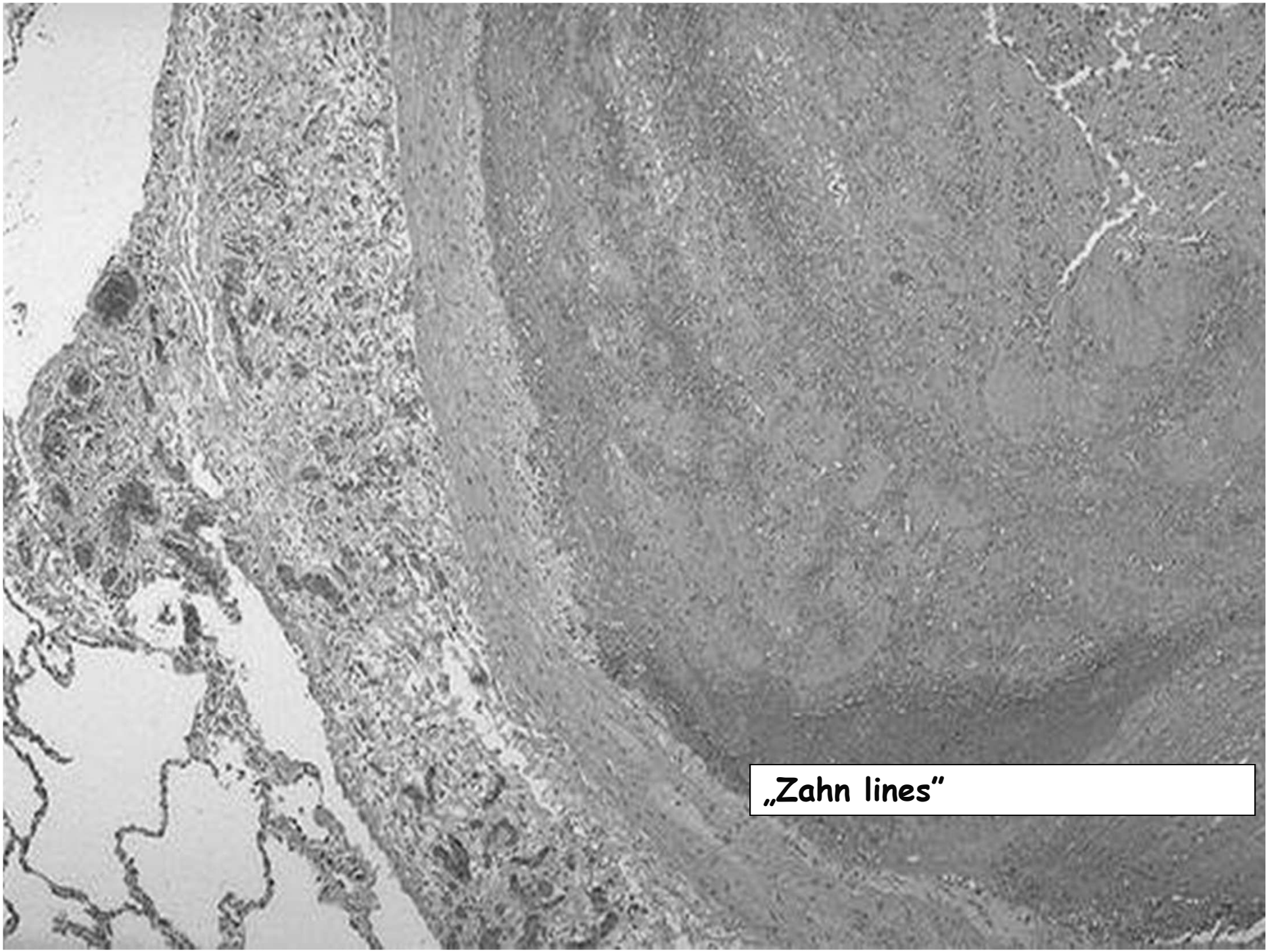
Pulmonary thromboembolism

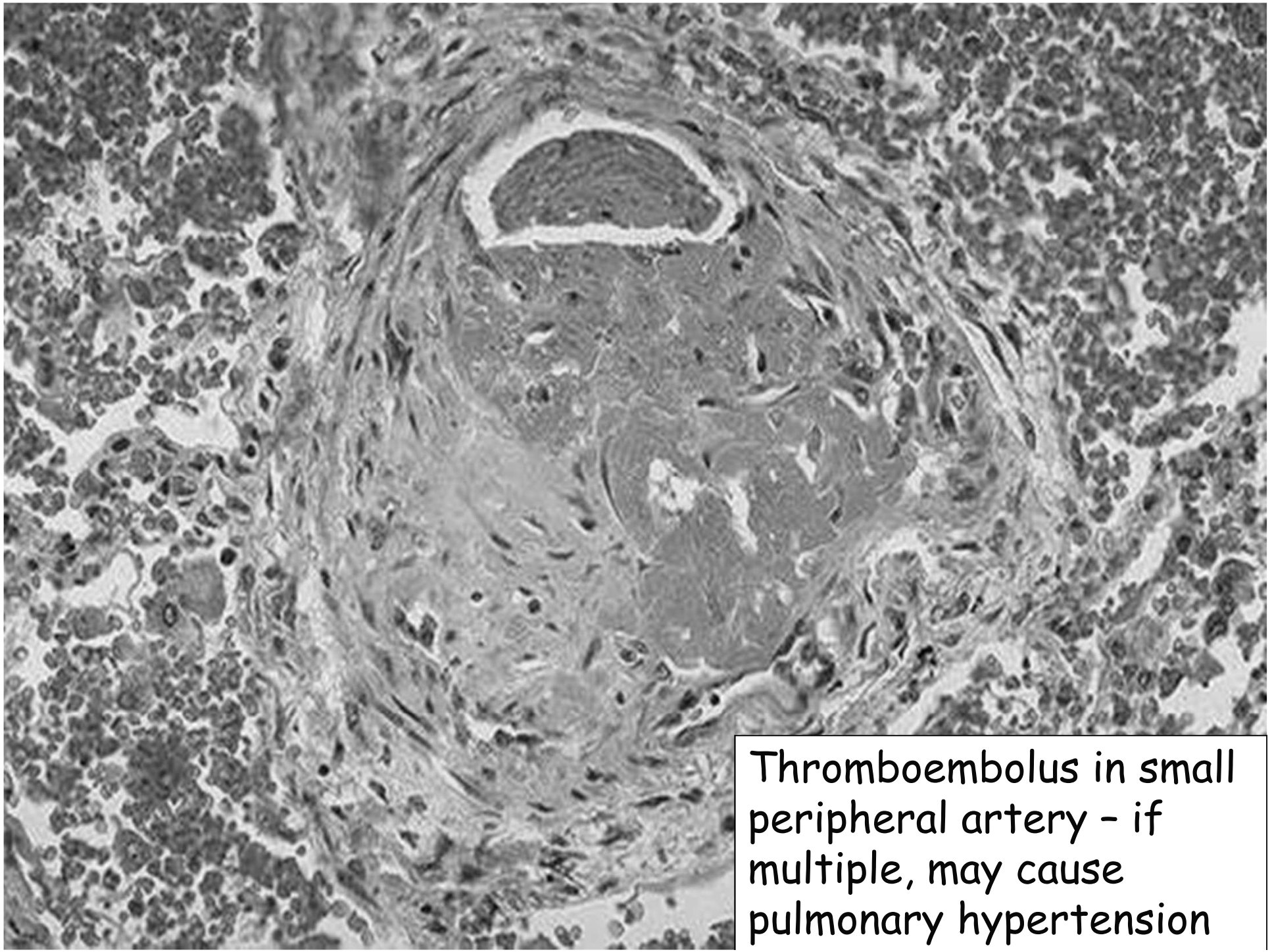
- Deep veins of the limbs
- Pelvic veins
- Immobilised patients
- Oral contraceptives
- Cardiac failure
- Pregnancy
- Old age
- Protein C, S insufficiency
- Trousseau' sign (carcinomas)


Saddle embolus

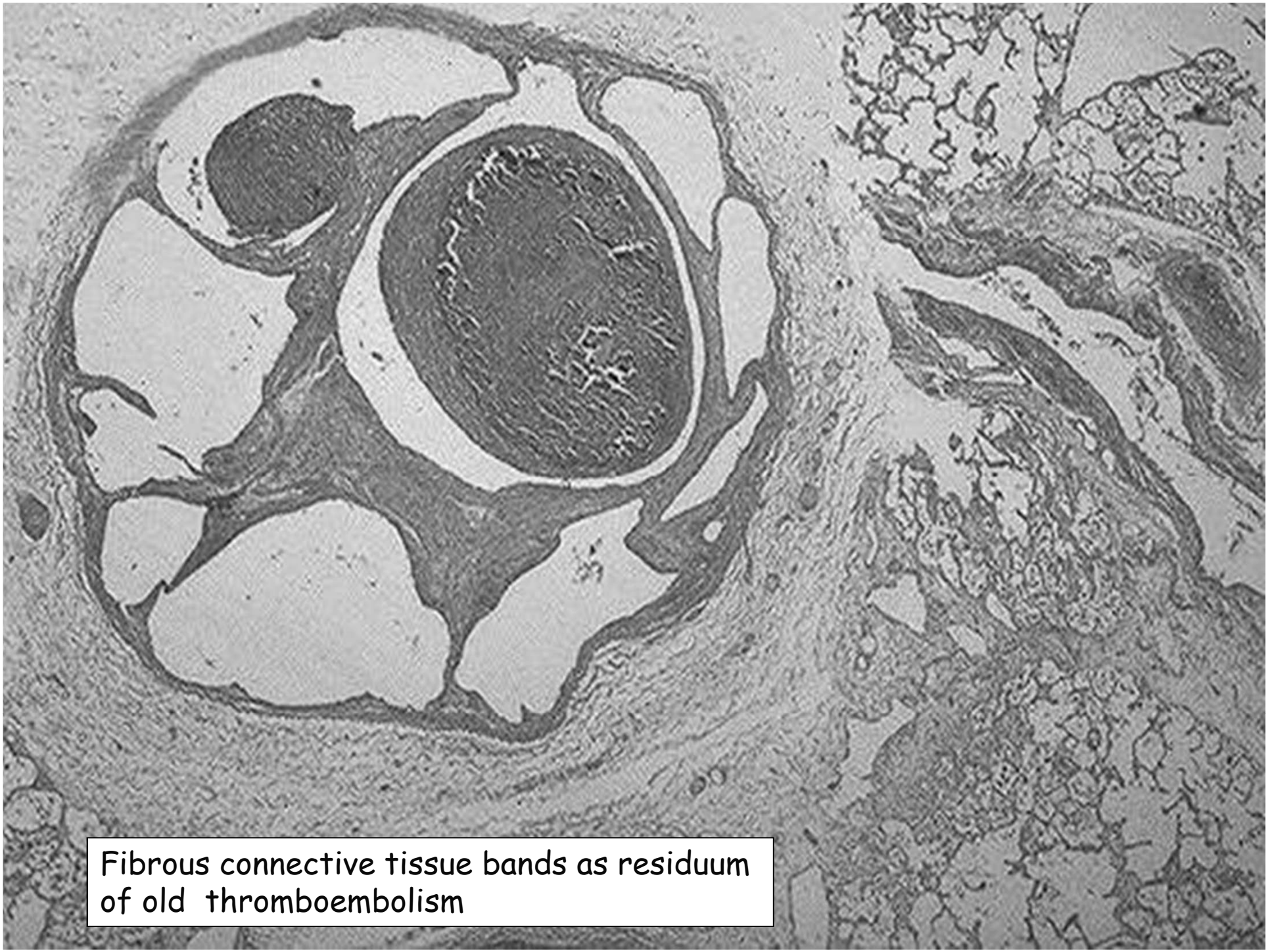


Laminated embolus in pulmonary artery

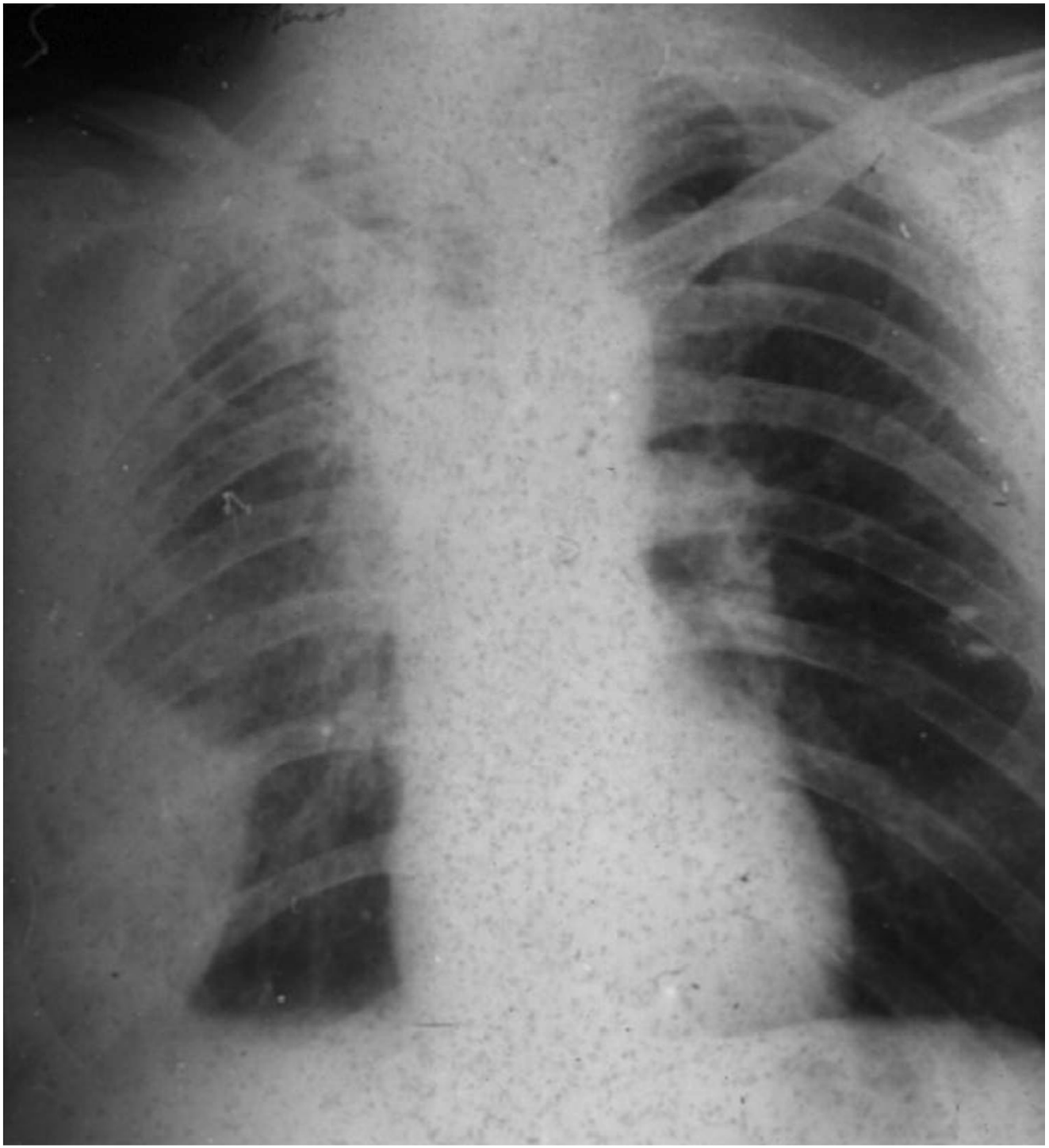

„Zahn lines“



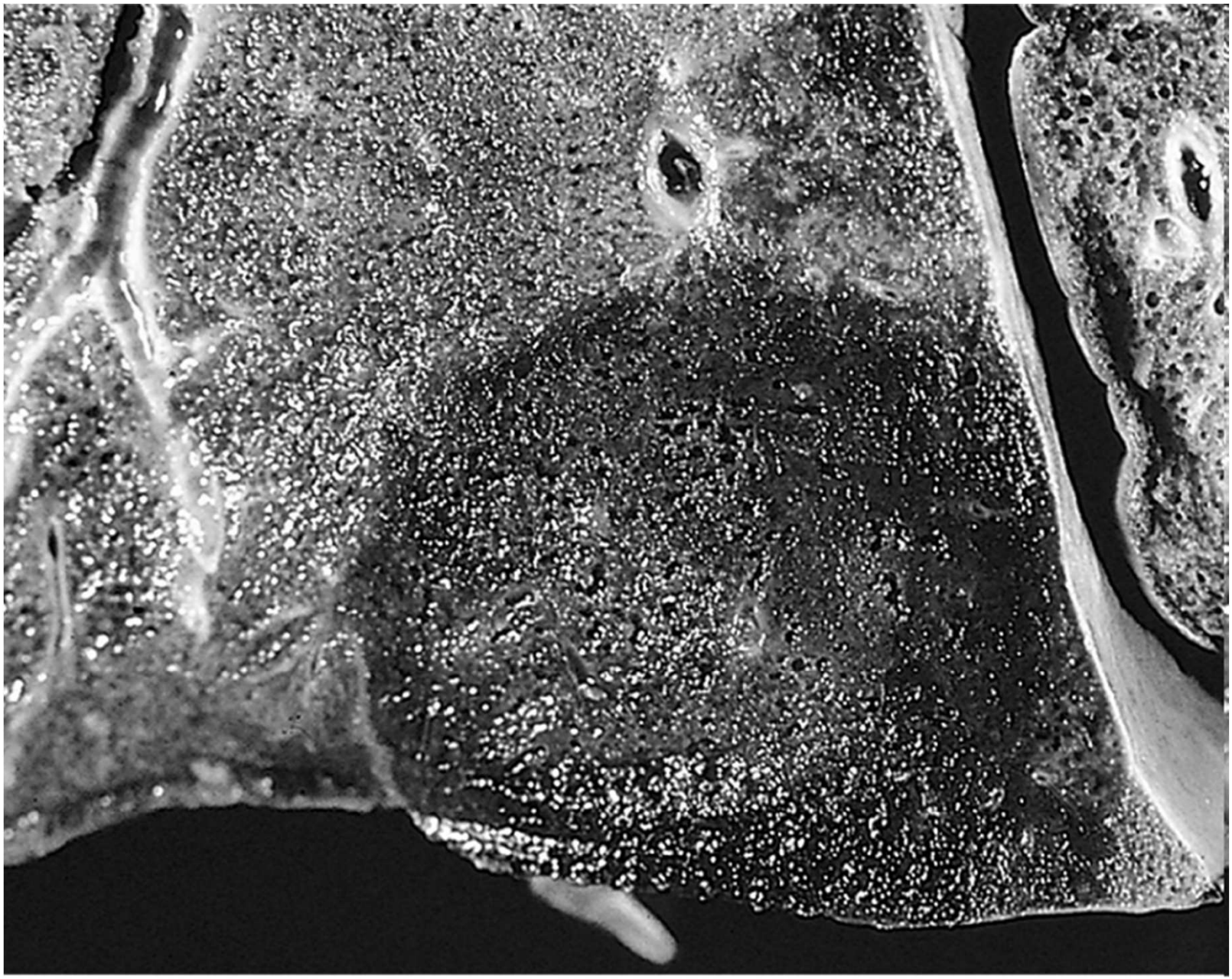
Thromboembolus in small peripheral artery – if multiple, may cause pulmonary hypertension

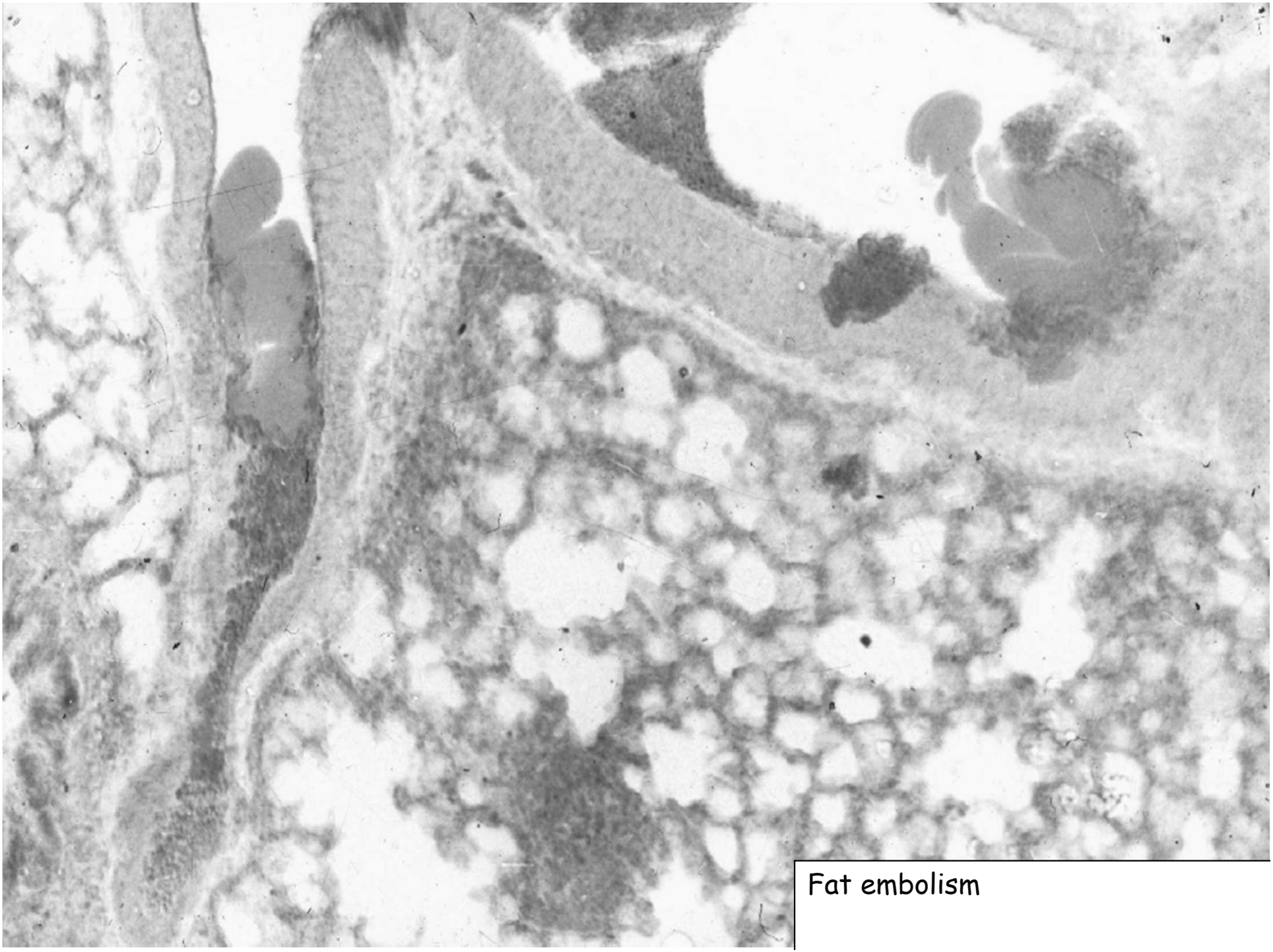


Fibrous connective tissue bands as residuum of old thromboembolism

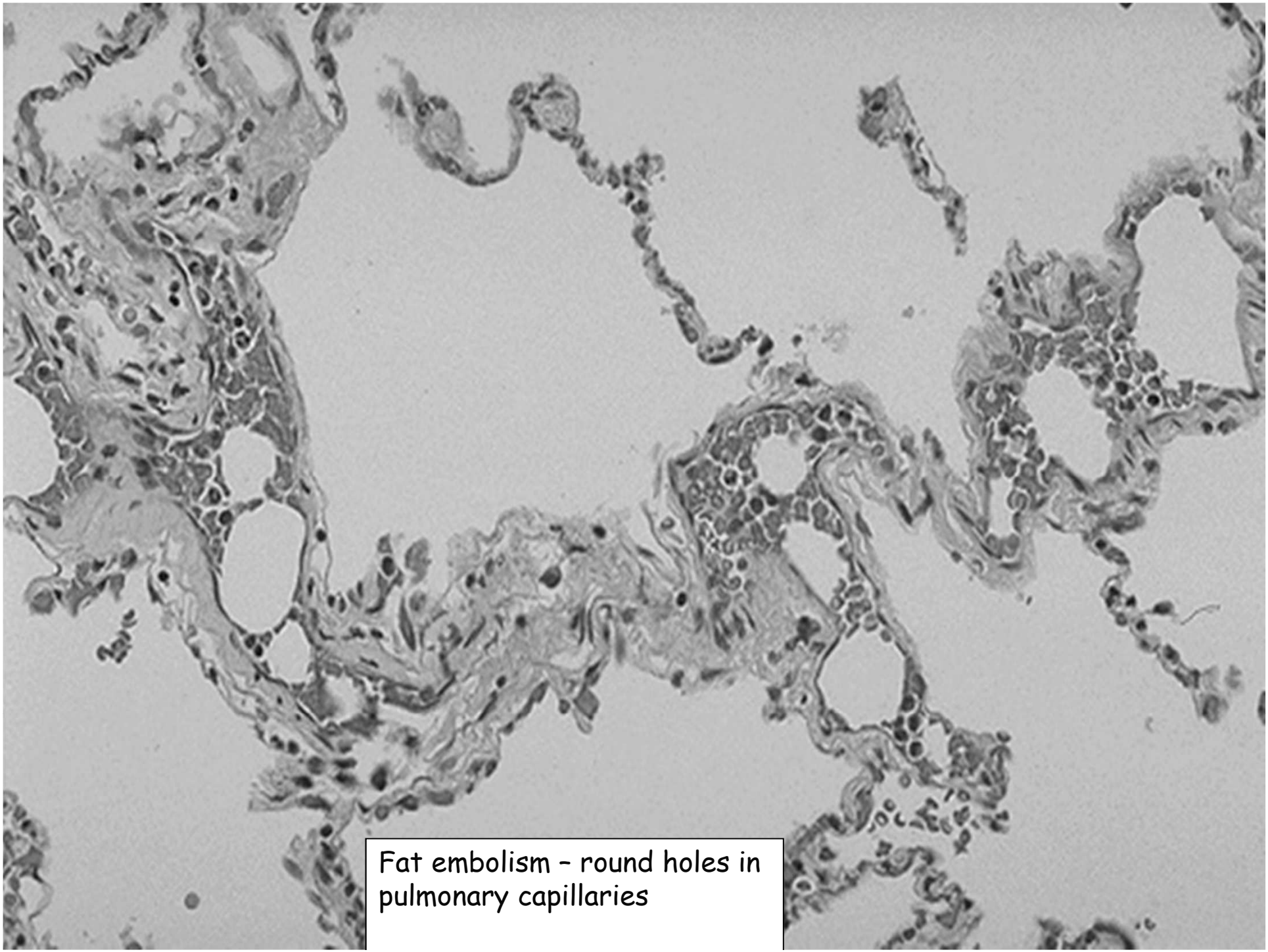


Pulmonary infarction

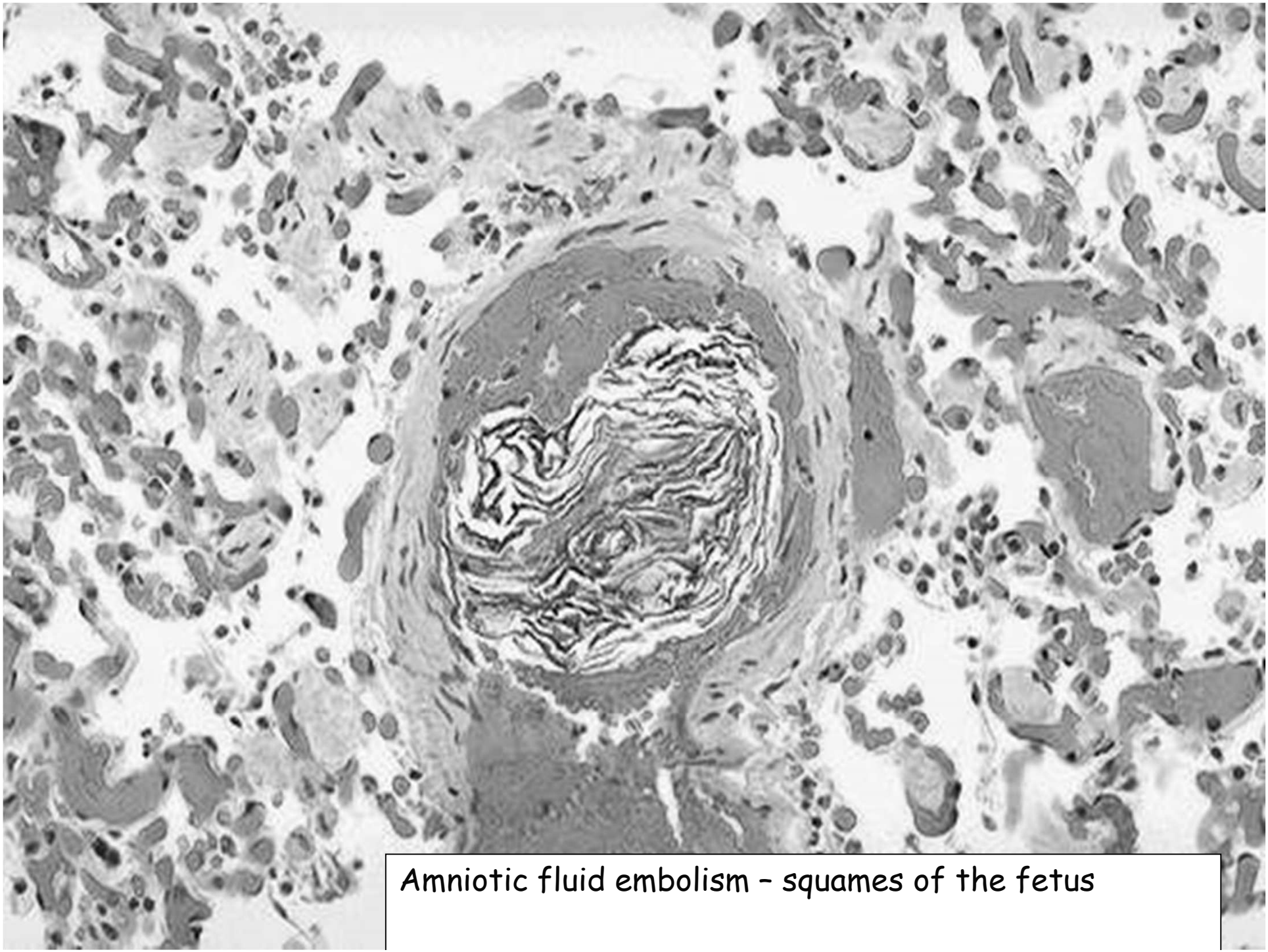




Fat embolism

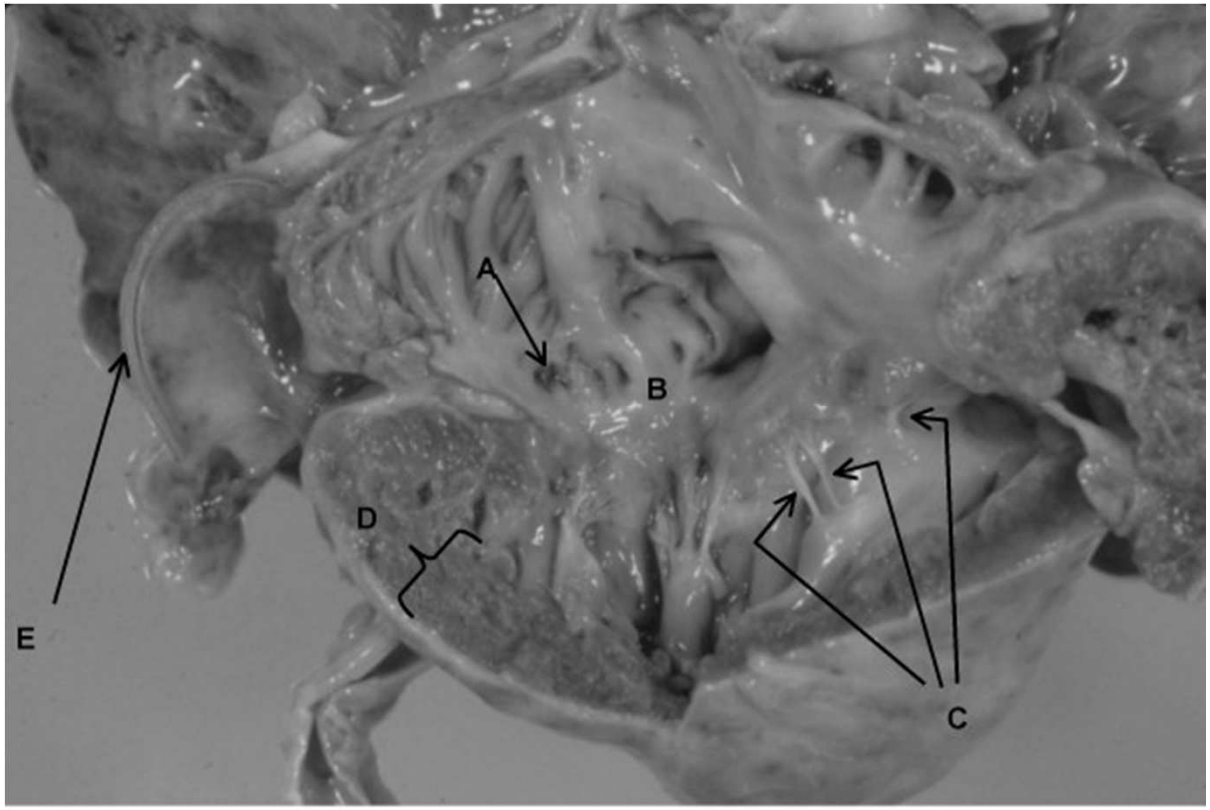


Fat embolism – round holes in pulmonary capillaries



Amniotic fluid embolism – squames of the fetus

Pulmonary hypertension



Pulmonary hypertension

mean pulmonary pressures reach one-fourth or more of systemic levels

Most often

secondary to a decrease in the cross-sectional area of the pulmonary vascular bed, or to increased pulmonary vascular blood flow.

Secondary pulmonary hypertension

Chronic obstructive or interstitial lung disease

Recurrent pulmonary emboli

Antecedent heart disease, -ex.: mitral stenosis

Congenital left-to-right shunts

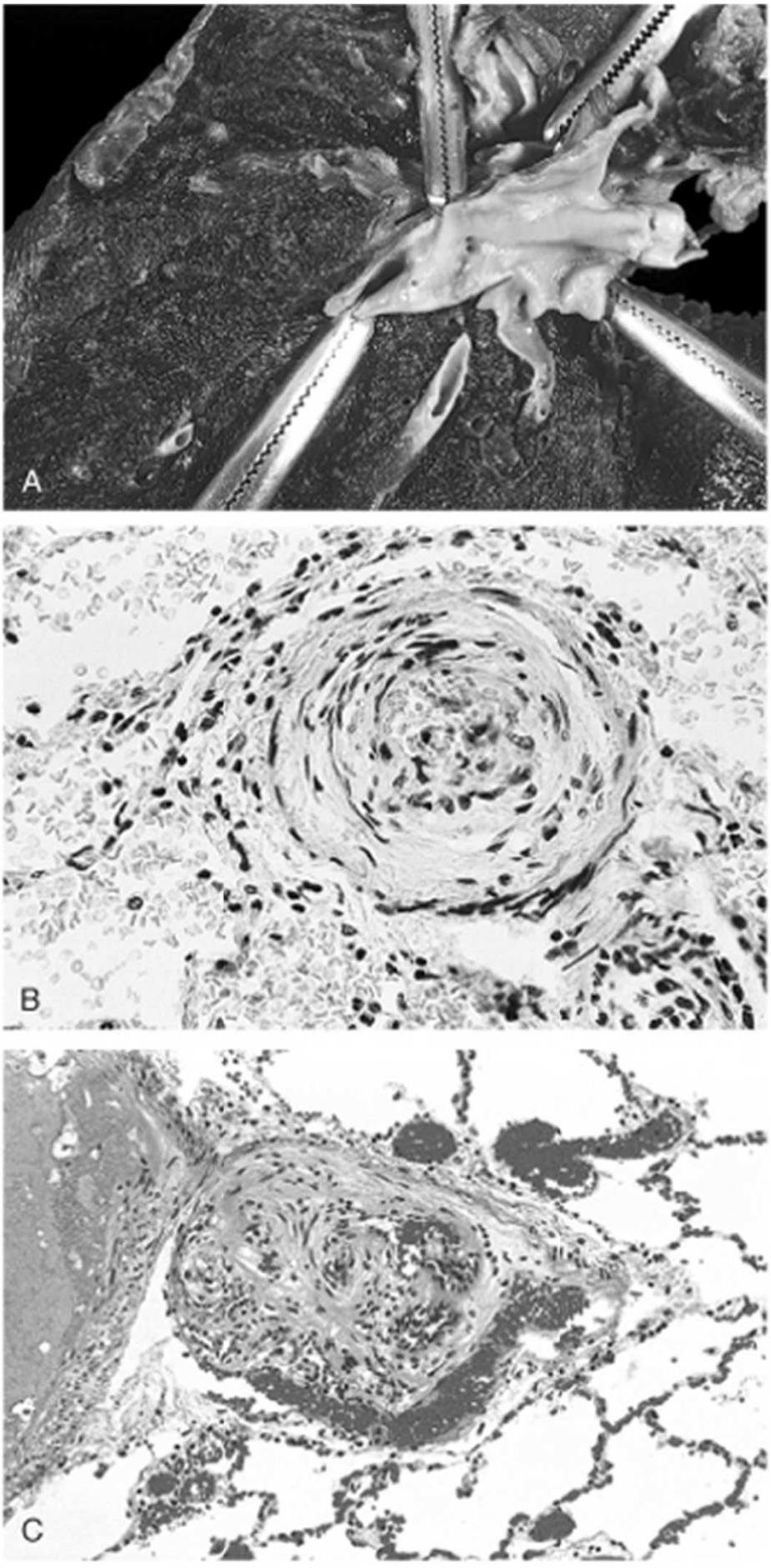
Primary pulmonary hypertension

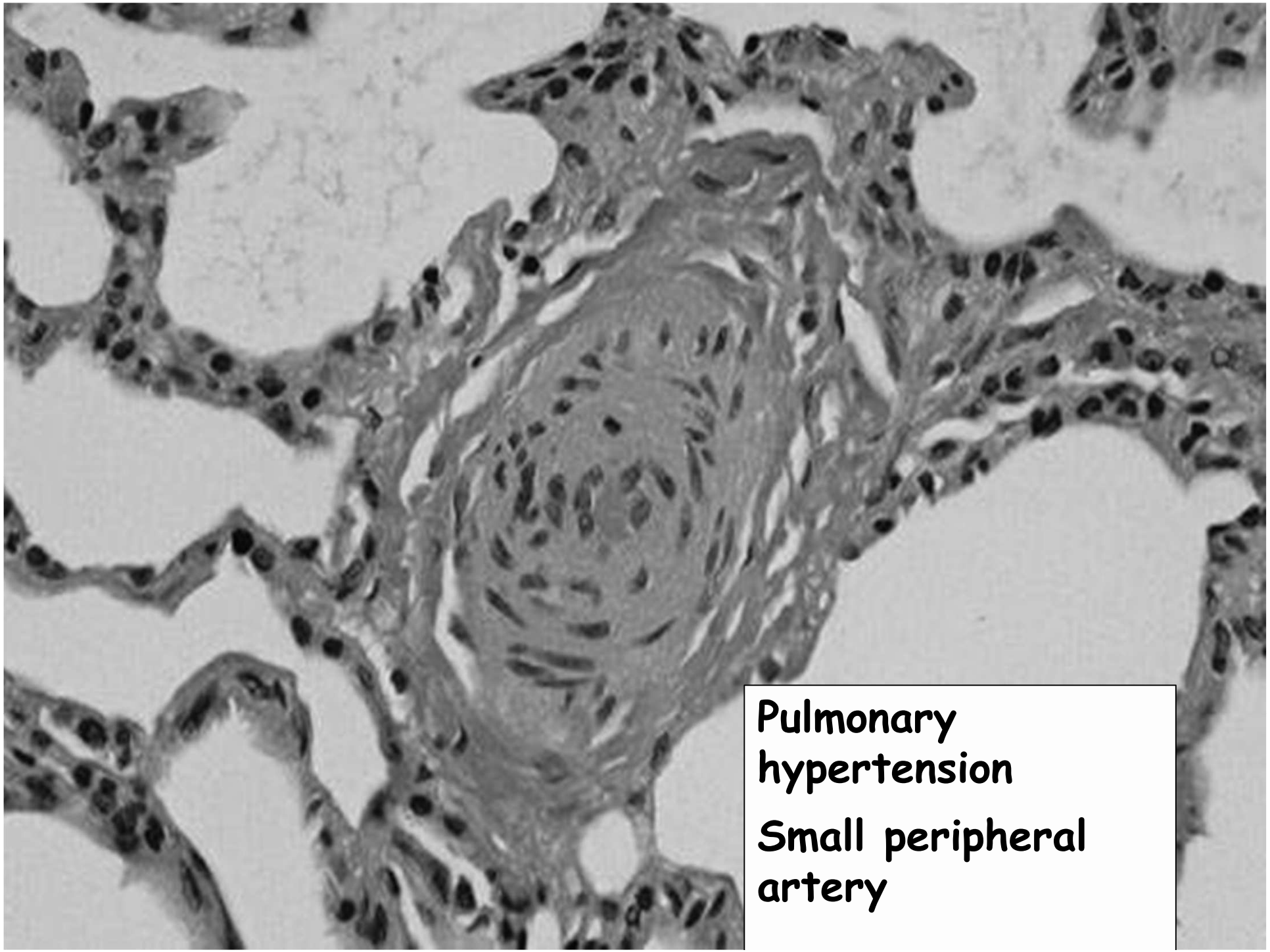
Young women

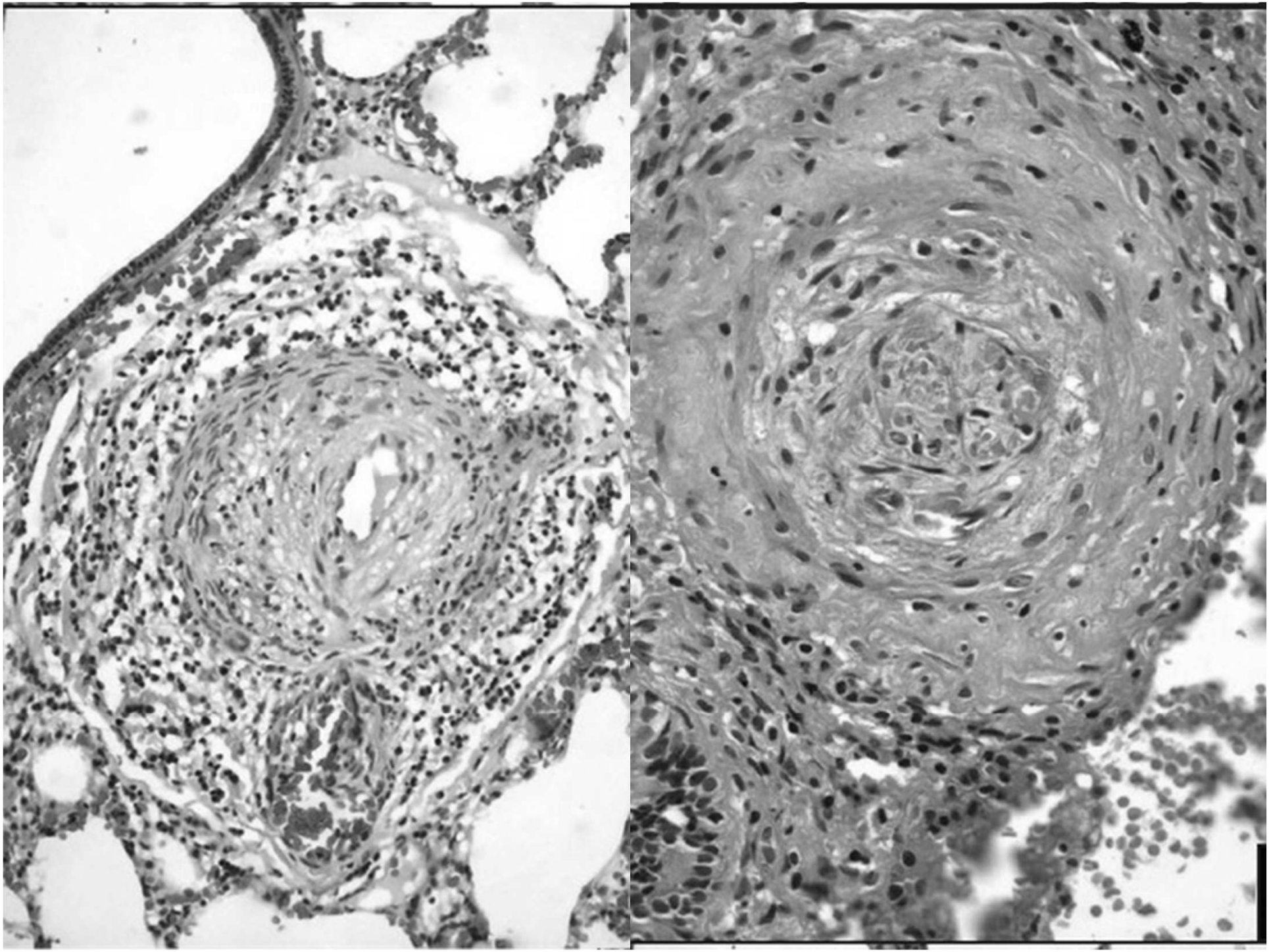
symptoms:

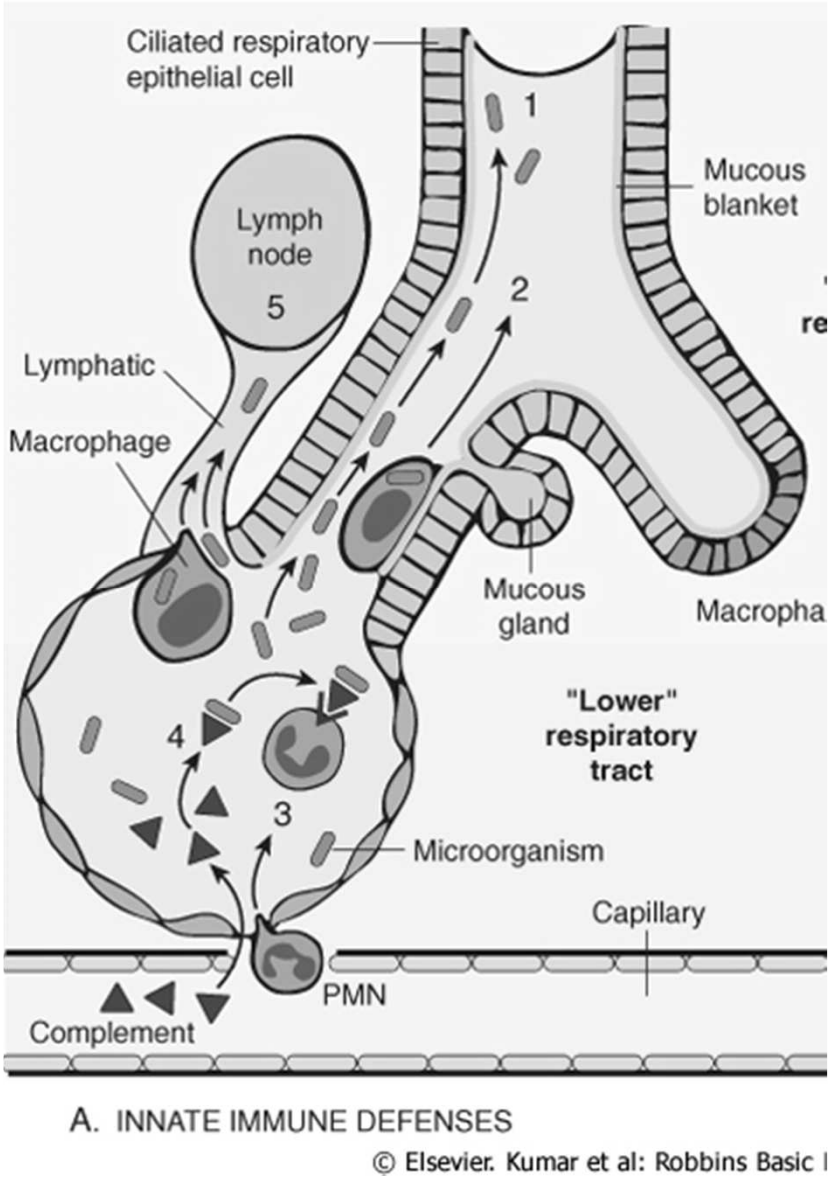
- fatigue, syncope dyspnea on exertion,
- chest pain,
- severe respiratory insufficiency,
- cyanosis,
- death - from right-sided heart failure

2-5 years after diagnosis




Pulmonary hypertension
Small peripheral artery





Lung defense mechanisms.

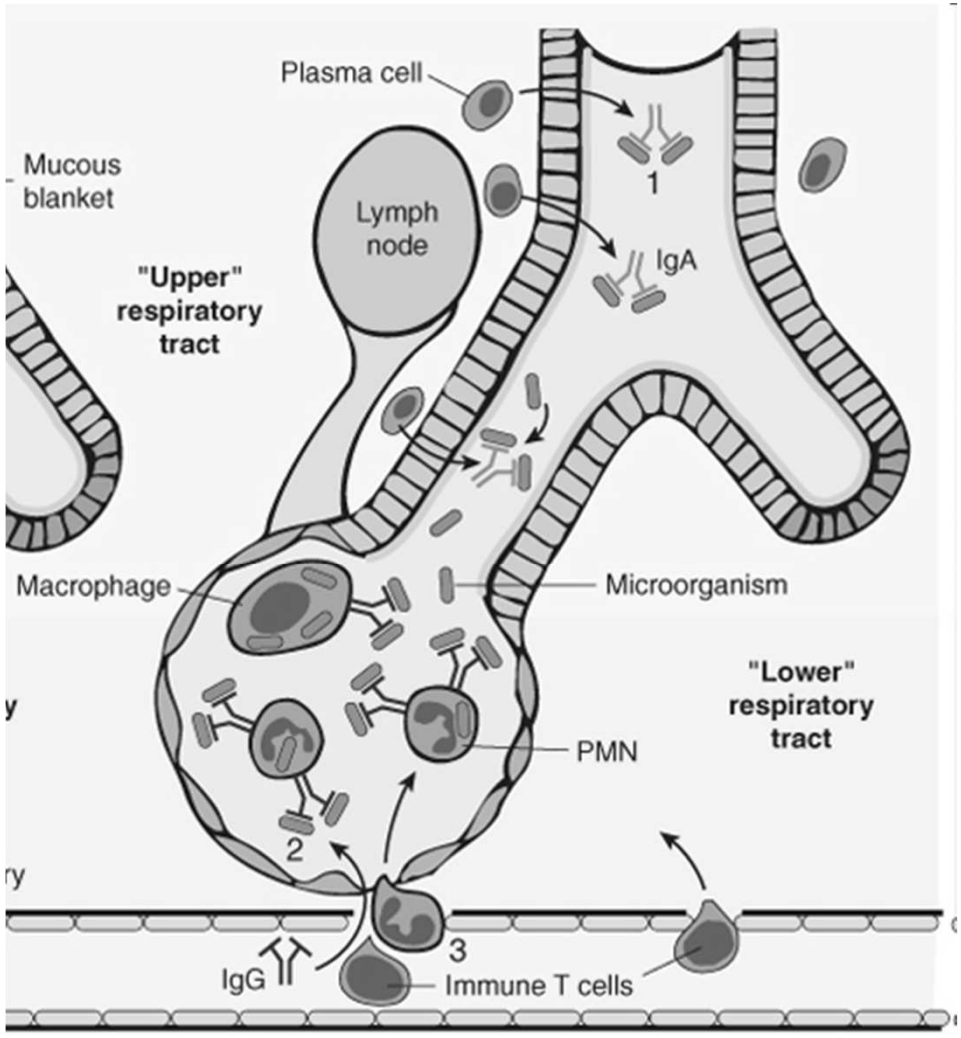
(1) In the nonimmune lung, removal of microbial organisms depends on entrapment in the mucous blanket and removal via the mucociliary elevator,

(2) Phagocytosis by alveolar macrophages that can kill and degrade organisms and remove them from the airspaces by migrating onto the mucociliary elevator, or

(3) Phagocytosis and killing by neutrophils recruited by macrophage factors.

(4) Serum complement may enter the alveoli and be activated by the alternative pathway to provide the opsonin C3b that enhances phagocytosis.

(5) Organisms, including those ingested by phagocytes, may reach the draining lymph nodes to initiate immune responses.



B. ADAPTIVE IMMUNE DEFENSES

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After development of adaptive immunity. (1) Secreted IgA can block attachment of the microorganism to epithelium in the upper respiratory tract.

(2) In the lower respiratory tract, serum antibodies (IgM, IgG) are present in the alveolar lining fluid. They activate complement more efficiently by the classic pathway, yielding C3b (not shown). In addition, IgG is opsonic.

(3) The accumulation of immune T cells is important for controlling infections by viruses and other intracellular microorganisms..

Pulmonary infections

Inhalation of virulent microorganisms

Lowered defense

Pulmonary infections

Bronchopneumonia

Lobar pneumonia

Fever, headache,

Chills

Cough (productive)

Cyanosis

Dyspnoe

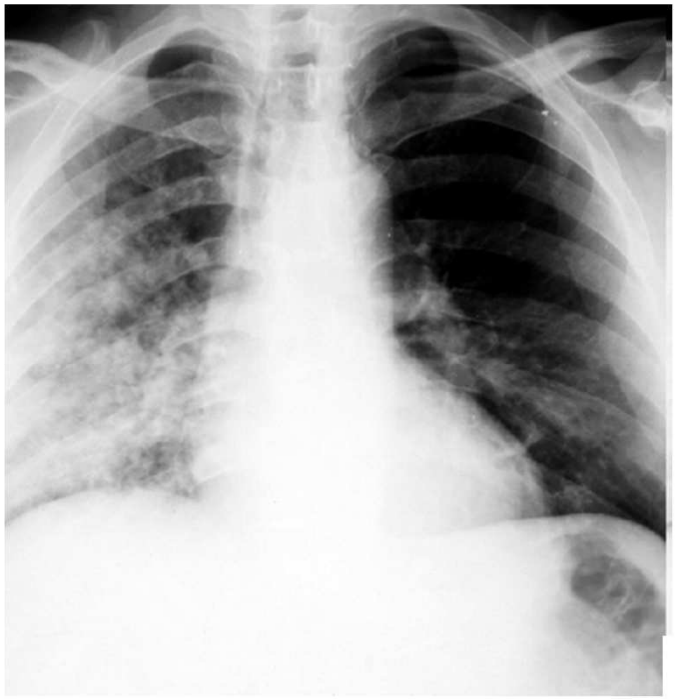
Crepitation

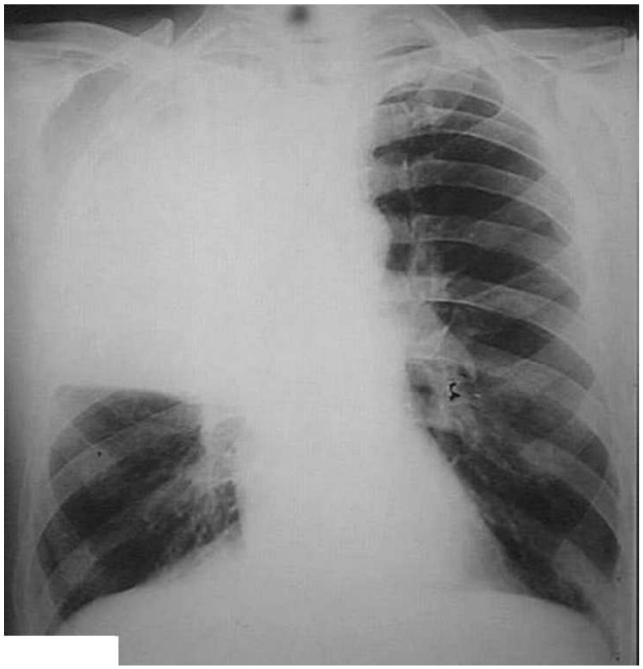
Consolidation

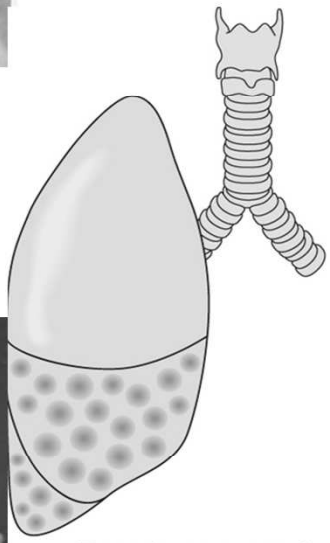
Atypical (interstitial) pneumonia

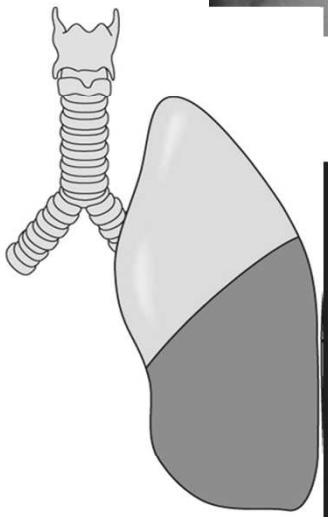
fever, headache

cough with minimal sputum. respiratory distress seemingly out of proportion to the physical and radiographic findings.

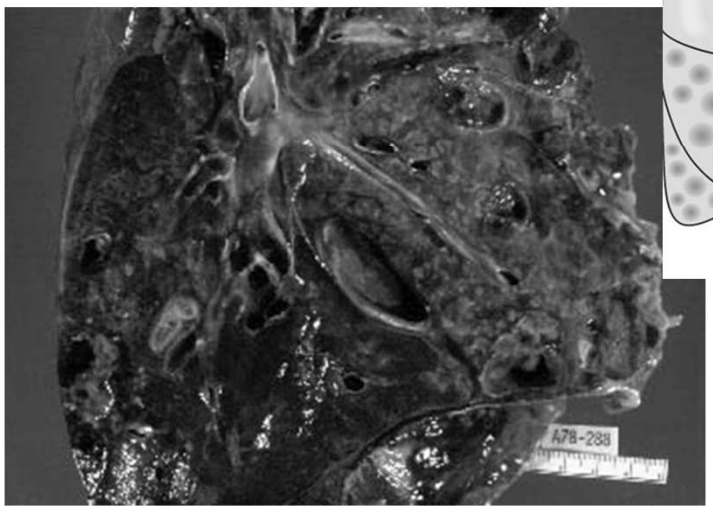


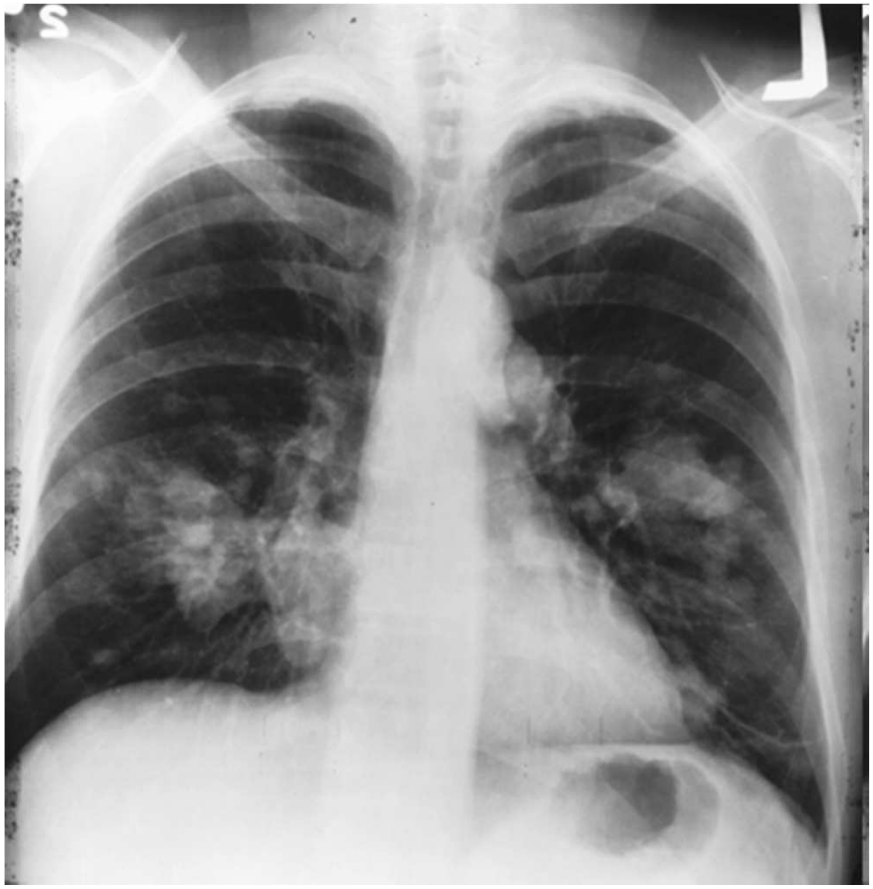






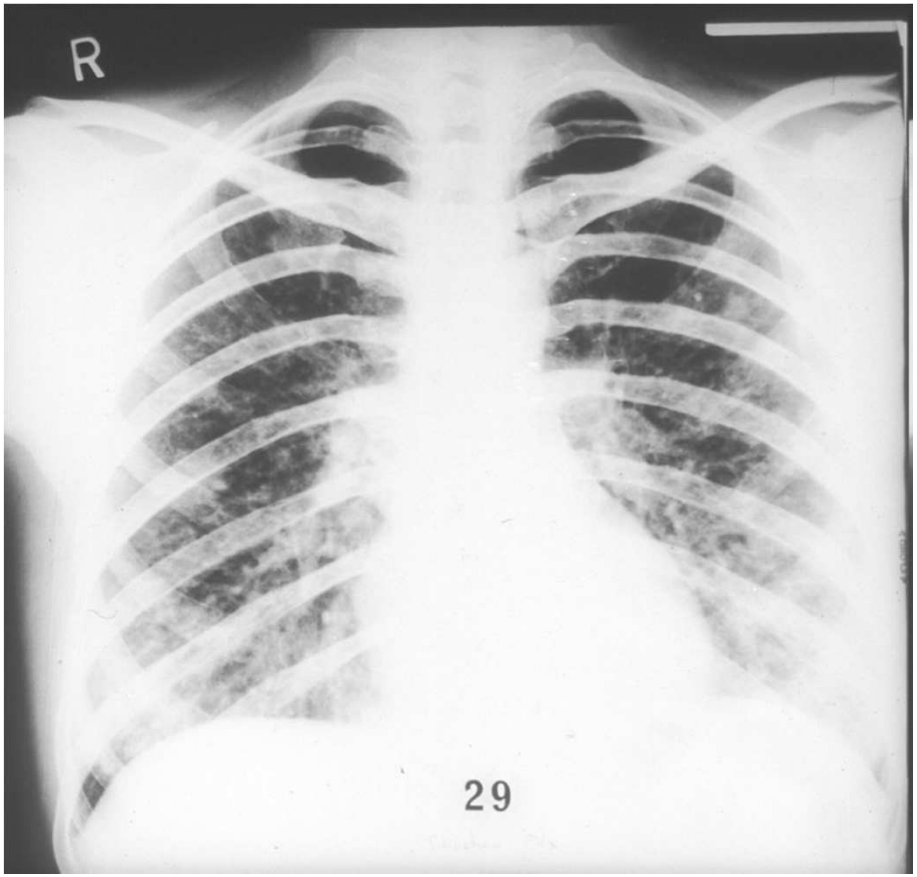
Bronchopneumonia Lobar pneumonia





Bacterial bronchopneumonia

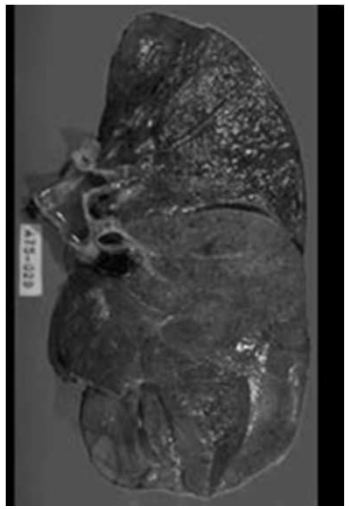
Patchy consolidation

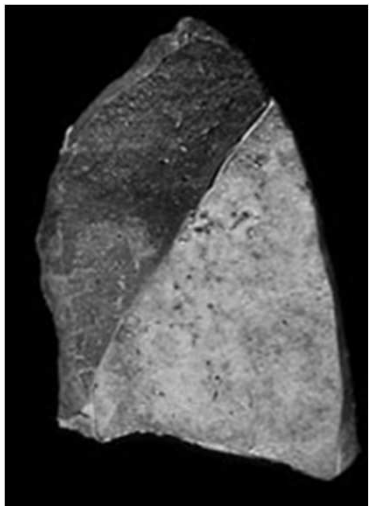


Varicella pneumonia

Increase in interstitial shadowing

Lobar pneumonia



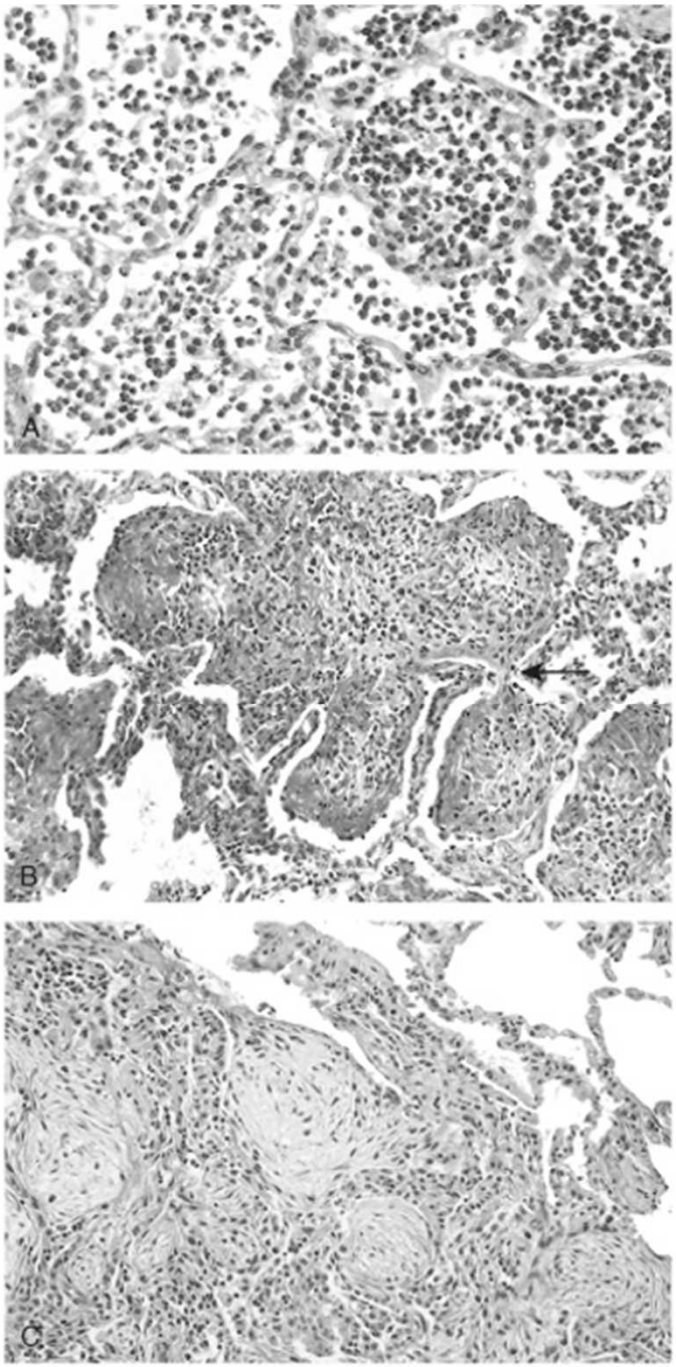


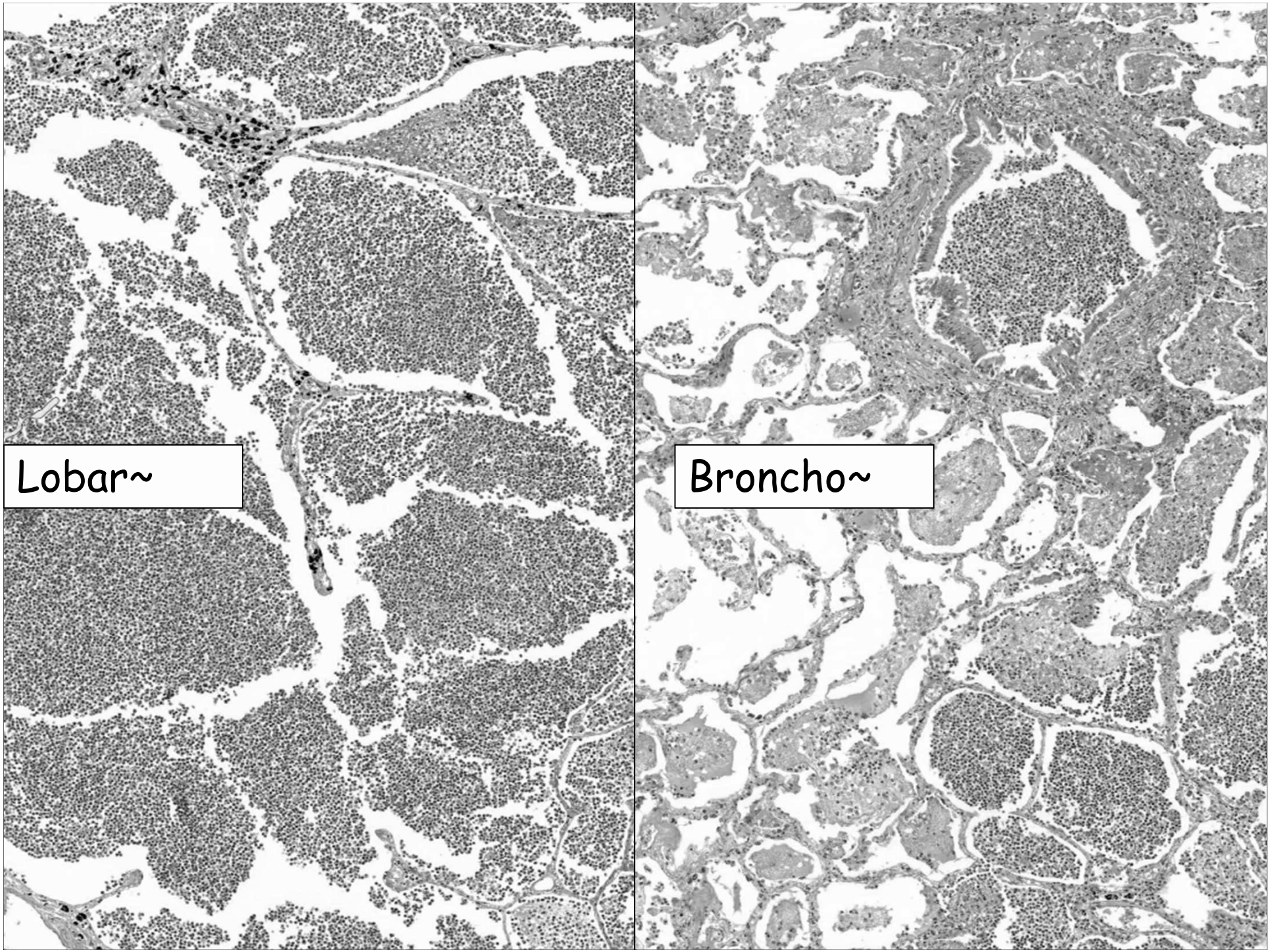
Stages

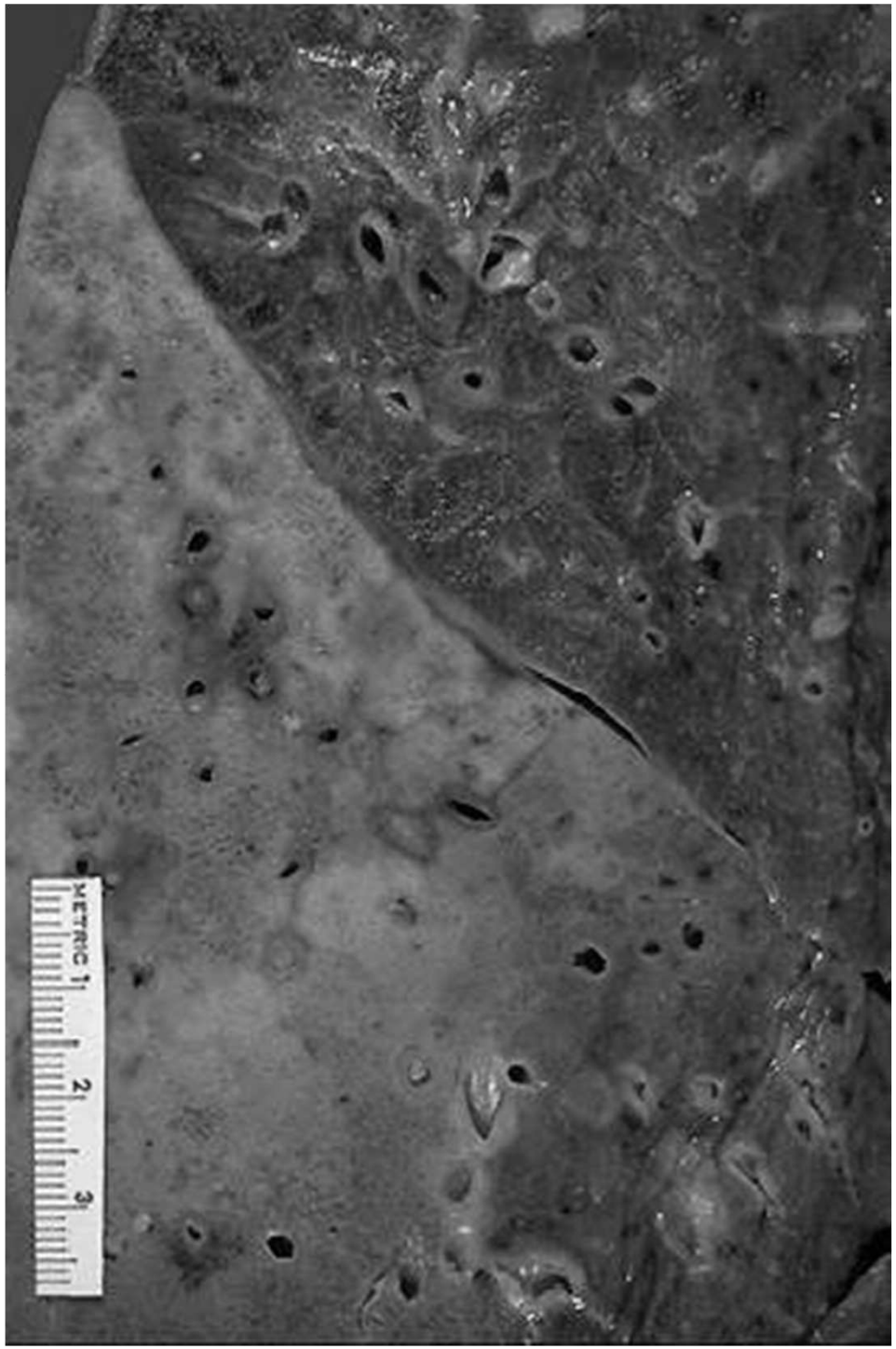
- Congestion
- Hepatization

(red, gray, yellow)

- Resolution

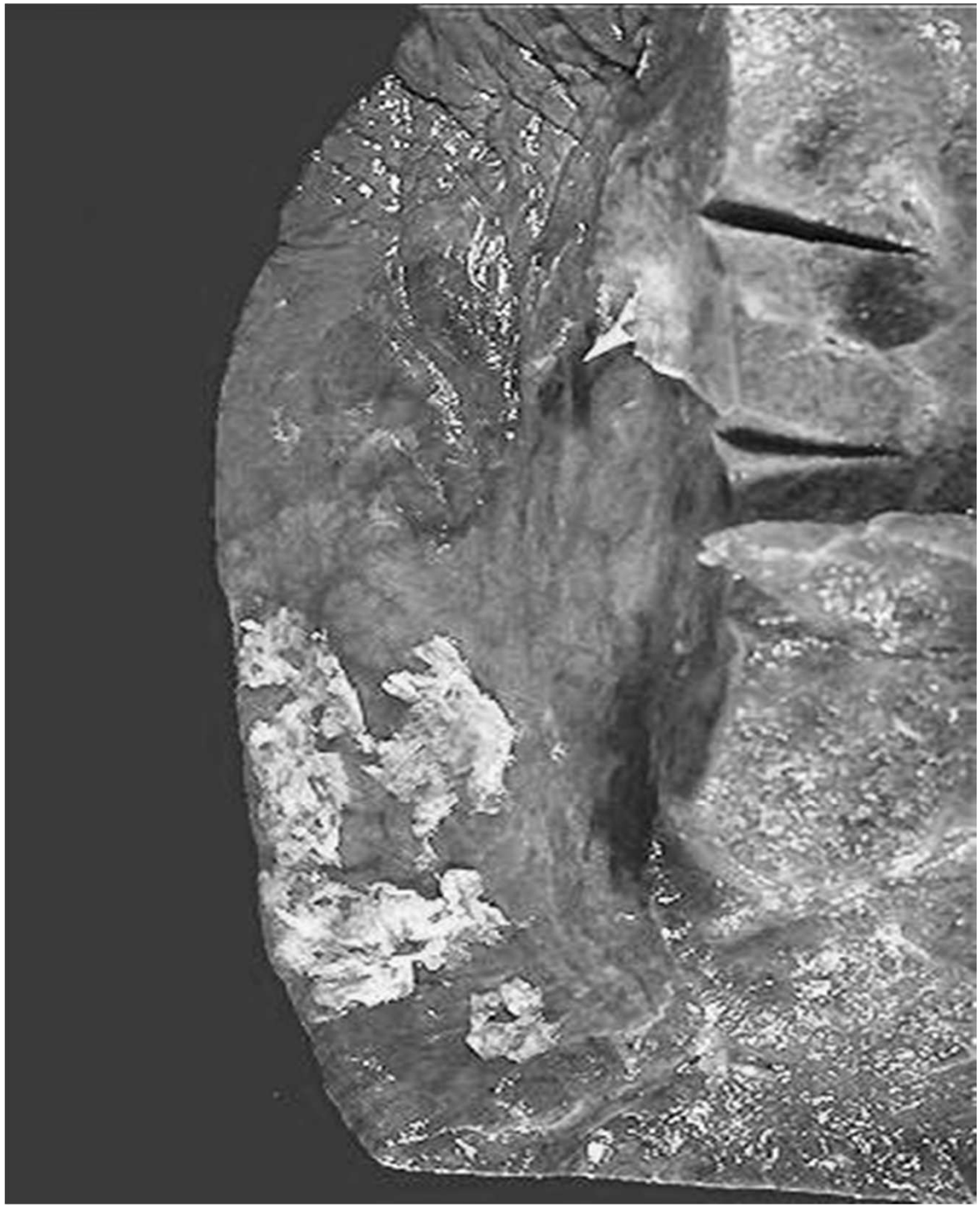



Lobar~
Broncho~



Complications

- Abscess formation
- Empyema
- Carnification
- Metastatic abscesses
    - Meningitis
    - Endocarditis
    - Arthritis

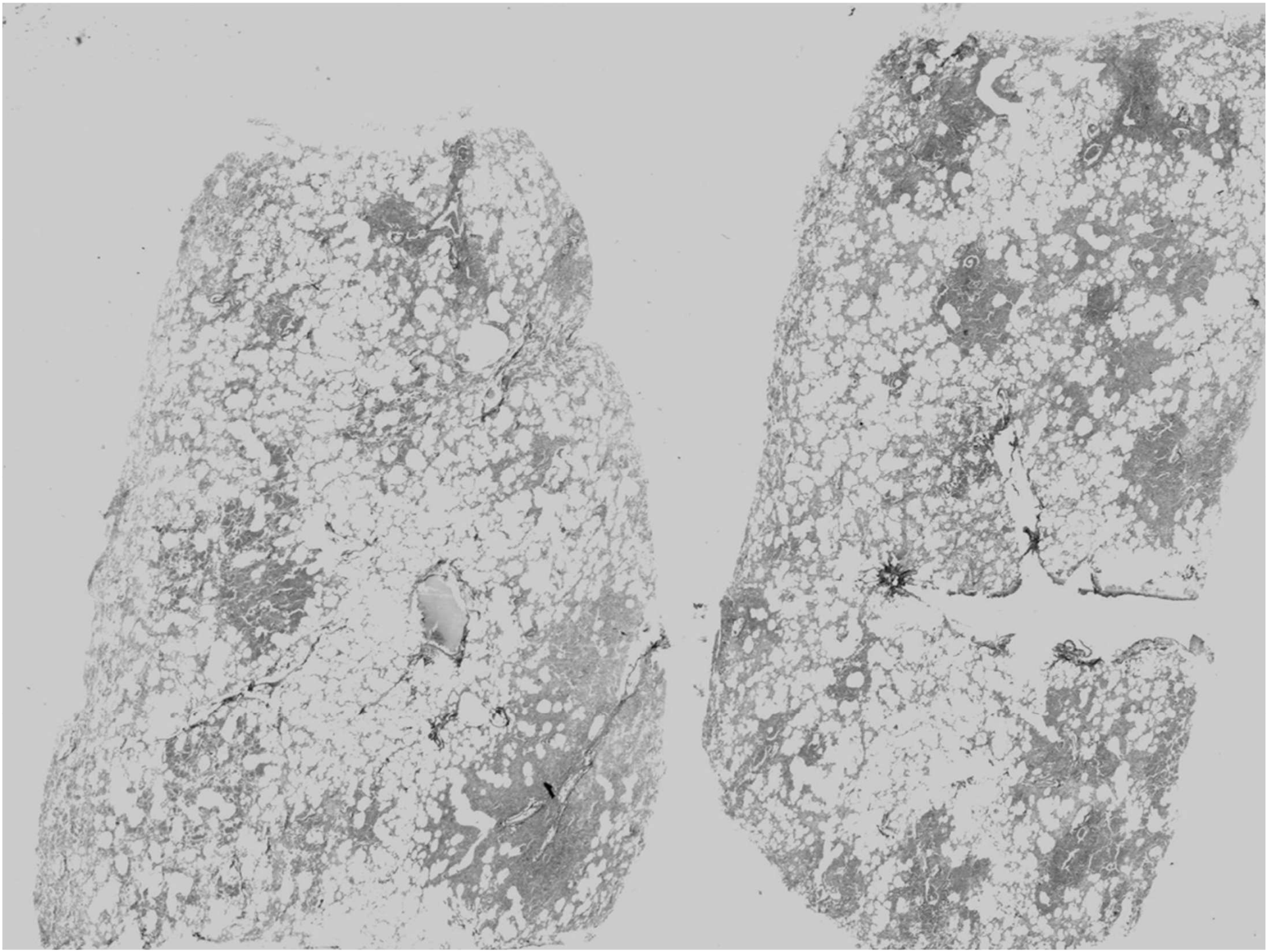


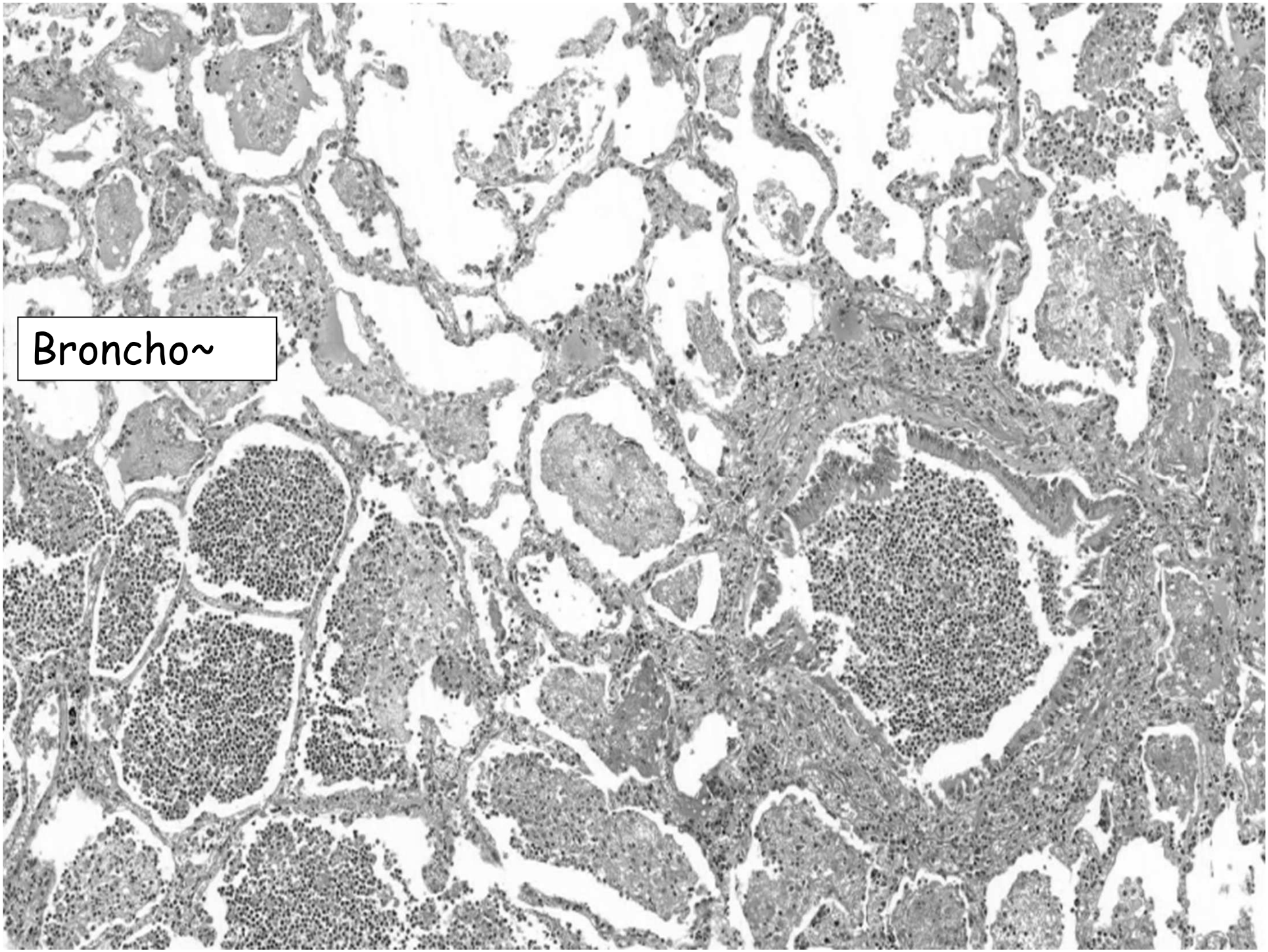
Patchy distribution of a bronchopneumonia -

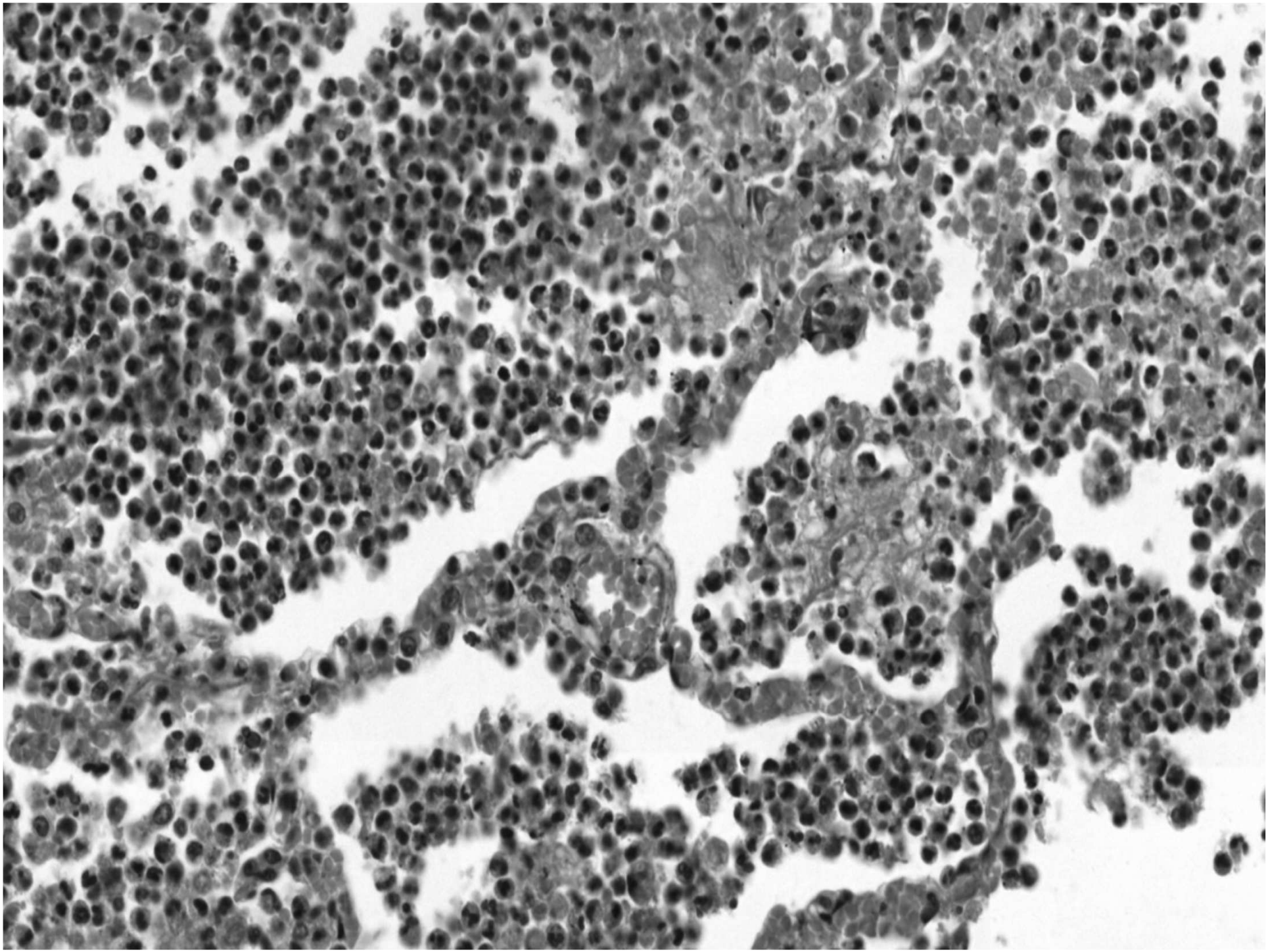
Consolidated areas closely match the pattern of lung lobules
<u>"lobular" pneumonia</u>

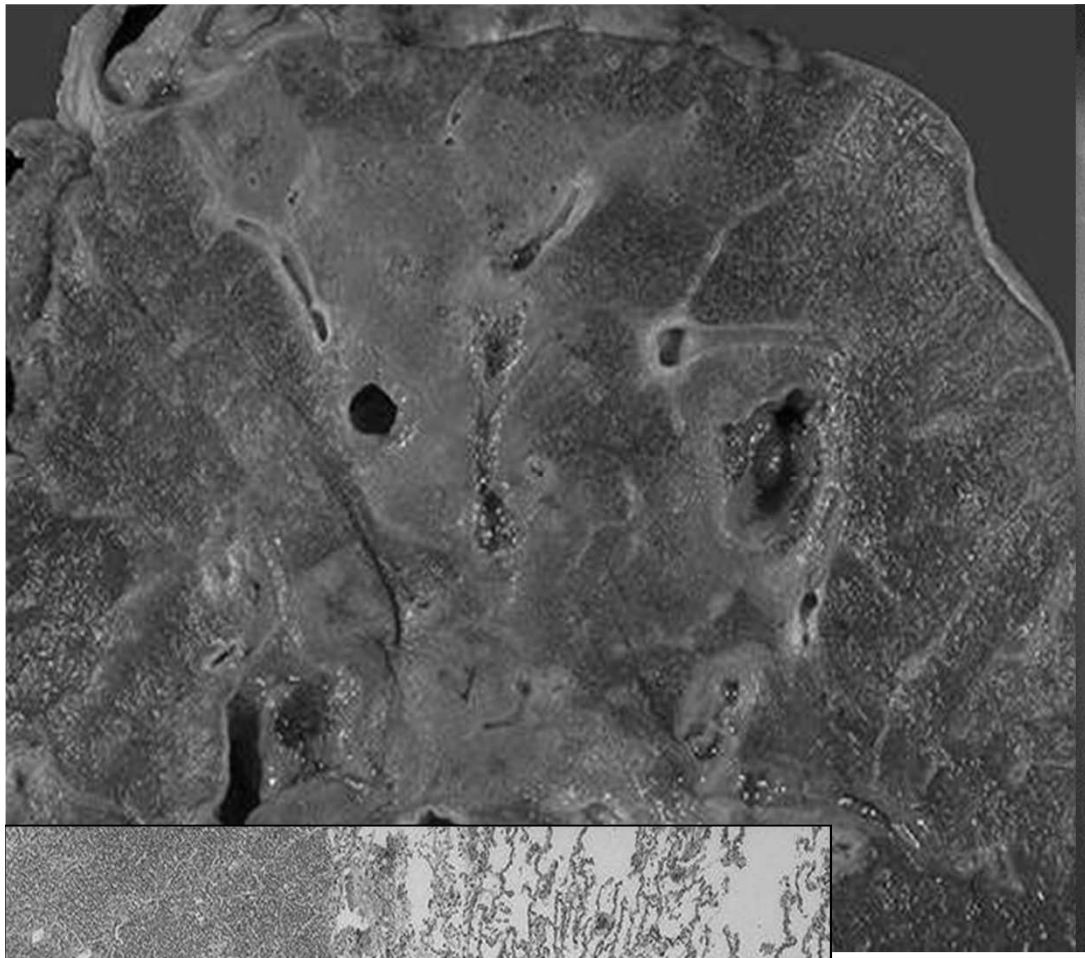
Typical bacterial organisms include:

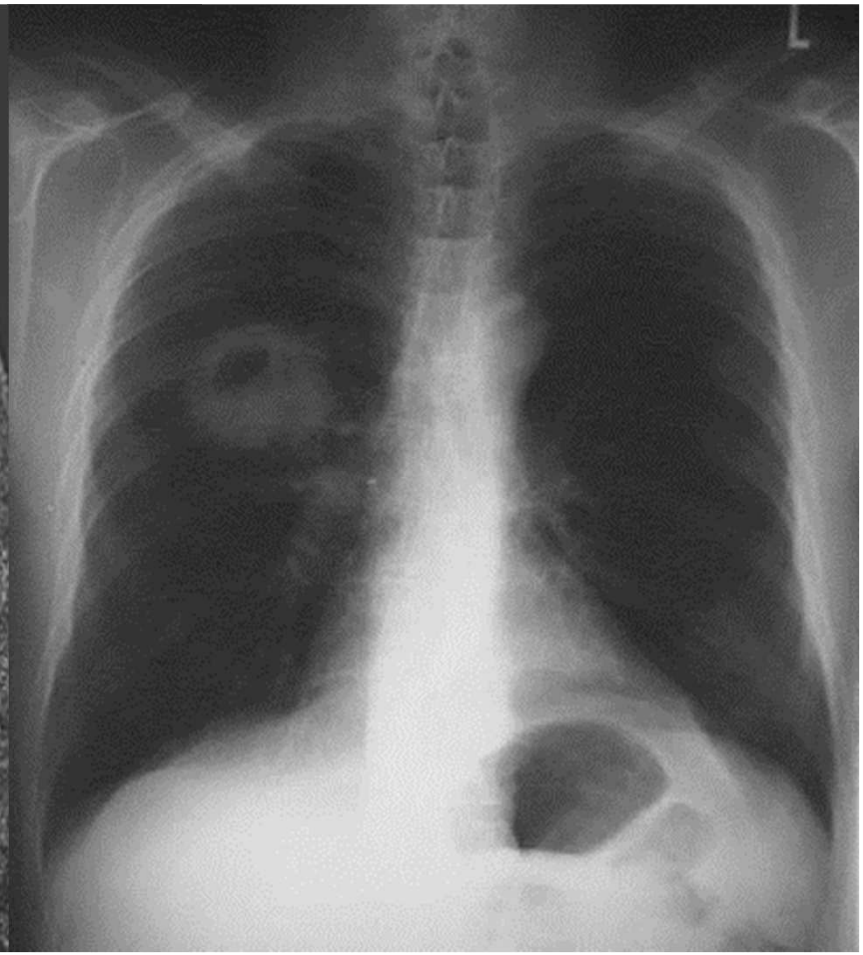
- **Haemophilus Influenzae**
- **Moraxella catarrhalis**
- **Staphylococcus aureus**
- **Klebsiella**
- **Pseudomonas**
- **Legionella**



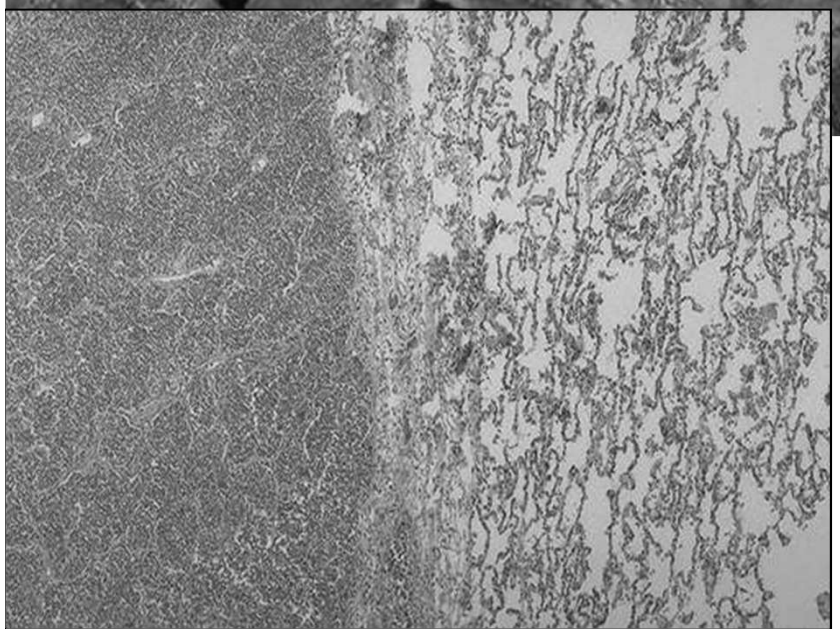

Broncho~





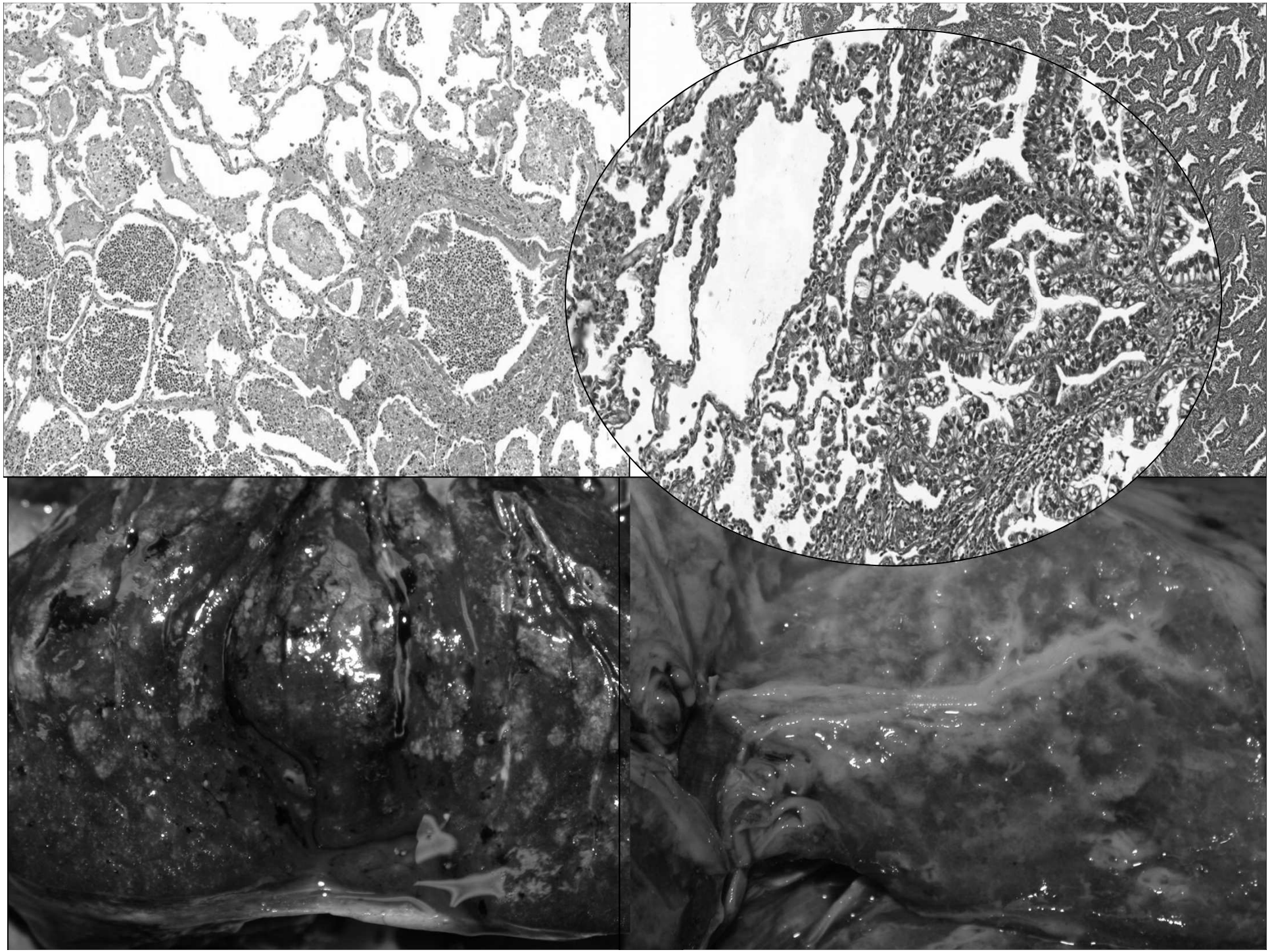


Air-fluid level

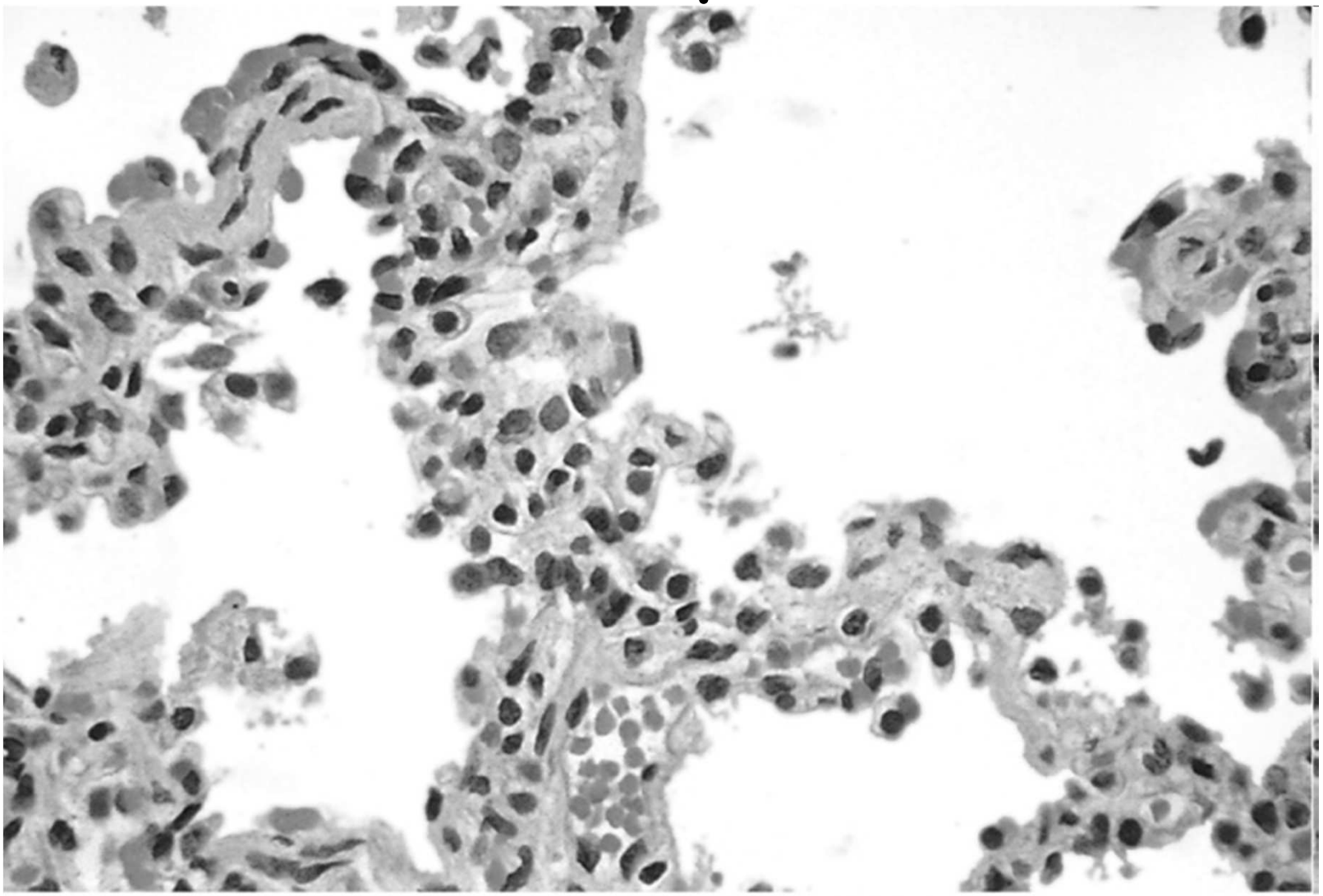


Bronchopneumonia

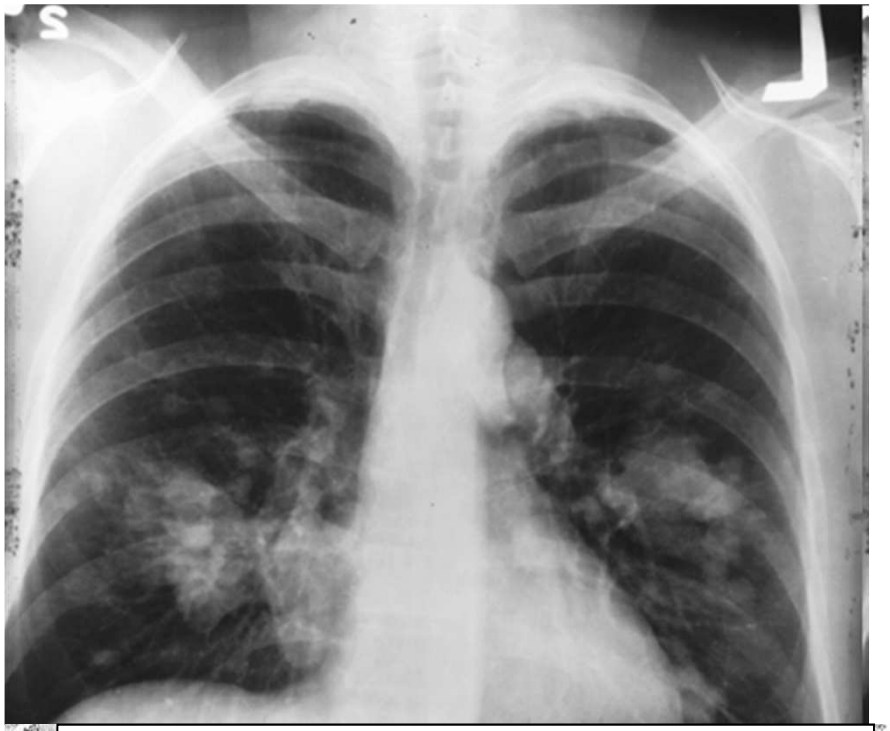
Abscess formation



Interstitial pneumonia

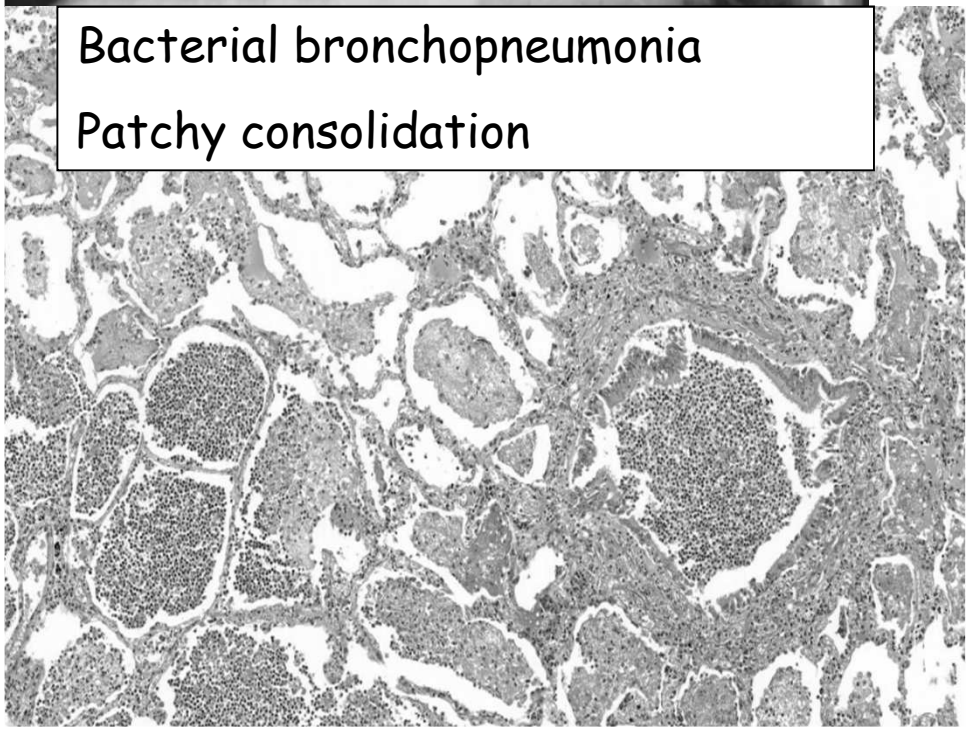


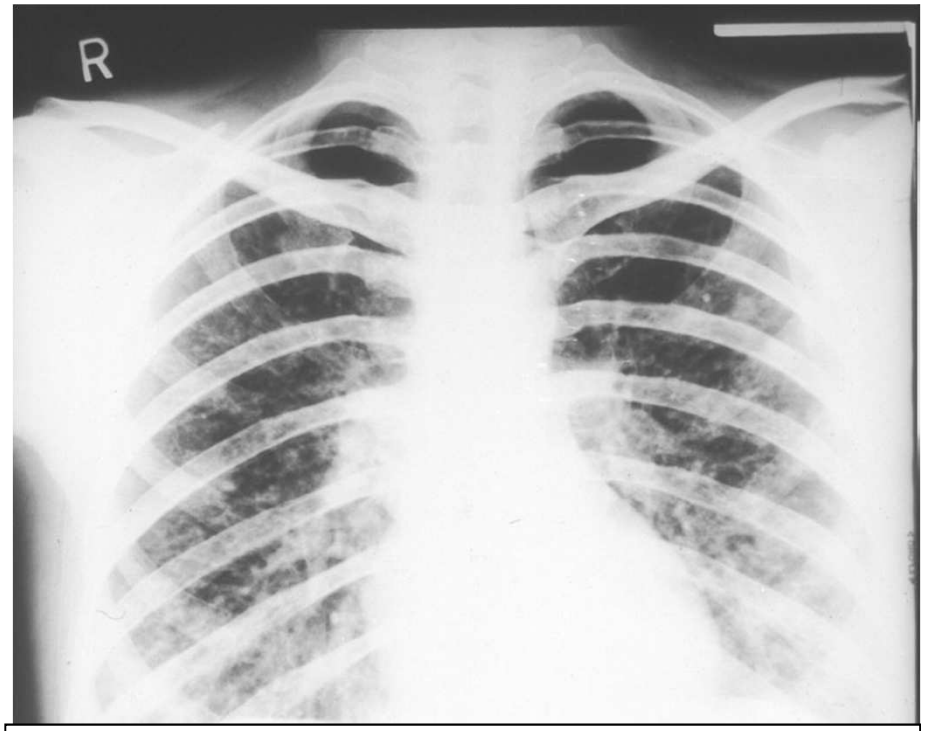
Thickened alveolar walls are heavily infiltrated with mononuclear leukocytes



Bacterial bronchopneumonia

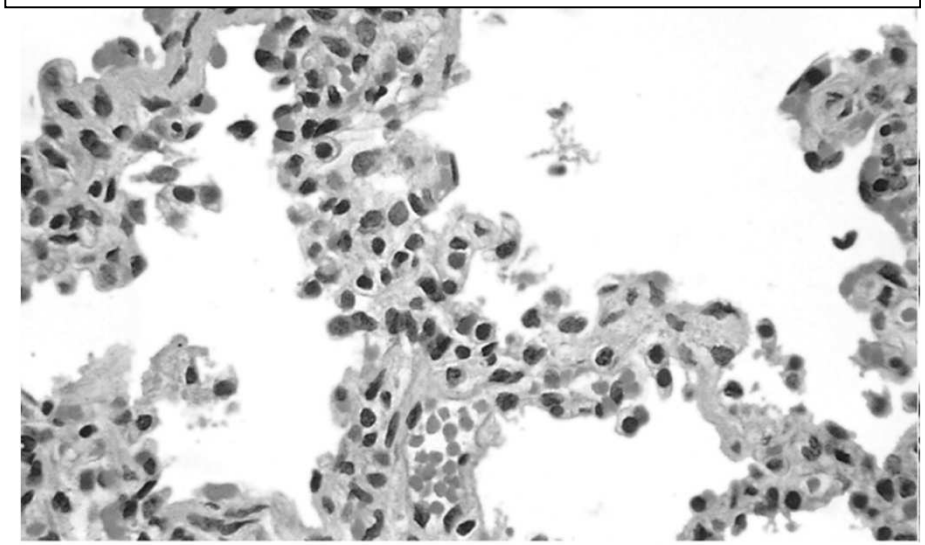
Patchy consolidation





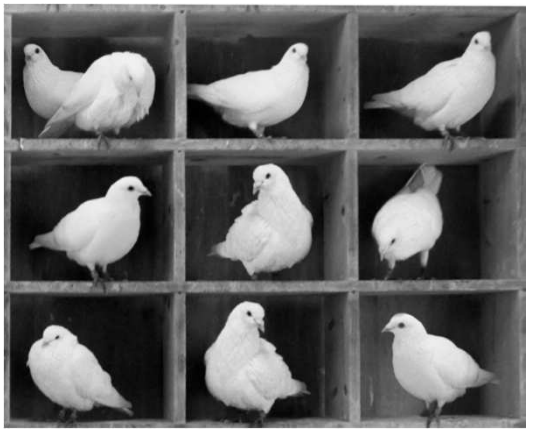
Varicella pneumonia

Increase in interstitial shadowing



Pulmonary infections

- Community-Acquired Acute Pneumonia
- Community-Acquired Atypical Pneumonia
- Nosocomial Pneumonia
- Aspiration Pneumonia
- Chronic Pneumonia



- Necrotizing Pneumonia and Lung Abscess
- Pneumonia in the Immunocompromised Host

What is the relevance of this classification?

Pulmonary infections

Community-acquired acute pneumonia

- most are bacterial in origin.

- follows a viral upper respiratory tract infection

- onset is usually abrupt

  high fever, shaking chills, pleuritic chest pain, productive mucopurulent cough, occasionally hemoptysis.

S. pneumoniae (or *pneumococcus*) is the most common cause of community-acquired acute pneumonia

Haemophilus Influenzae

encapsulated (children vaccination, meningitis,epiglottitis)
unencapsulated (COPD patients)

Moraxella catarrhalis

elderly, COPD patients, otitis media-children

Staphylococcus aureus

after viral illness, empyema, abscess, drug abusers

Klebsiella

alcoholics, malnourished, thick sputum

Pseudomonas

nosocomial, neutropenic persons (chemoth), angioinvasive

Legionella

immunocompromised-50% fatal Pontiac fever

Community-Acquired Atypical Pneumonia

"***primary atypical pneumonia***"

acute febrile respiratory disease

patchy inflammatory changes in the lungs,

confined to the alveolar septa and pulmonary interstitium.

moderate amounts of sputum,

absence of physical findings of consolidation,

moderate elevation of white cell count,

lack of alveolar exudates.

respiratory distress seemingly out of proportion to the physical and radiographic findings.

Community-Acquired Atypical Pneumonia

"*primary atypical pneumonia*"

Mycoplasma pneumoniae

viruses, – influenza types A, B, RSV, adeno~, rhino ~, rubeola, varicella *Chlamydia pneumoniae* , *Coxiella burnetti* (Q fever)

Sporadic or local epidemics in closed communities (schools, military camps,prisons).

Nearly all agents can also cause a *primarily upper respiratory* tract infection ("common cold").

Nosocomial Pneumonia

Gram-negative rods

S. aureus (MRSA)

Aspiration Pneumonia

partly chemical, - extremely irritating effects of the gastric acid,

partly bacterial

Pulmonary infections

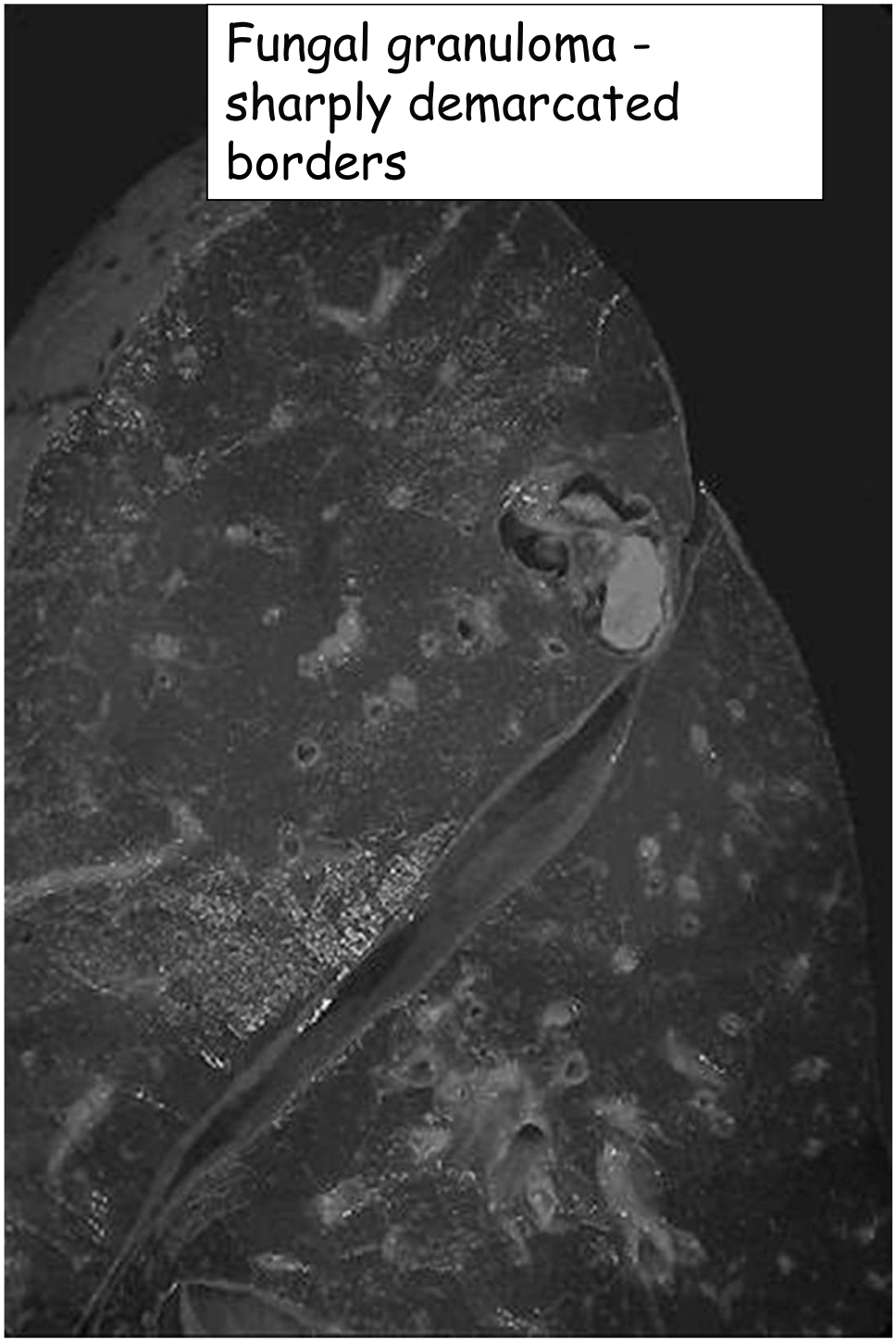
Chronic Pneumonia

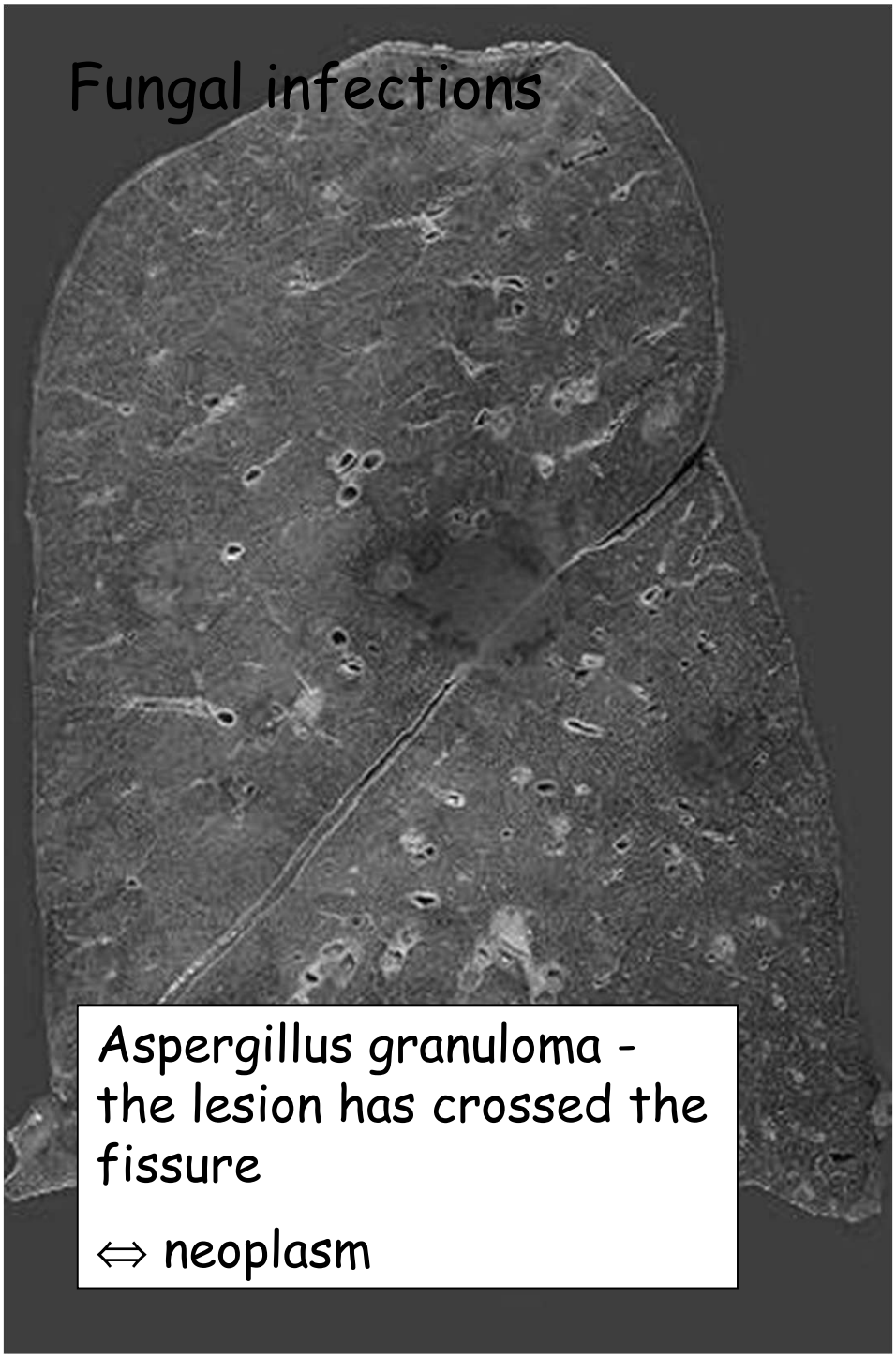
TBC, fungal pneumonias….

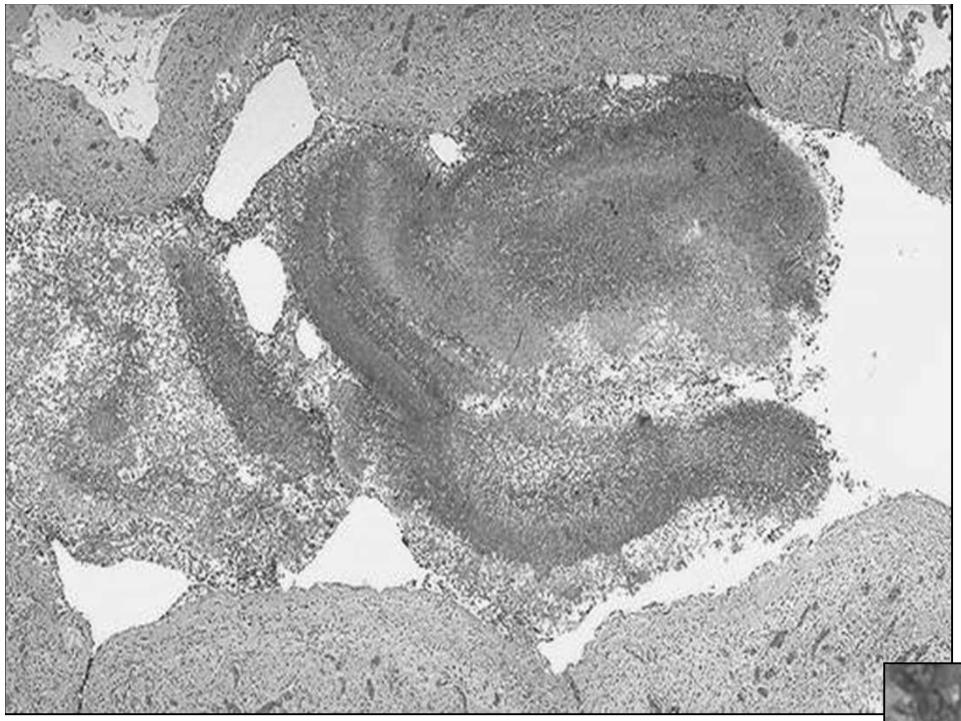
Necrotizing Pneumonia and Lung Abscess – anaerob and Sta

Pneumonia in the Immunocompromised Host

CMV *Pneumocystis jiroveci Mycobacterium AI,* Invasive aspergillosis Invasive candidiasis


Fungal granuloma - sharply demarcated borders

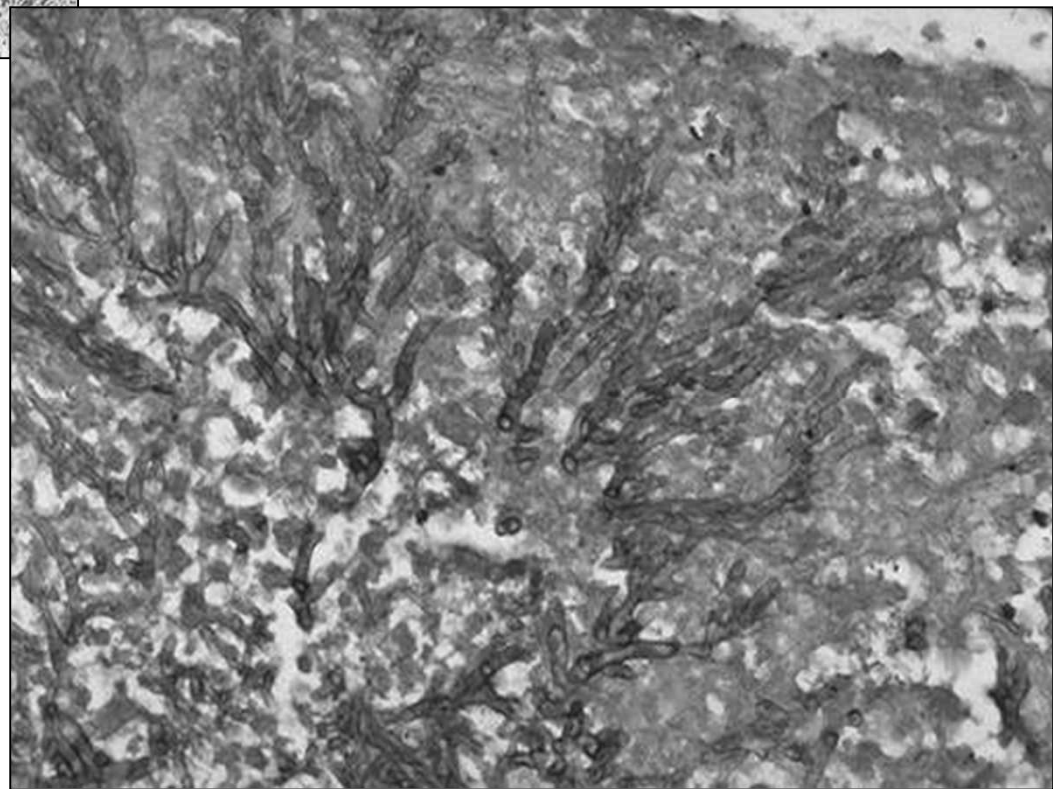

Fungal infections
Aspergillus granuloma - the lesion has crossed the fissure
⇔ neoplasm



Fungal infections

- Aspergillosis
- Candidiasis
- Histoplasmosis
- Blastomycosis
- Coccidiosis

bronchial fungus ball composed of blue-staining hyphal elements of Aspergillus



Pulmonary infections

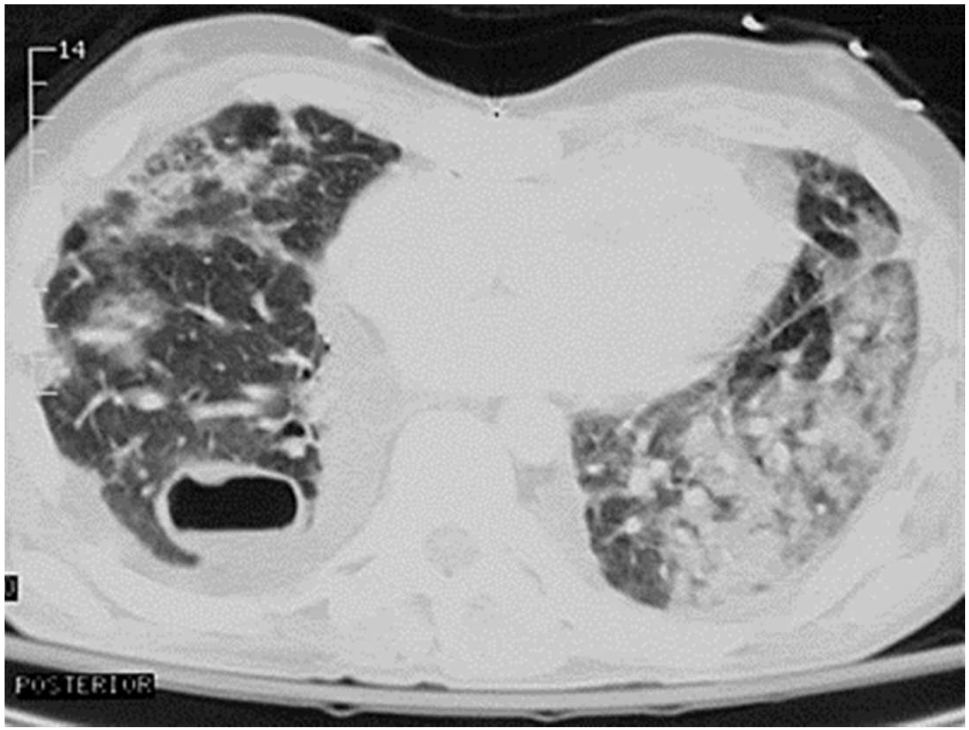
Chronic Pneumonia

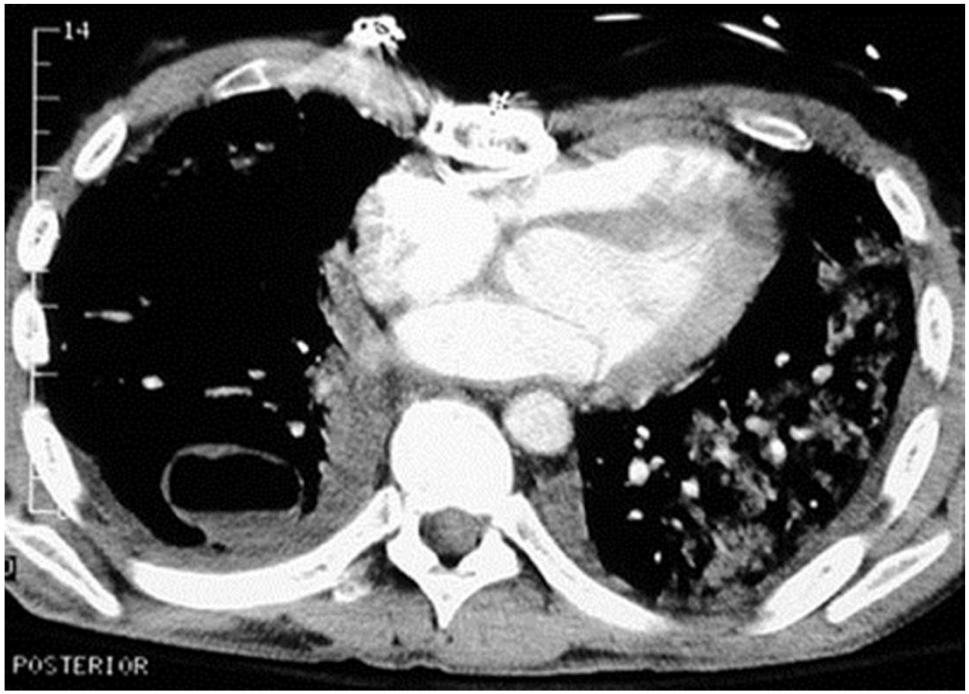
TBC, fungal pneumonias….

Necrotizing Pneumonia and Lung Abscess – anaerob and Sta

Pneumonia in the Immunocompromised Host

CMV *Pneumocystis jiroveci Mycobacterium AI,* Invasive aspergillosis Invasive candidiasis



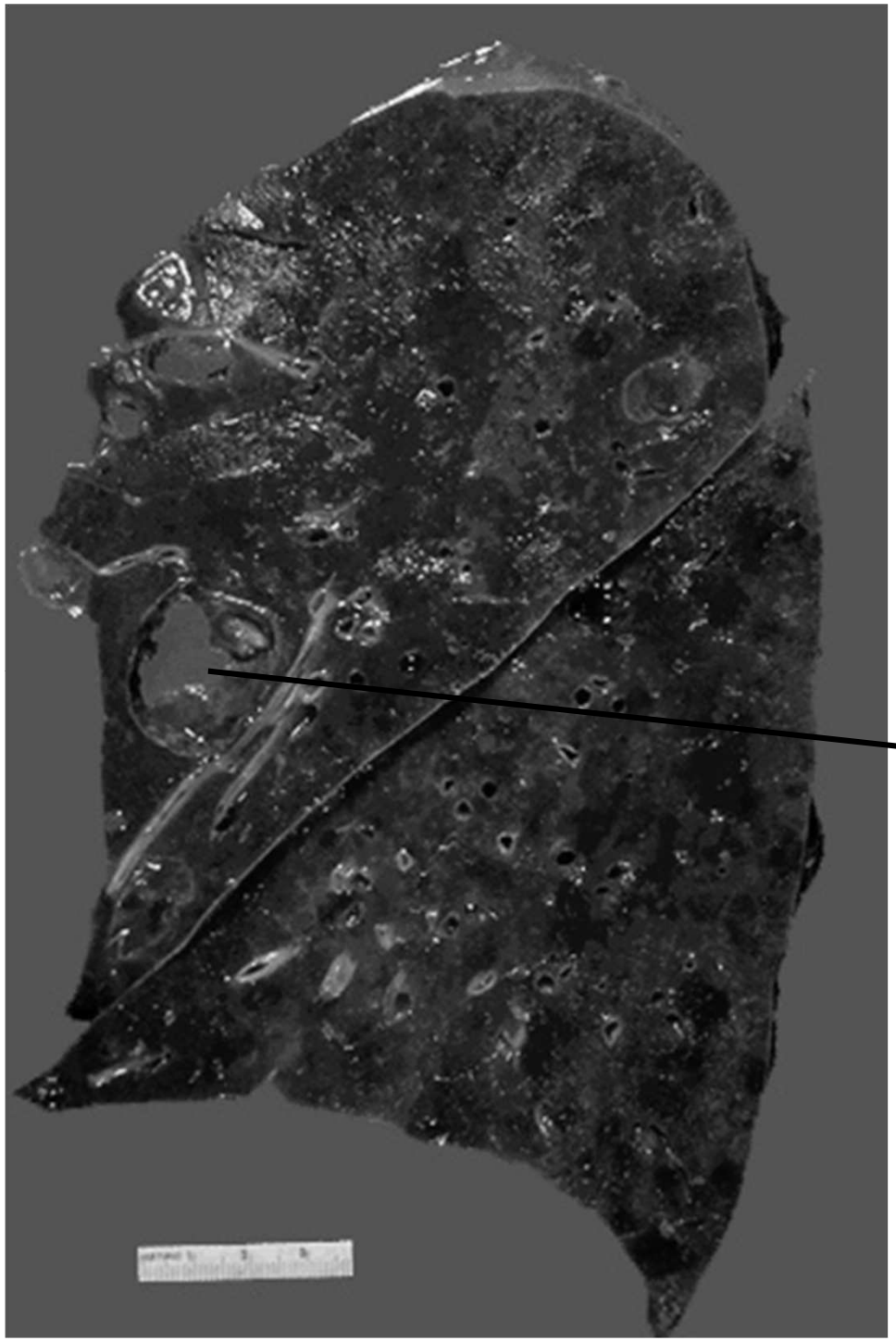


Chest CT scan

(Lung window)

Air-fluid level within an abscess
pneumonic consolidation on both sides

(Bone window)



Lung abscesses

The purulent exudate has drained

Abscesses can be source of septicemia and are difficult to treat

Pulmonary infections

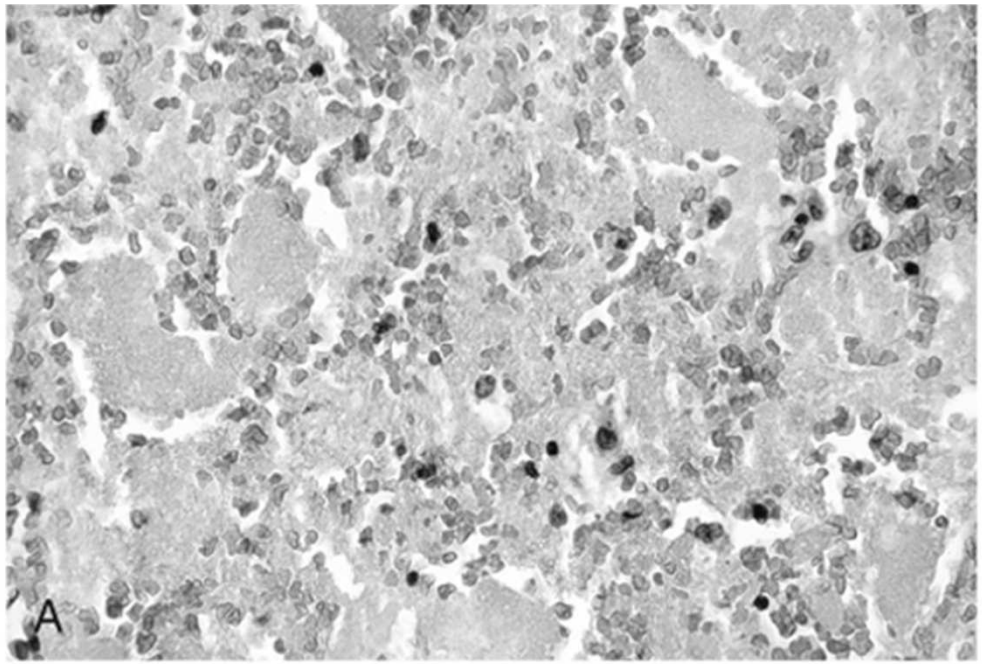
Chronic Pneumonia

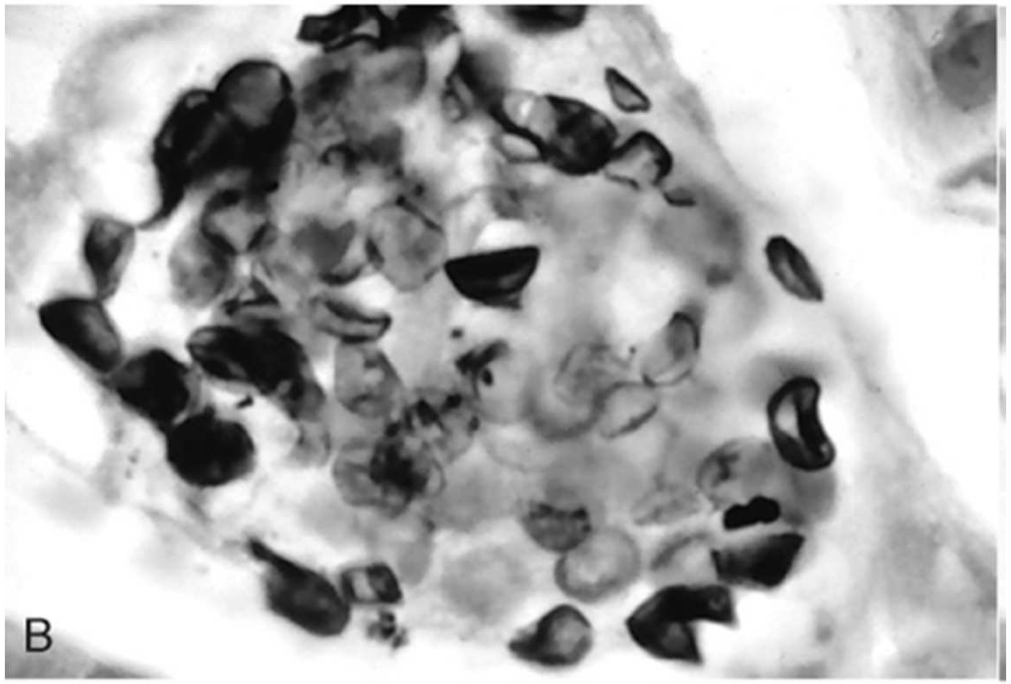
TBC, fungal pneumonias….

Necrotizing Pneumonia and Lung Abscess – anaerob and Sta

Pneumonia in the Immunocompromised Host

CMV, *Pneumocystis jiroveci*
***Mycobacterium AI,* Invasive aspergillosis**
Invasive candidiasis



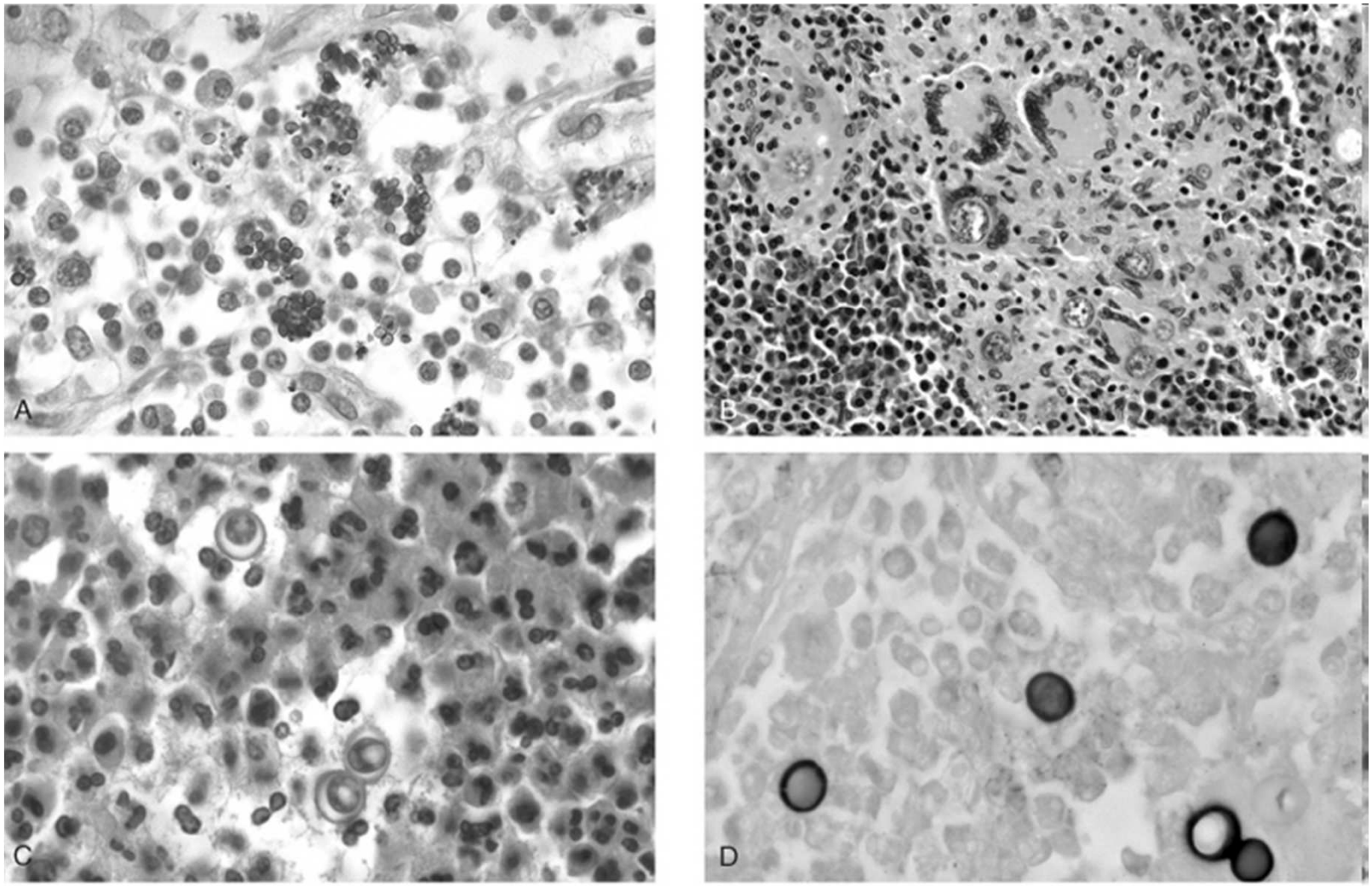


Pneumocystis jiroveci

Pneumocystis pneumonia.

A, The alveoli are filled with a characteristic foamy "cotton candy" exudate.

B, Silver stain demonstrates cup-shaped cyst walls within the exudate.



A, *Histoplasma capsulatum* yeast forms fill phagocytes in a lymph node of a person with disseminated histoplasmosis. **B**, Coccidioidomycosis with intact spherules within multinucleated giant cells. **C**, Blastomycosis, with rounded budding yeasts, larger than neutrophils. Note the characteristic thick wall and nuclei (not seen in other fungi). **D**, Silver stain highlighting broad-based budding.

OBSTRUCTIVE / RESTRICTIVE

LUNG DISEASES

Increase in resistance to airflow due to obstruction at any level

Reduced expansion of lung, with decreased total lung capacity

$\frac{\text{FEV1}}{\text{FVC}}$ lowered

$\frac{\text{FEV1}}{\text{FVC}}$ near normal

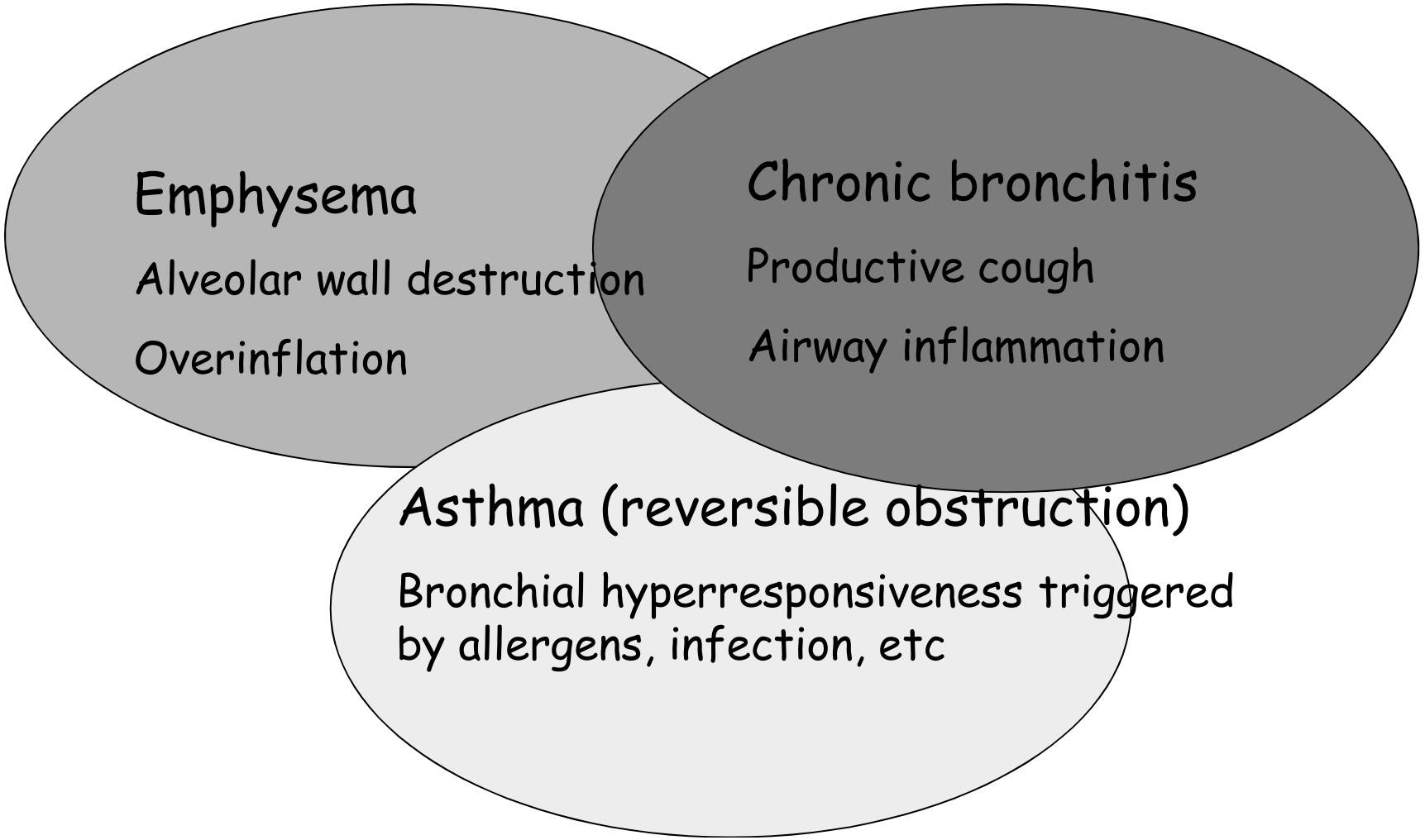
Emphysema
Chronic bronchitis
Bronchiectasis
Bronchial asthma

Chest wall disorders

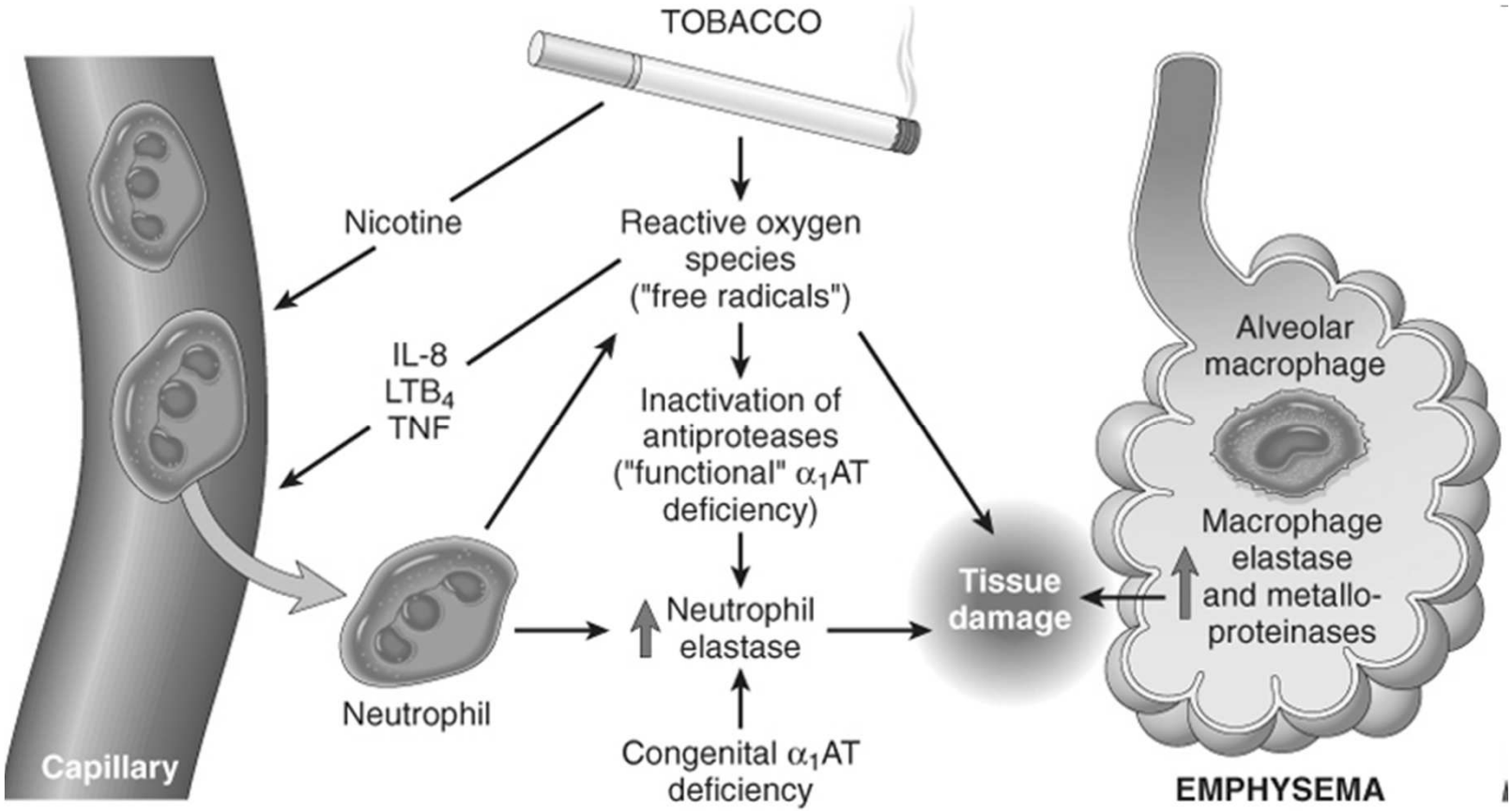
Chronic interstitial diseases

OBSTRUCTIVE LUNG DISEASES

Chronic injury (smoking)



Emphysema - pathogenesis

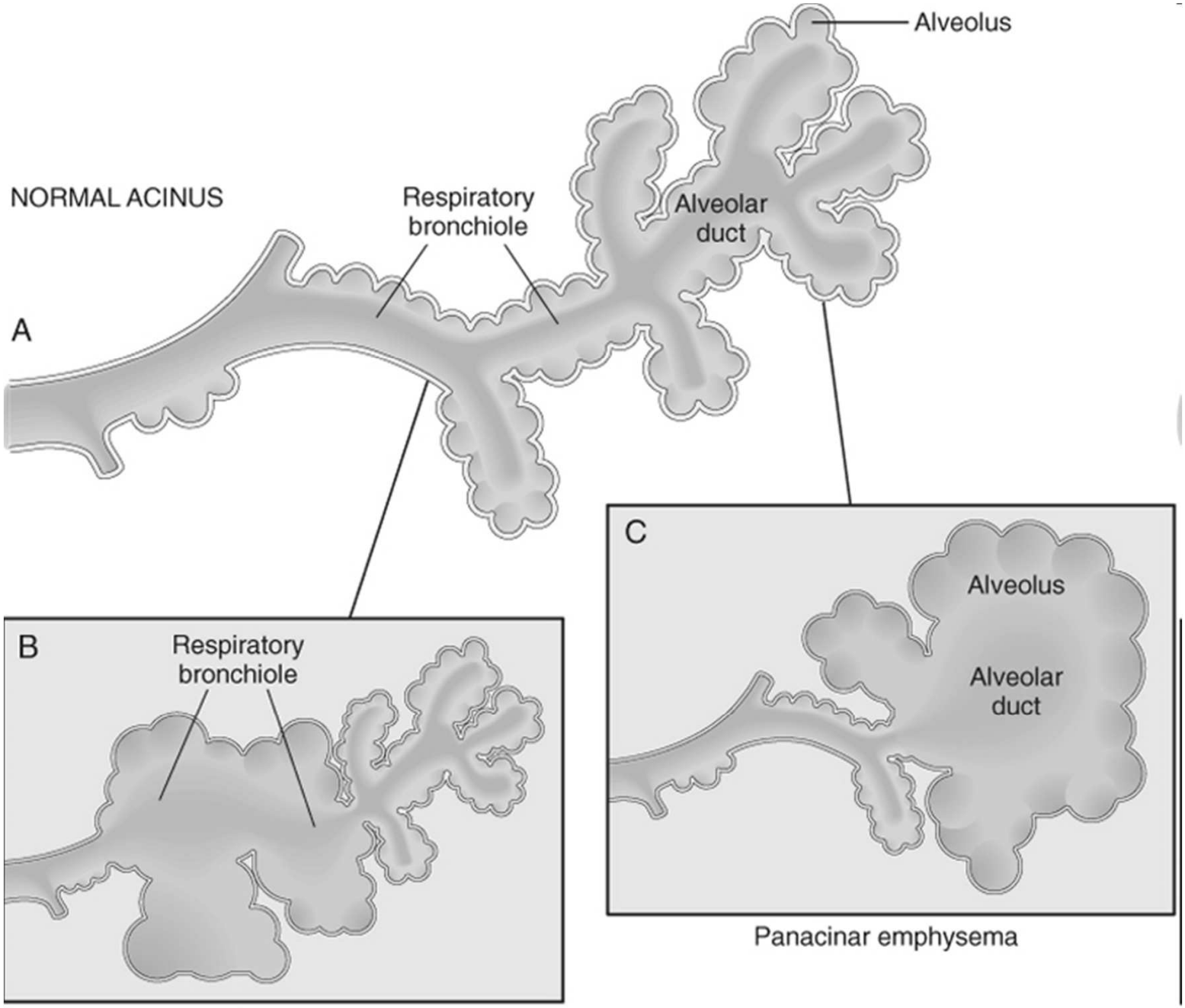


The protease-antiprotease imbalance and oxidant-antioxidant imbalance are additive in their effects and contribute to tissue damage. a1-Antitrypsin (a1AT) deficiency can be either congenital or "functional" as a result of oxidative inactivation.

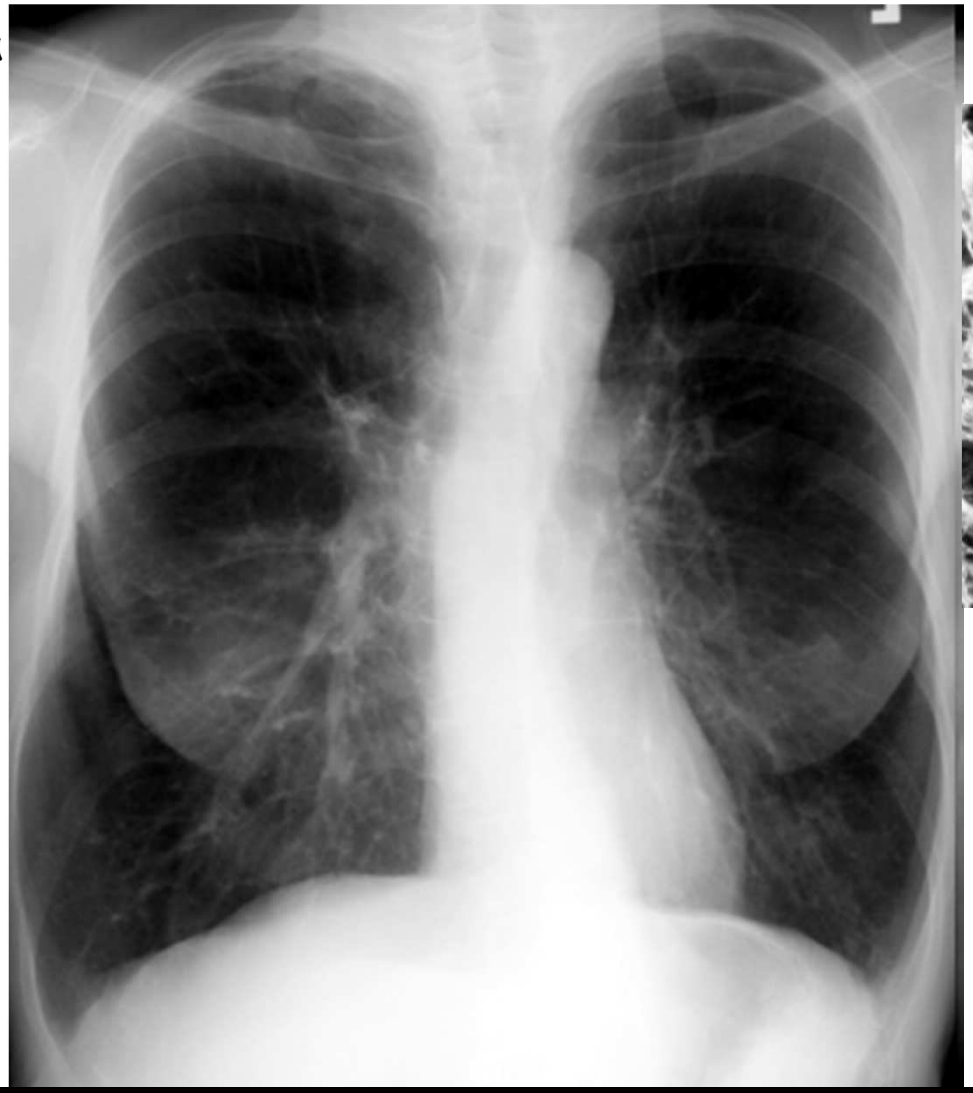
Emphysema

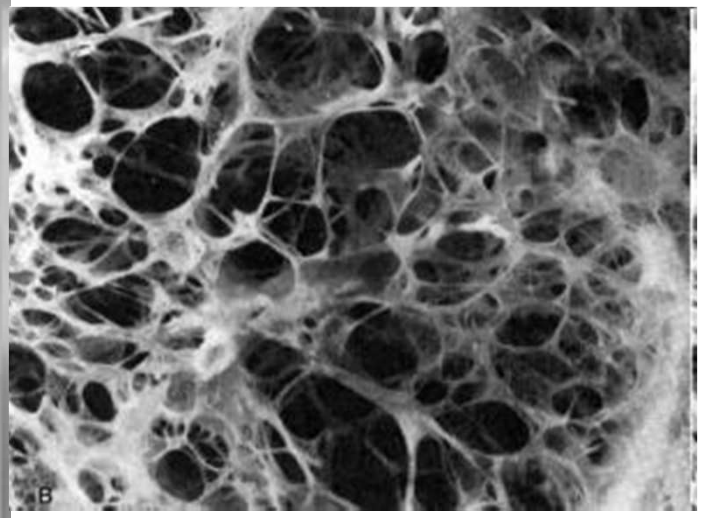
Forms

Centroacinar	upper lobes, smokers, often with bronchitis
Panacinar	lower lobes, a1 AT def.
Paraseptal	close to pleura, scars, often associated with spontaneous PTX
Irregular	along scars, asymptomatic

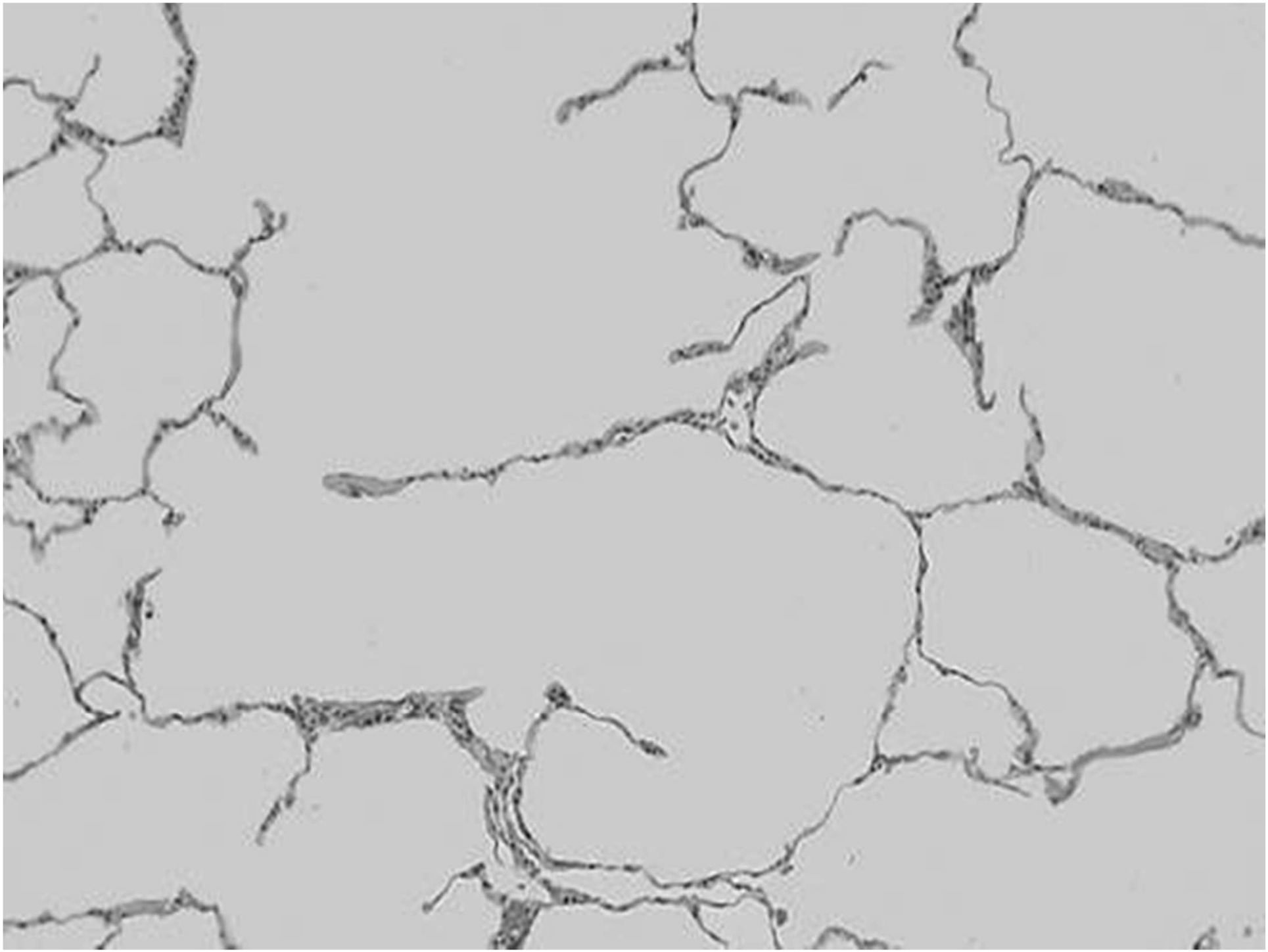


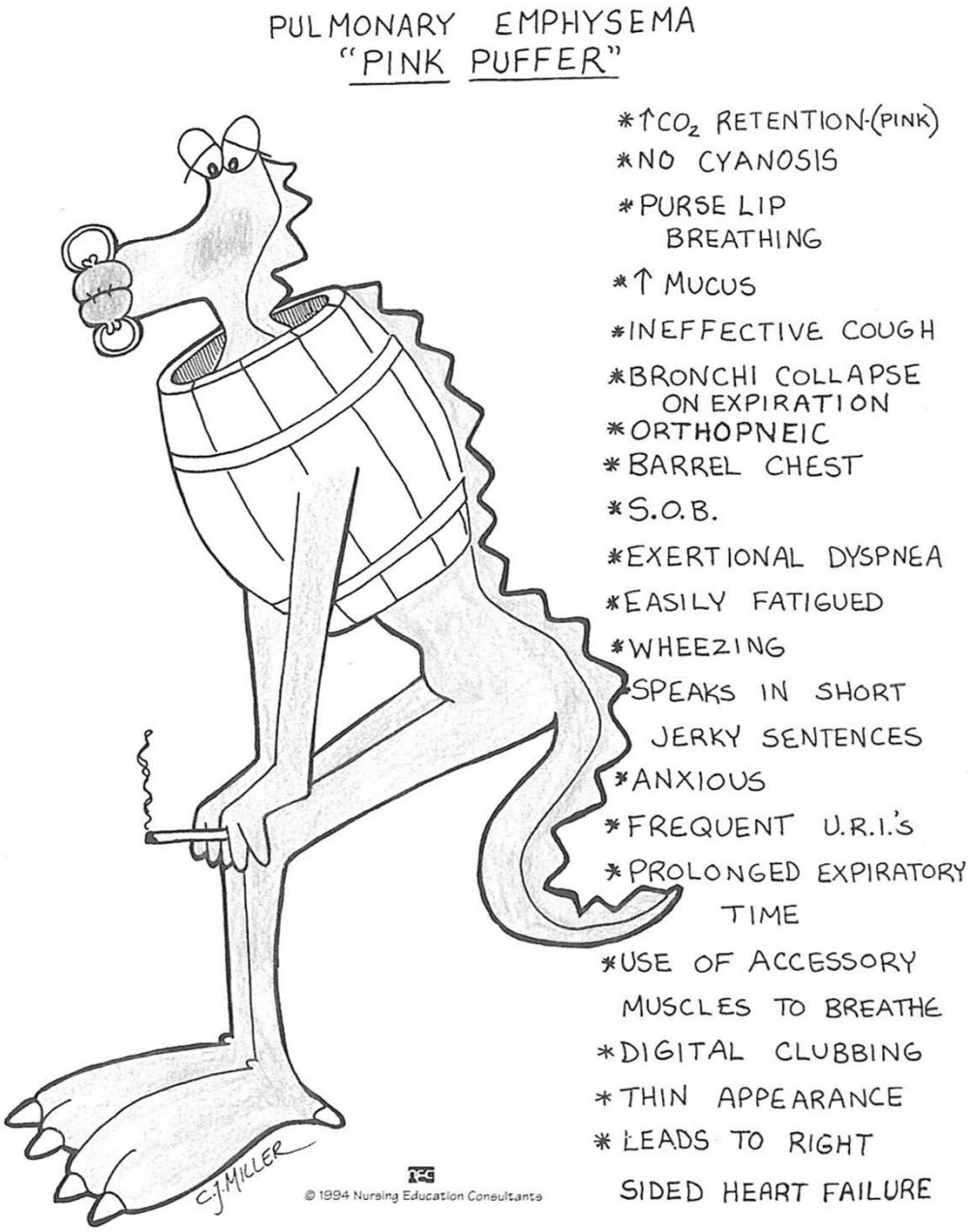
emphysema



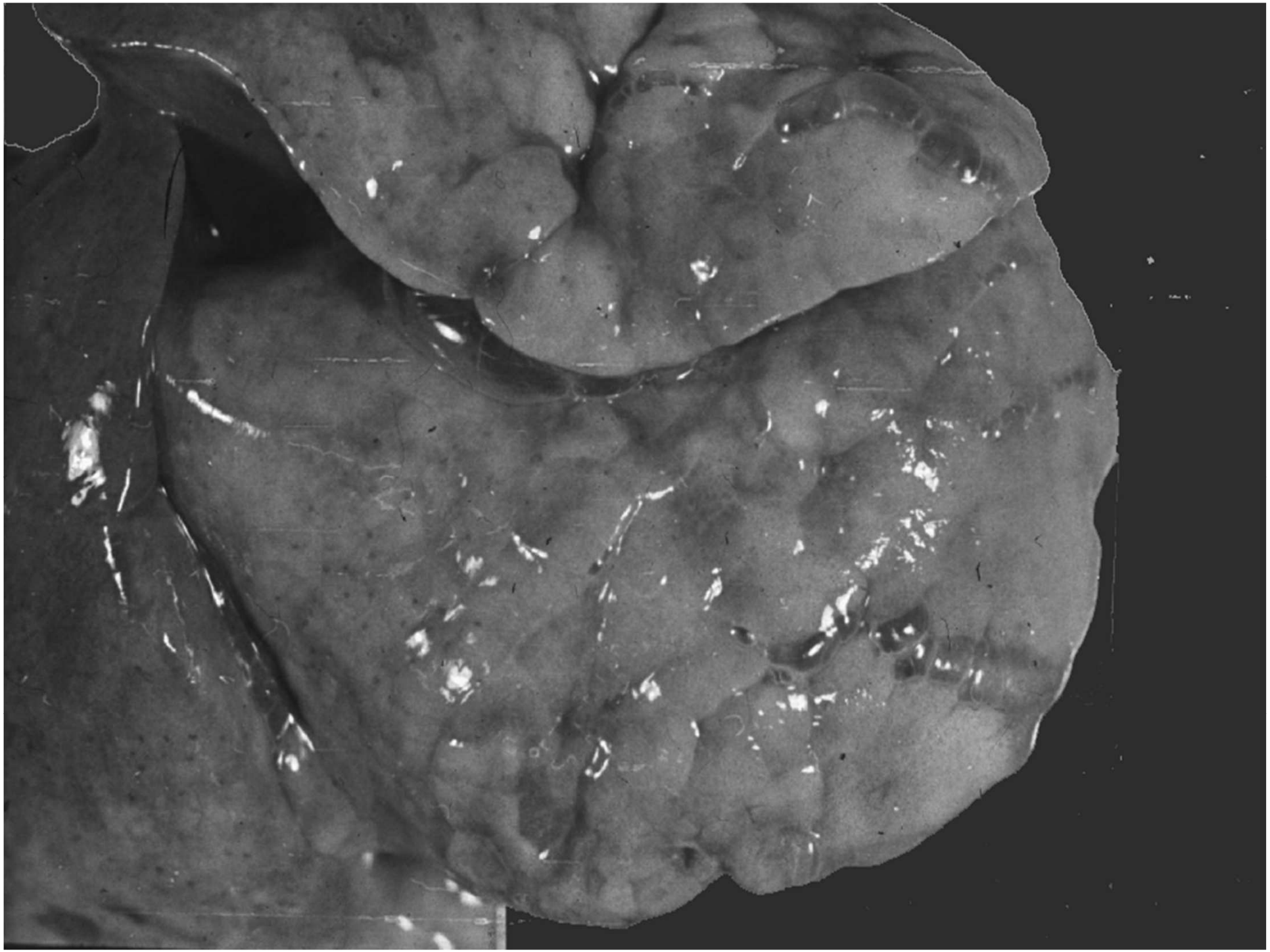


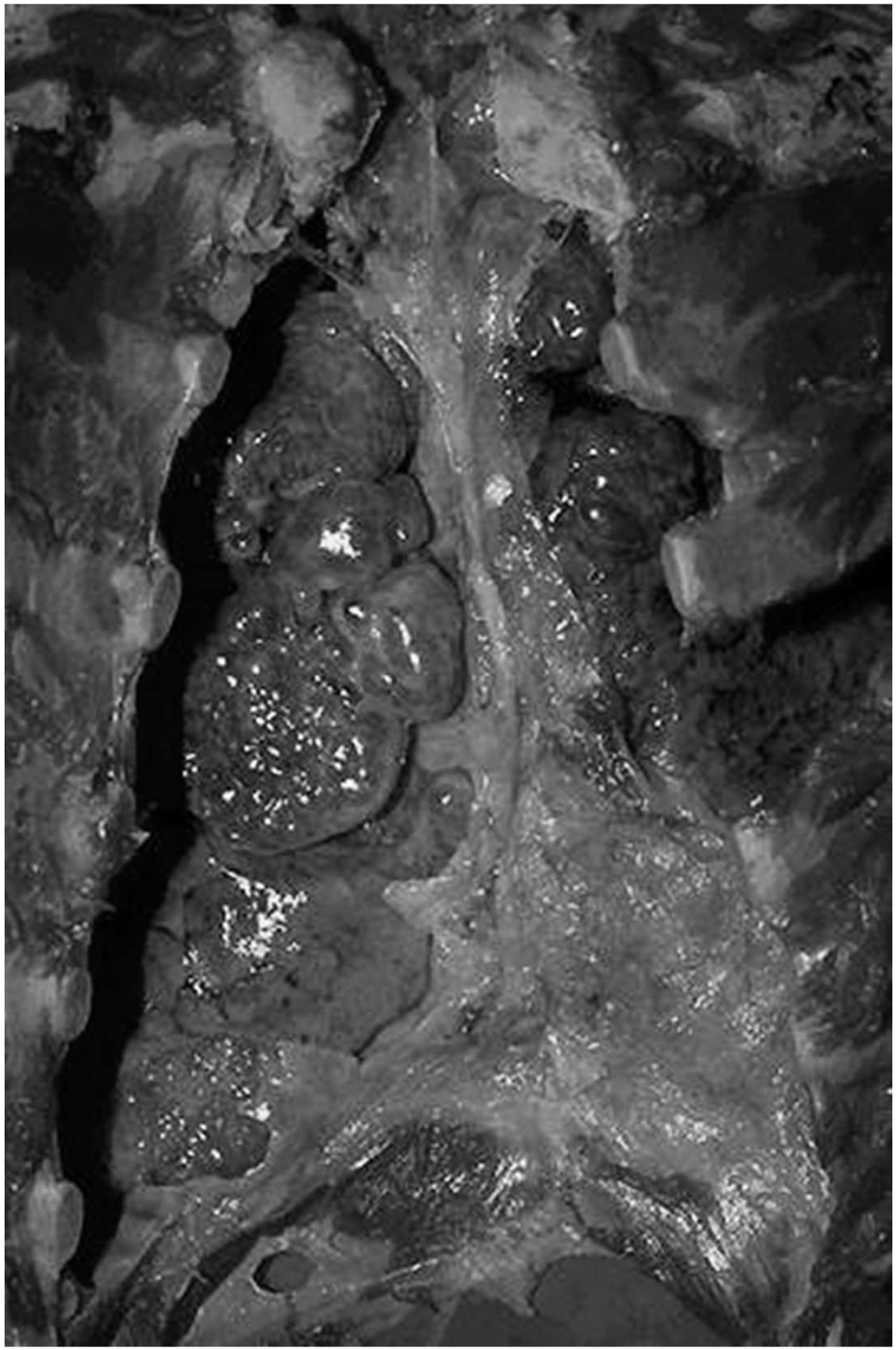
The lung volumes appear to be increased; in normal sized lungs, the **number of ribs** in the lung field is 9. With increased lung volumes, the number of ribs seen the lung fields is **higher**. Other clues include are **flattened diaphragms**, **wide separation of the ribs**, and an **elongated narrow heart shadow**. In addition, pulmonary vessels appear to be diffusely decreased in many patients with pulmonary emphysema and hyperinflated lung fields. Other terms used to describe increased lung volume are **hyperinflated and hyperaerated**.

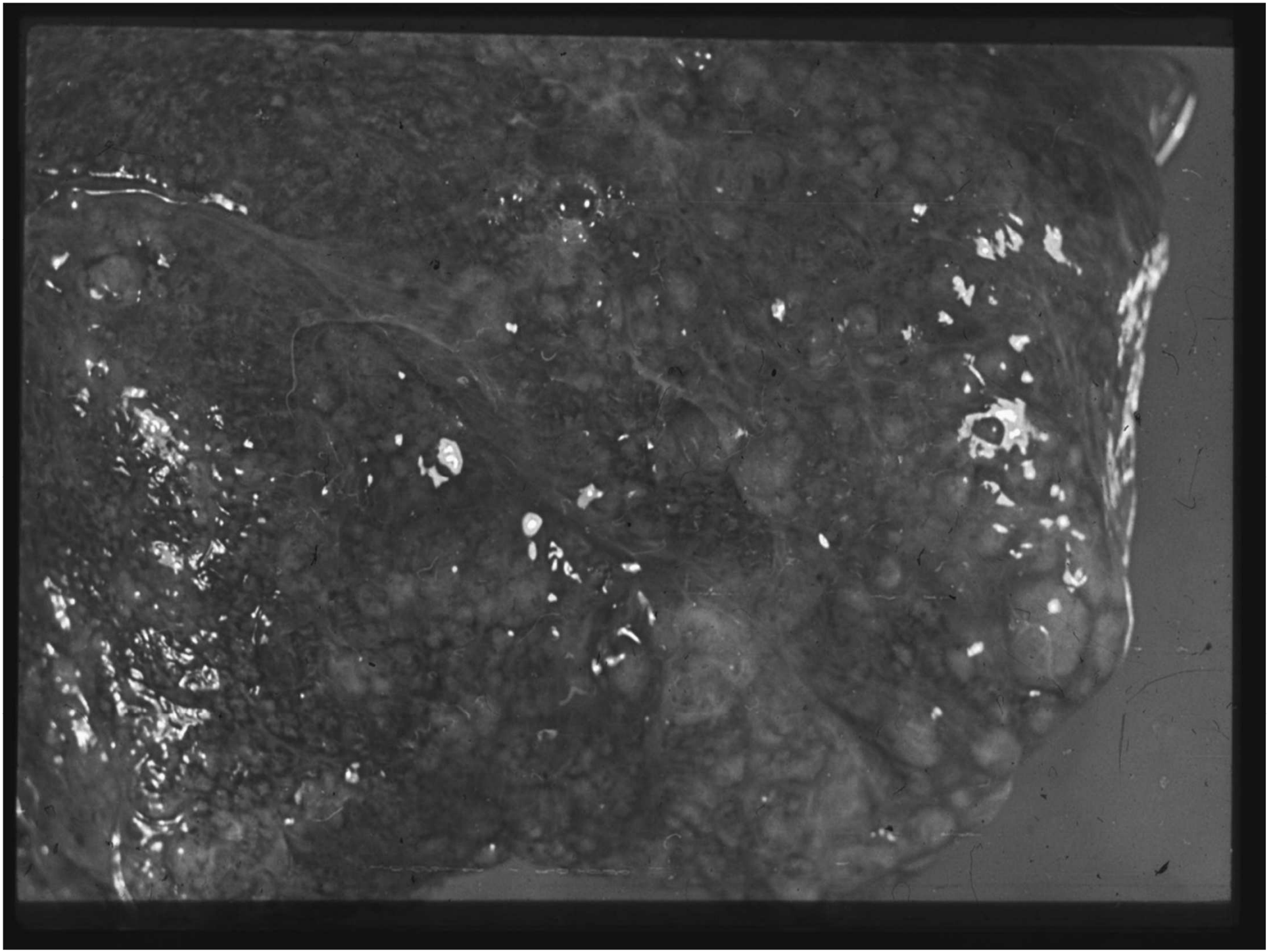


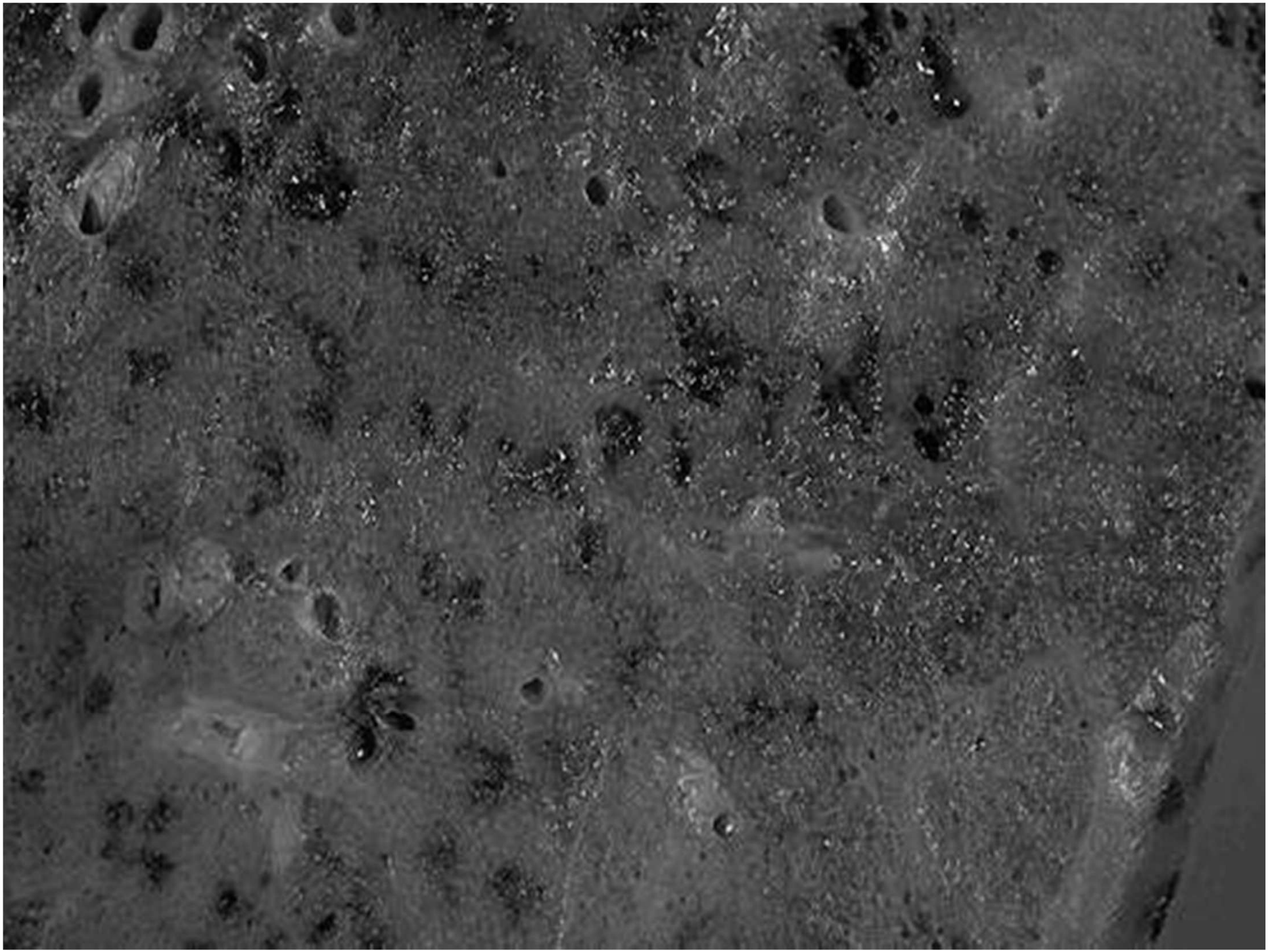


Loss of elastic tissue in the walls of the alveoli leads to loss of elastic recoil, during expiration, leading to functional airflow obstruction









Emphysema

Clinical course

- No symptoms, until 1/3 of parenchyma is lost
- Dyspnoe
- Weight loss
- Wheezing
- Limited cough
- Expiratory airflow limitation
- Blood gas values are relatively normal

†

Respiratory acidosis

Cardiac failure (right sided)

PTX – lung collapse

Emphysema

OTHER FORMS

Compensatory hyperinflation ~

dilation of alveoli, but no tissue destruction

Obstructive overinflation ~

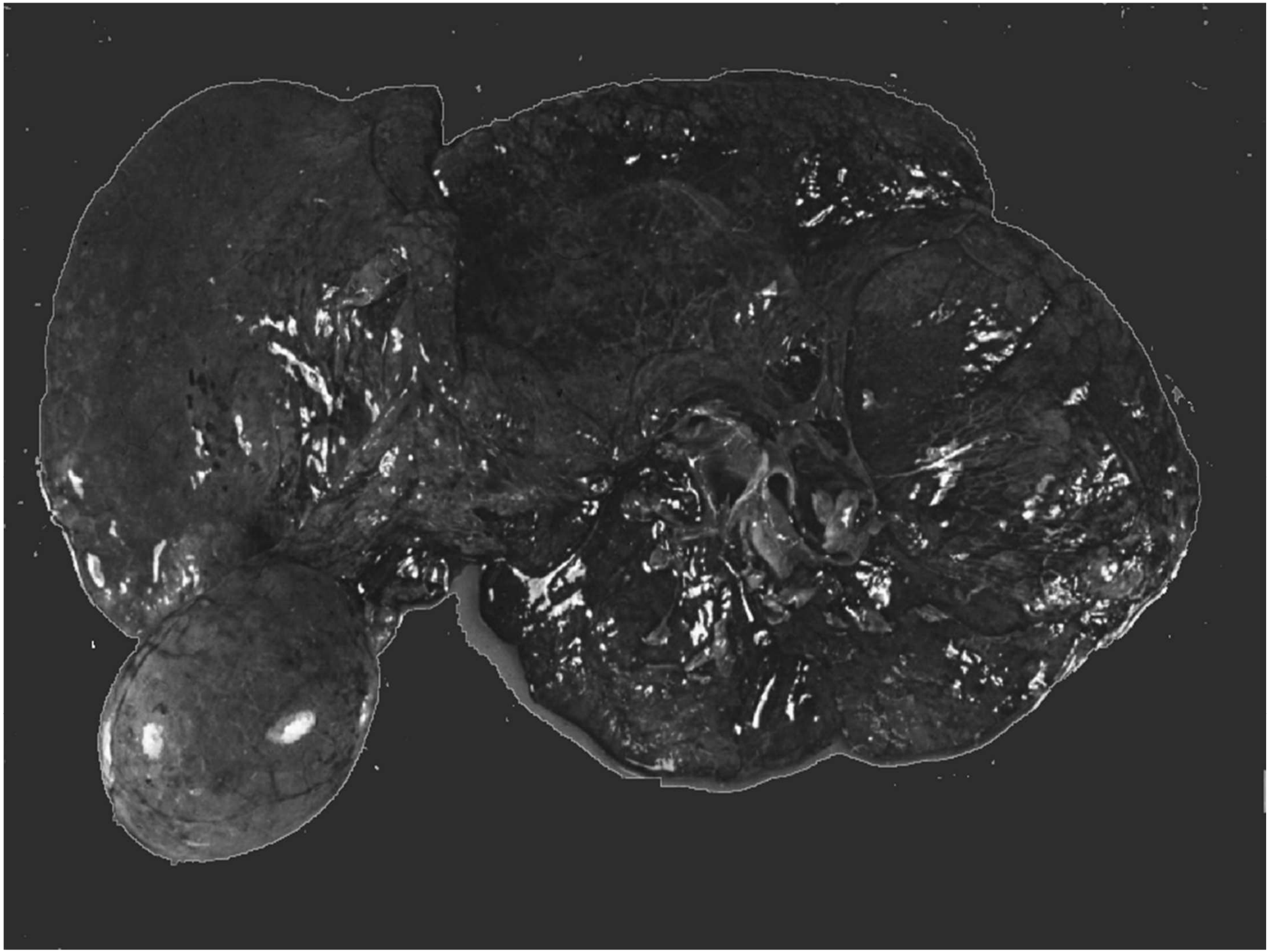
ball valve effect of an obstructing agent, may compress adjacent normal lung parenchyma

Bullous ~

large bullous parapleural blebs associated with TBC scarring

Interstitial ~ alveolar tears,

thoracic trauma, artefitial ventilation, whooping cough



Chronic bronchitis

Definition

Persistent productive cough during 3 month in two consecutive years without any other underlying disease.

Progresses to

COPD

Chronic cor pulmonale with cardiac failure

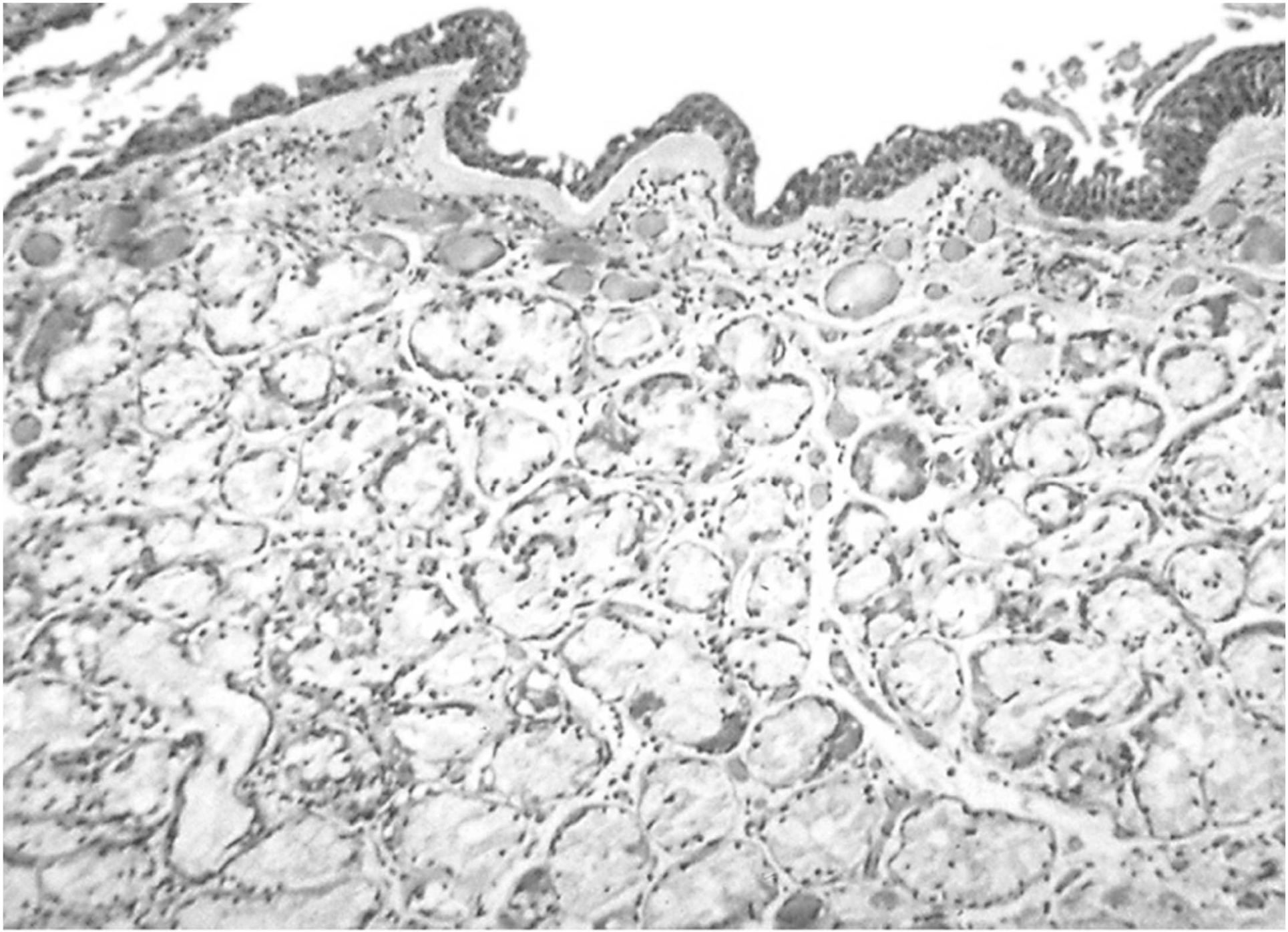
Metaplasia, dysplasia of respiratory epithelium, leading to cancer transformation

Cause: smoking, inhalation of dust

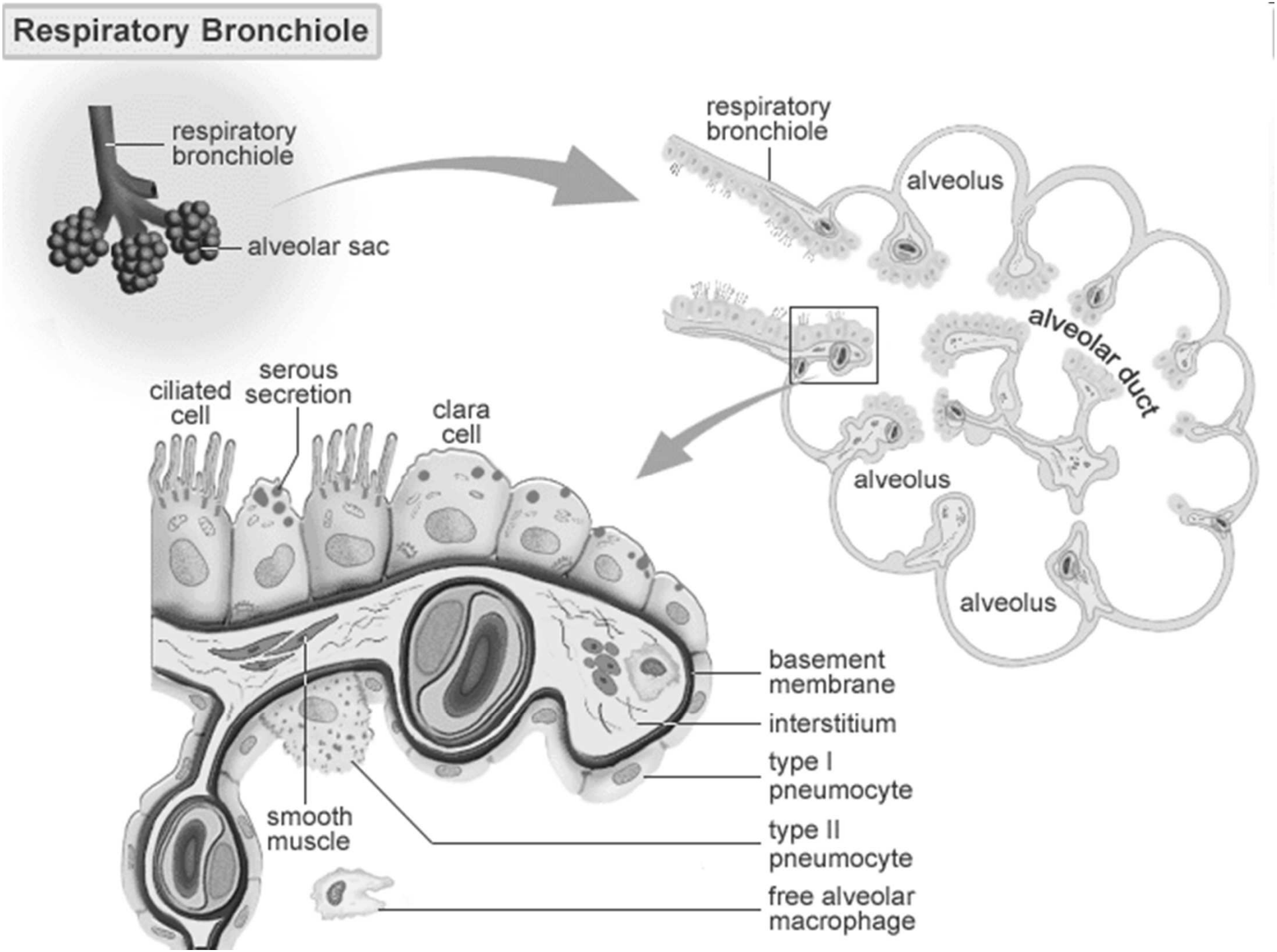
Chronic bronchitis

Hypersecretion of mucus associated with hyperplasia of bronchial mucous glands (due to granulocyte protease)

Goblet cell metaplasia and hyperplasia (protective against smoke)



Respiratory Bronchiole



Reid-Index

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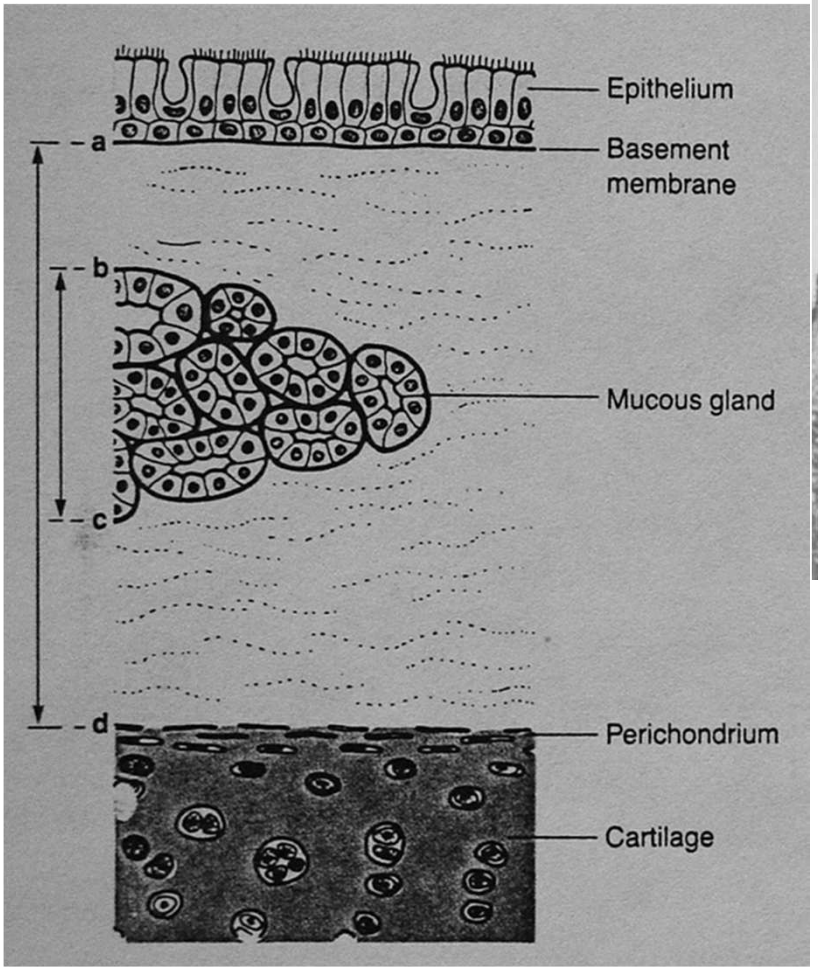
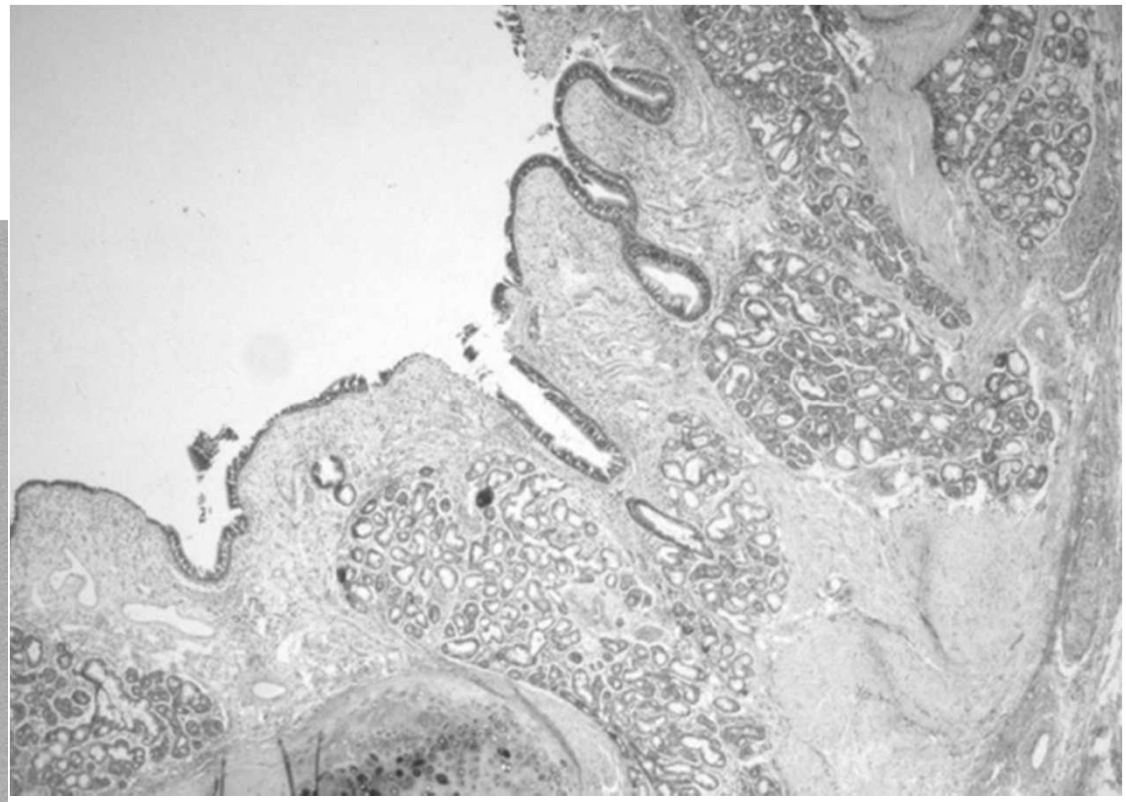
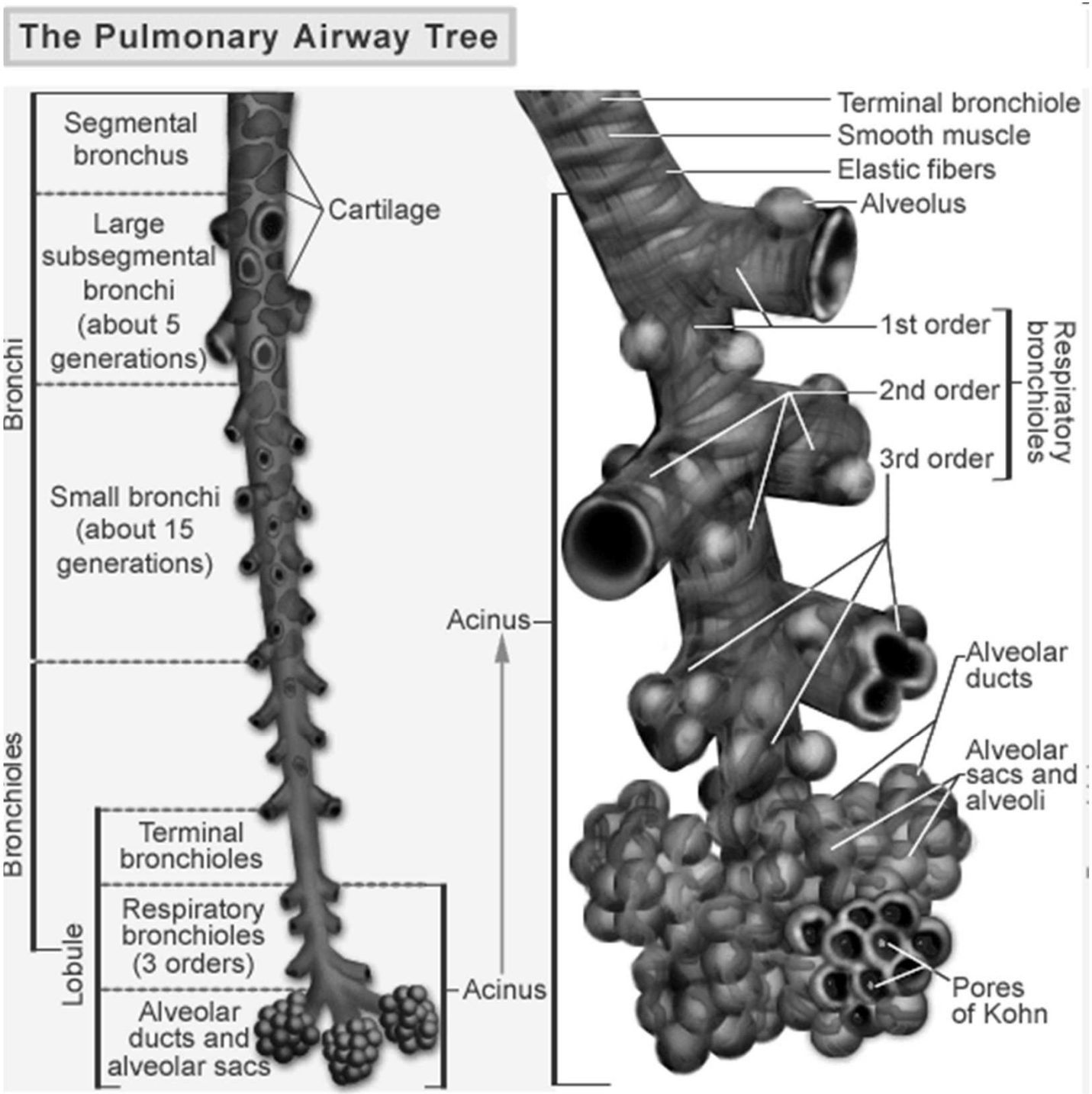




The Pulmonary Airway Tree



Chronic bronchitis

Hypercapnia

Hypoxia

Cyanosis

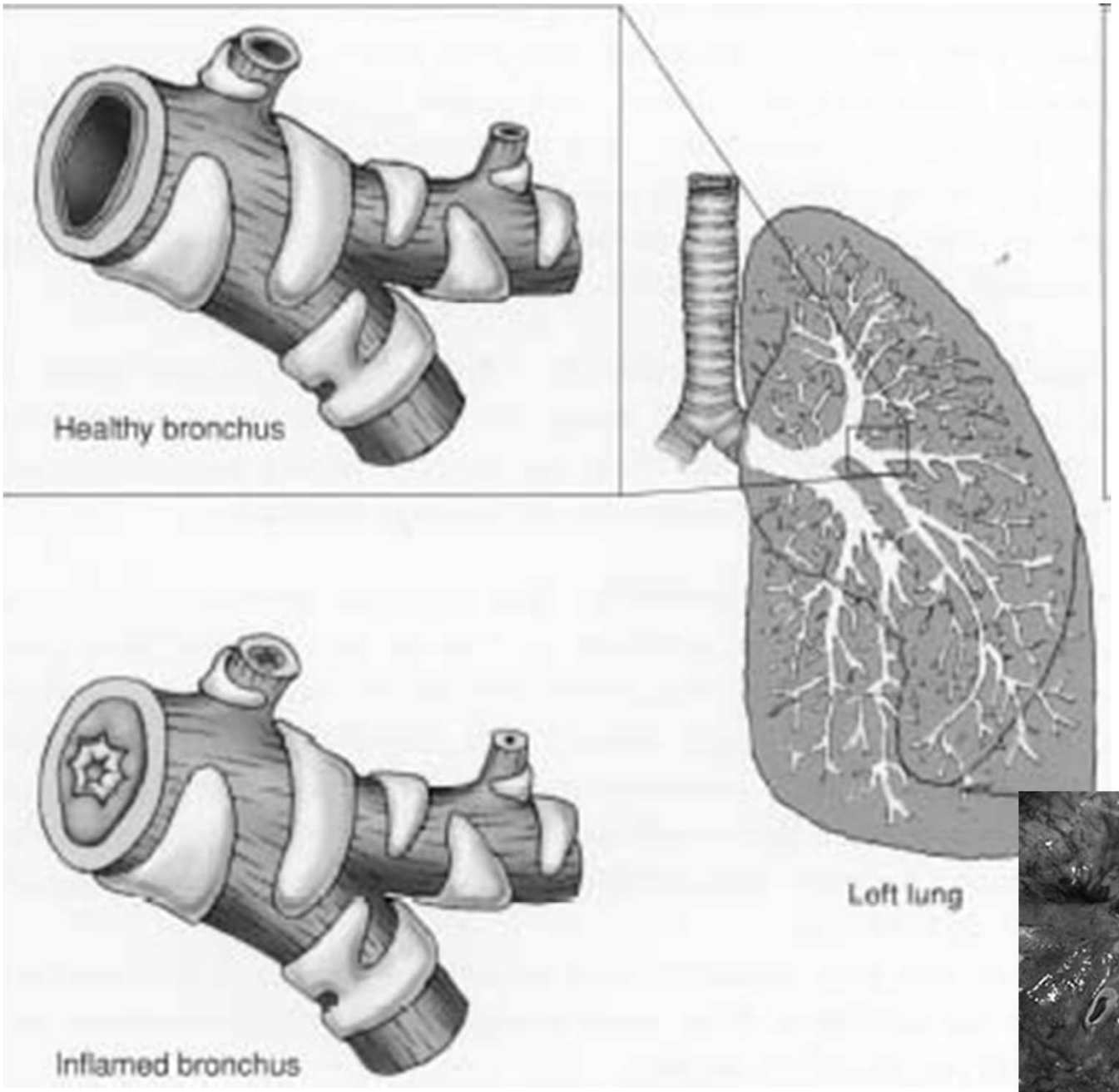
Cardiac insufficiency

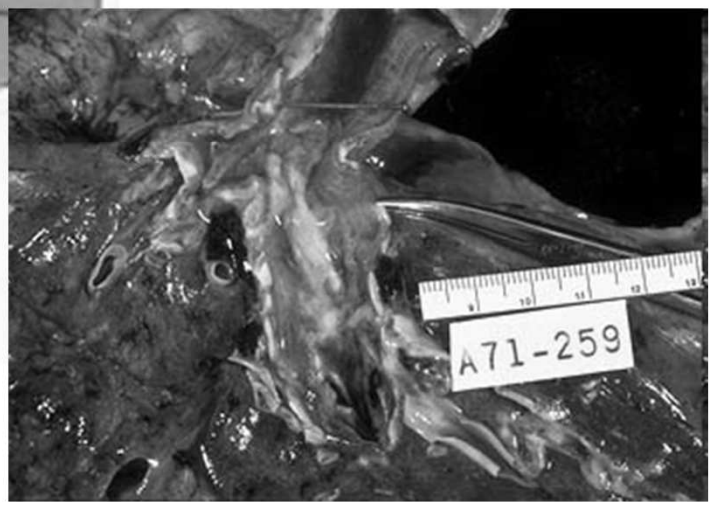
†

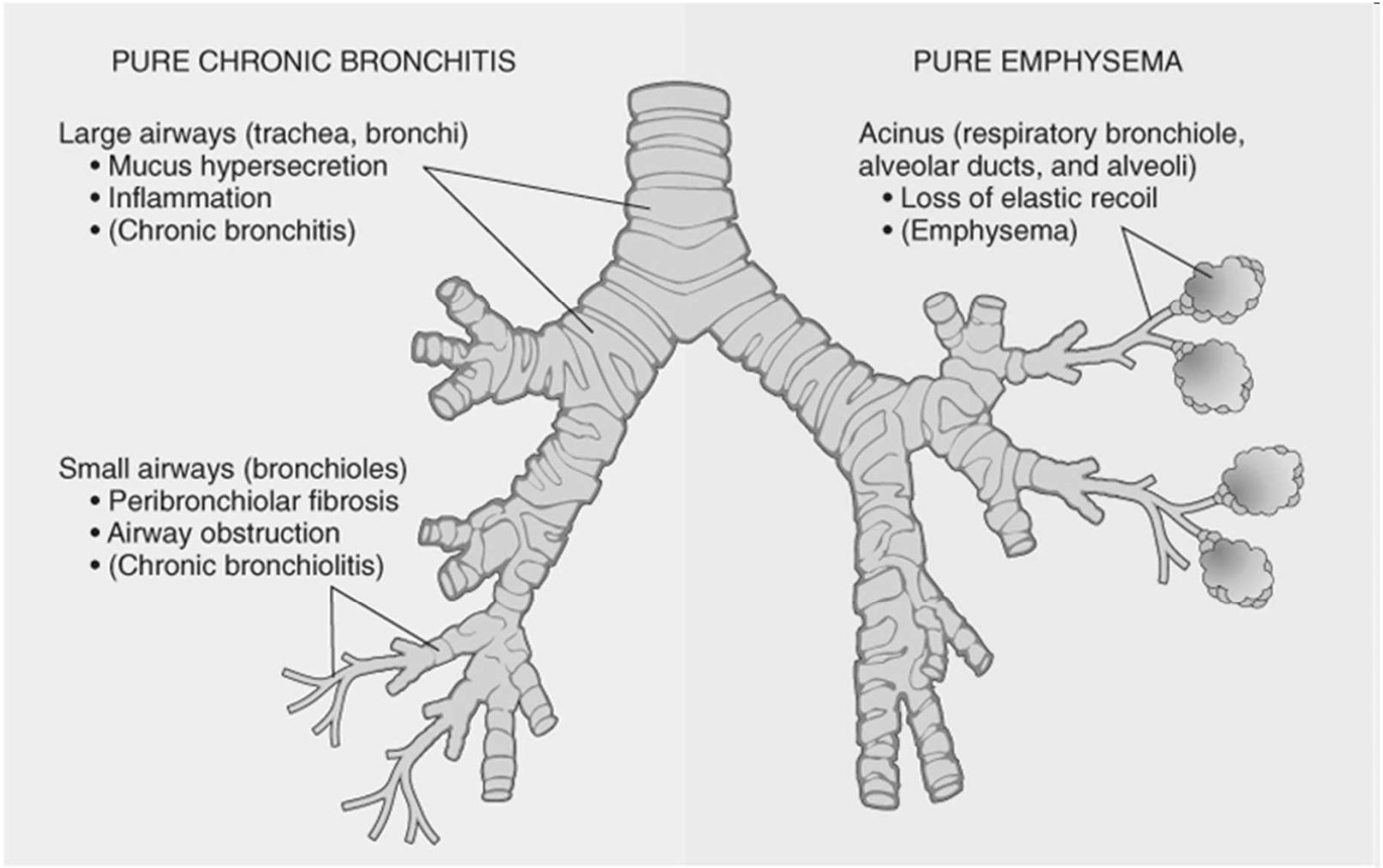
Cardiac failure

Superimposed infection

Bronchitis







Bronchial asthma

Chronic inflammatory disorder of the airways, that cause

- recurrent episodes of wheezing, breathlessness, tightness,

associated with

- bronchoconstriction,
- inflammation of the bronchial walls, and
- increased mucous secretion.

Bronchial asthma

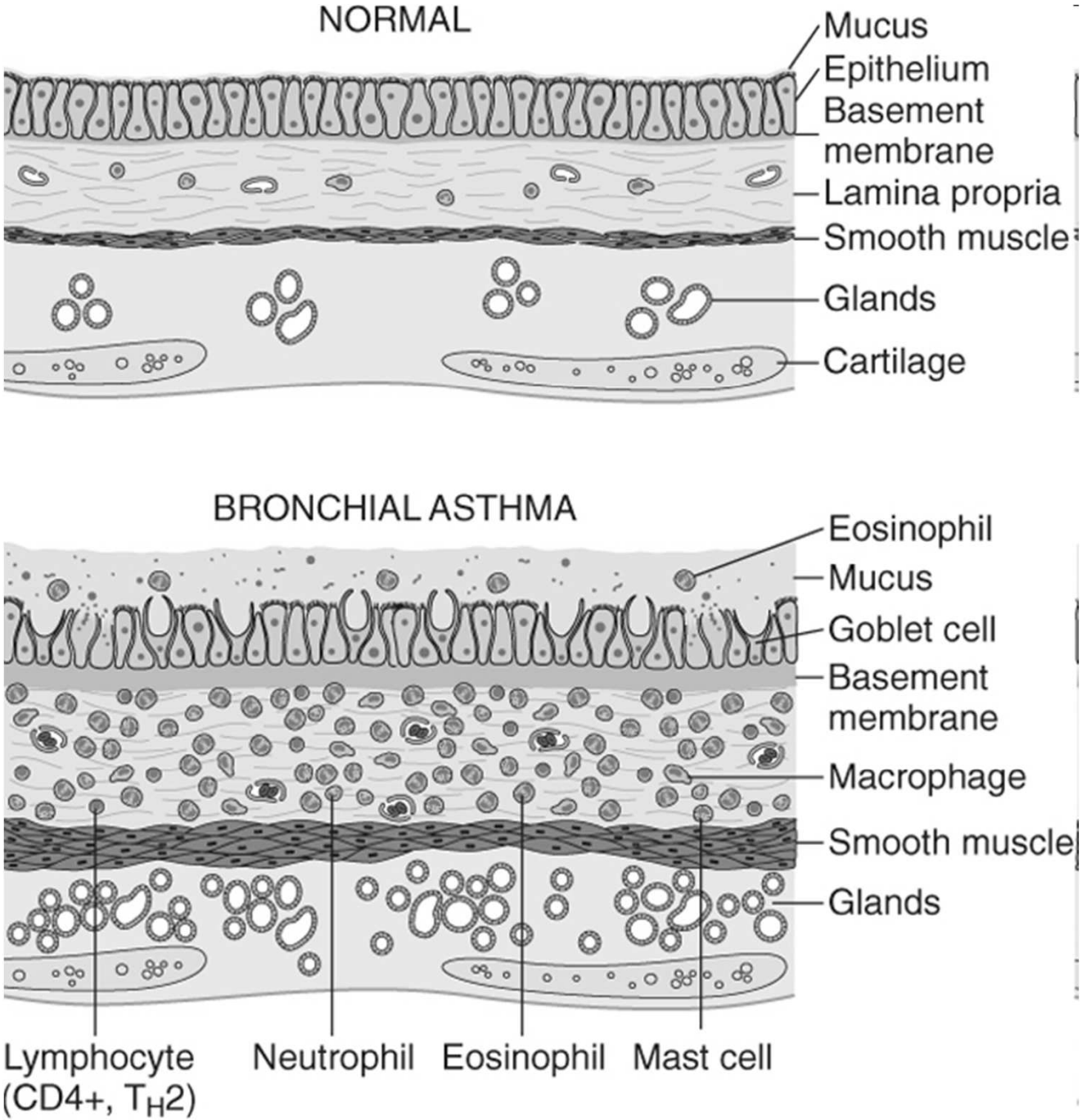
Forms:

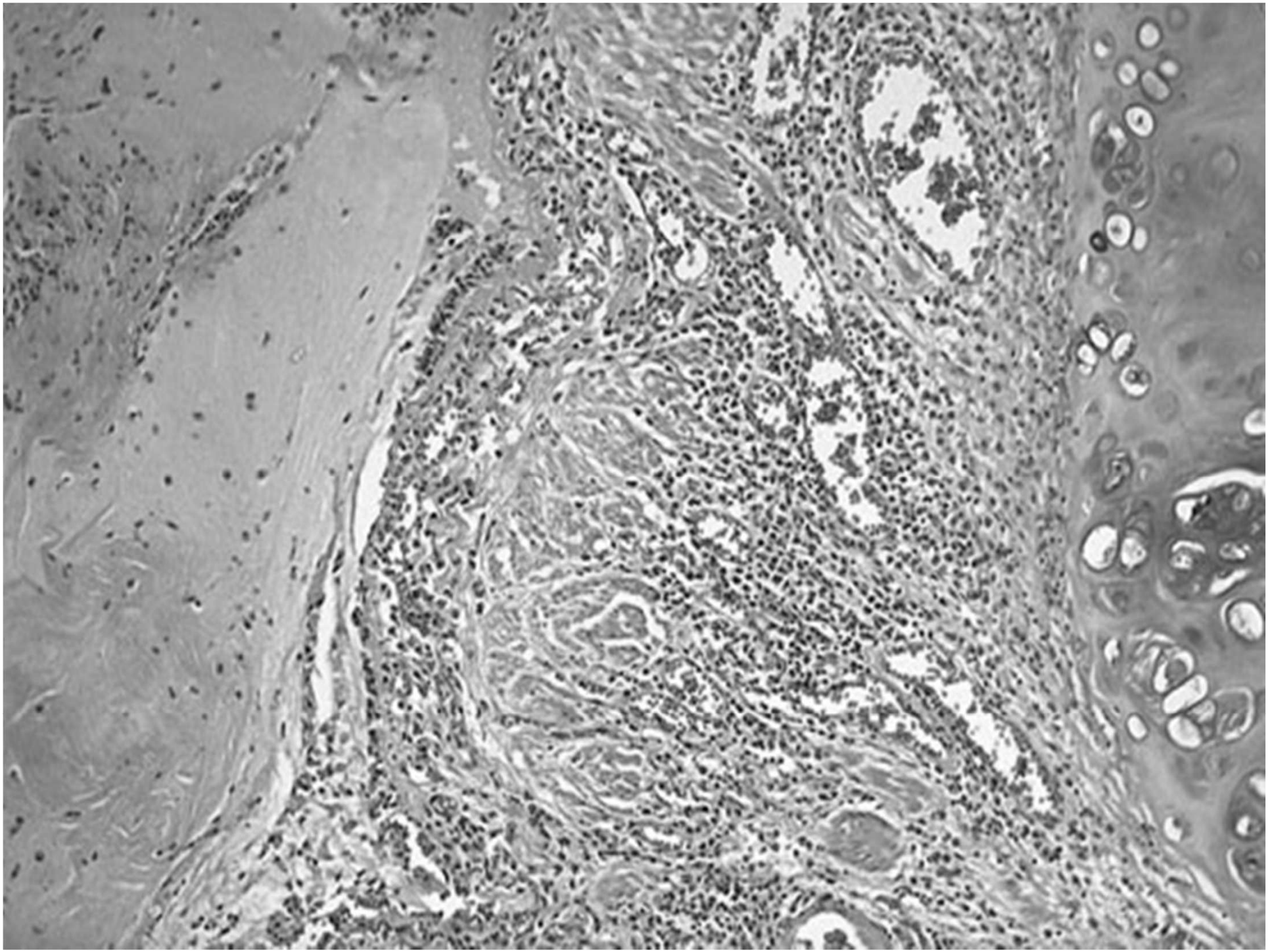
Atopic – IgE associated hypersensitivity to allergens

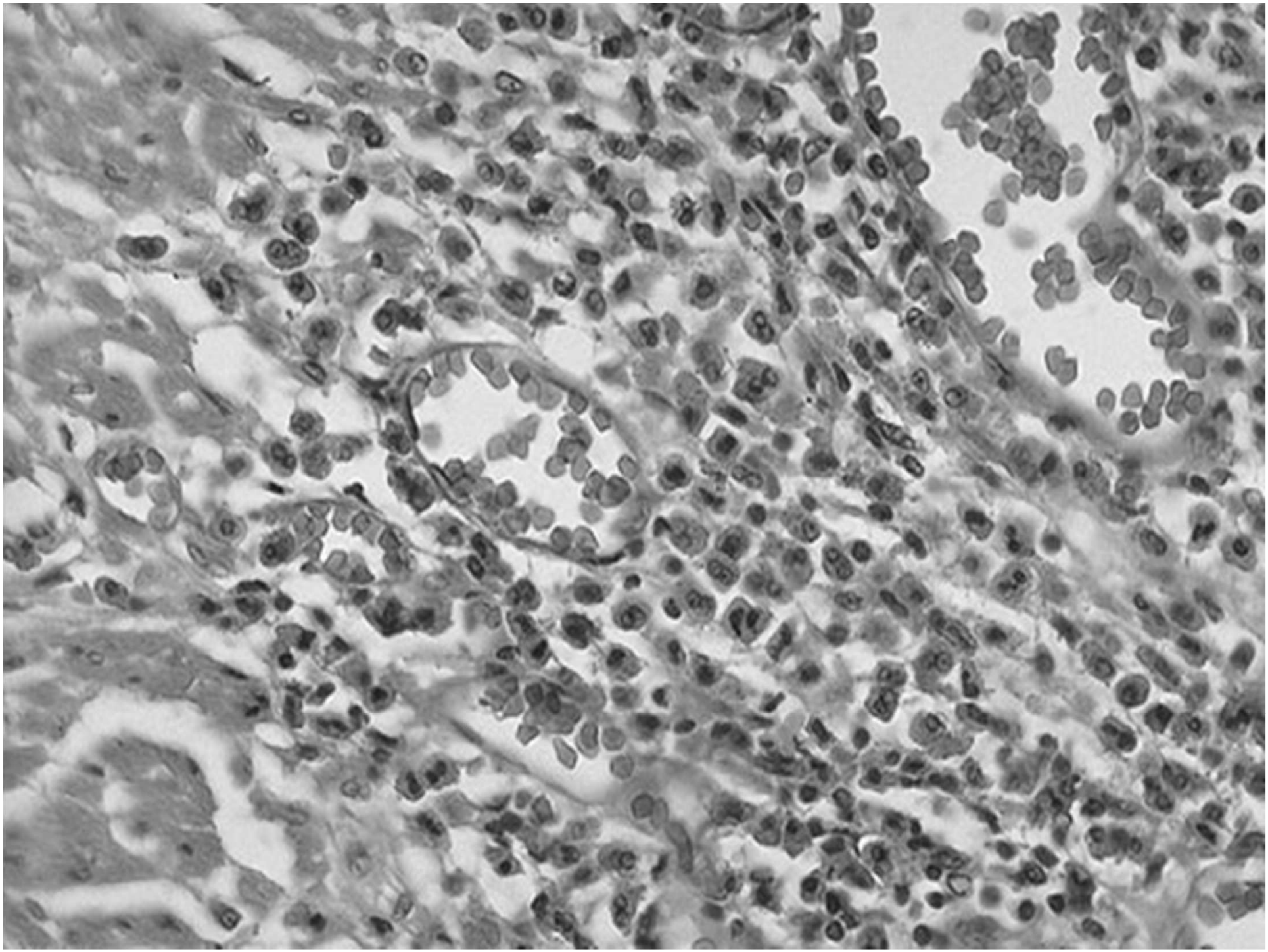
Non-atopic – Virus associated, infection lowers the threshold of subepithelial vagal receptors to irritants

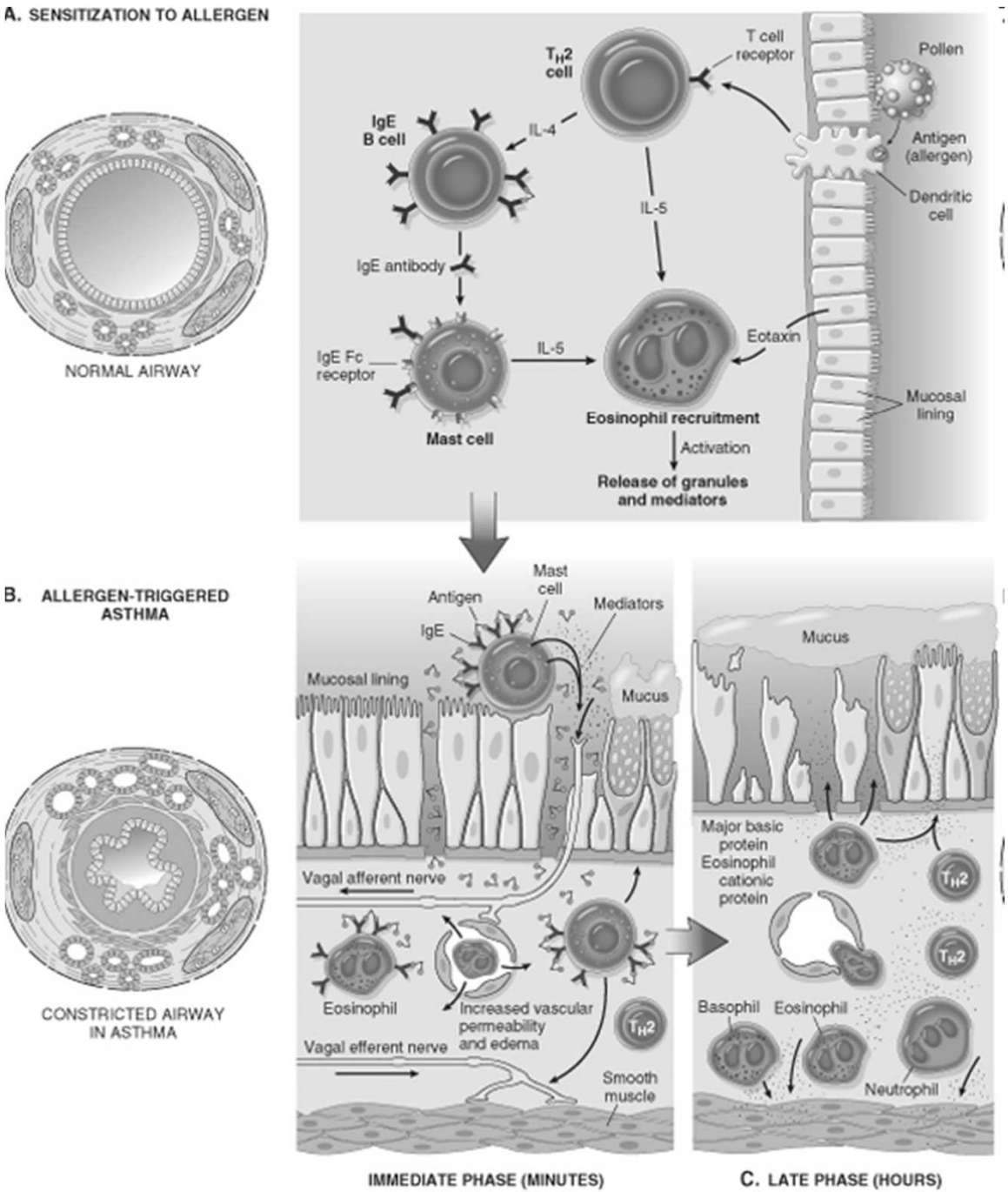
Drug-induced

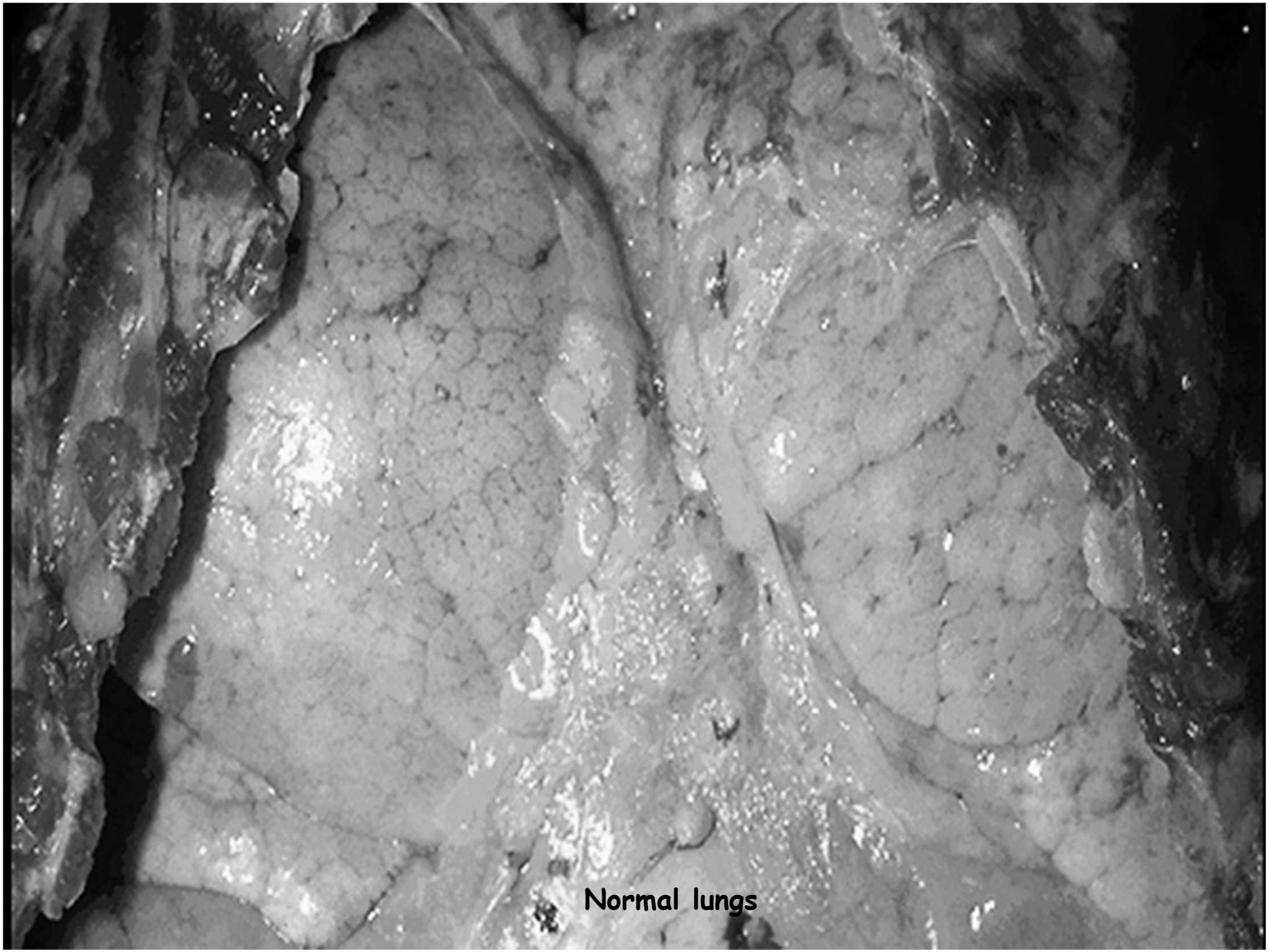
Occupational – like atopic

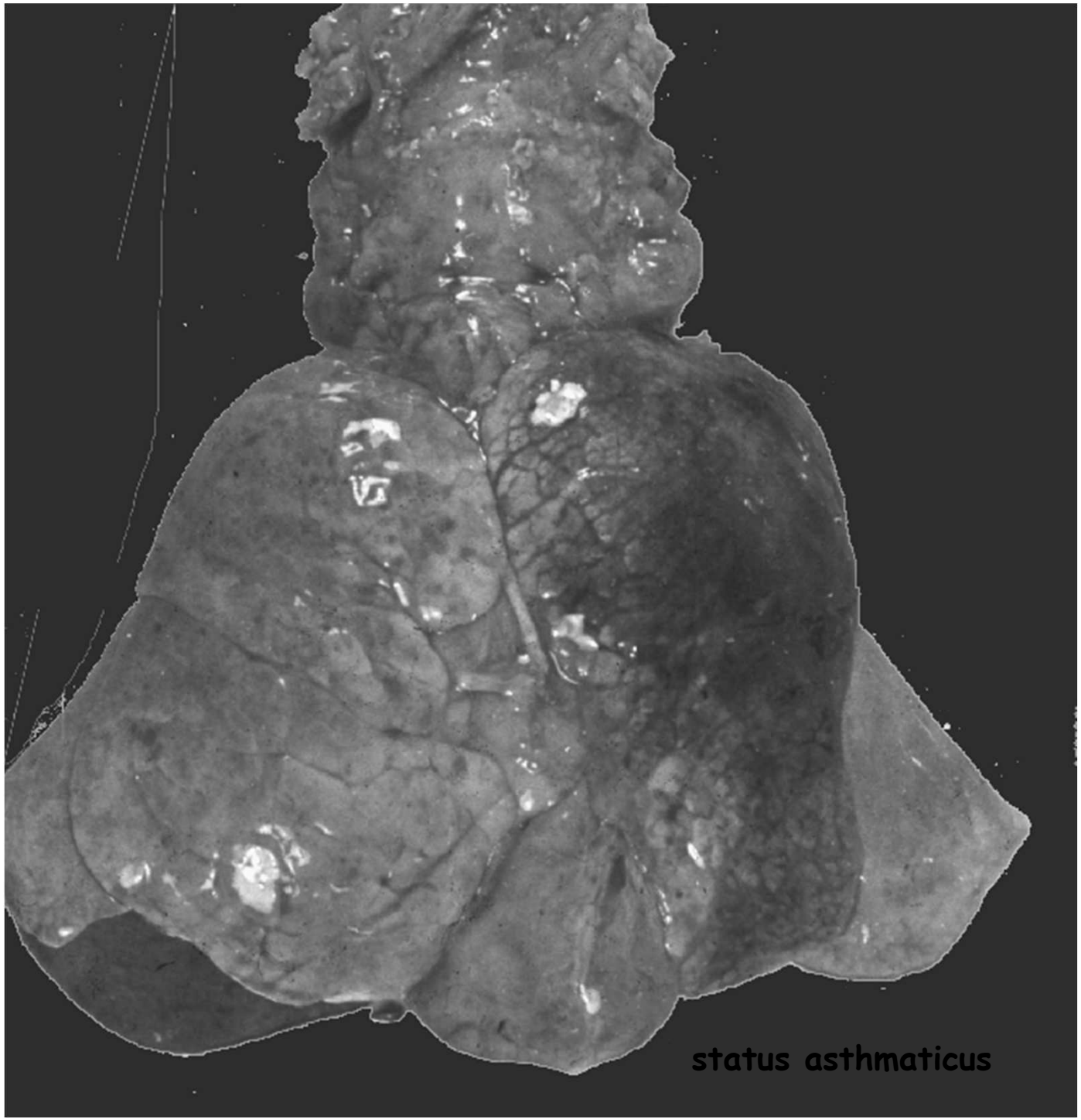


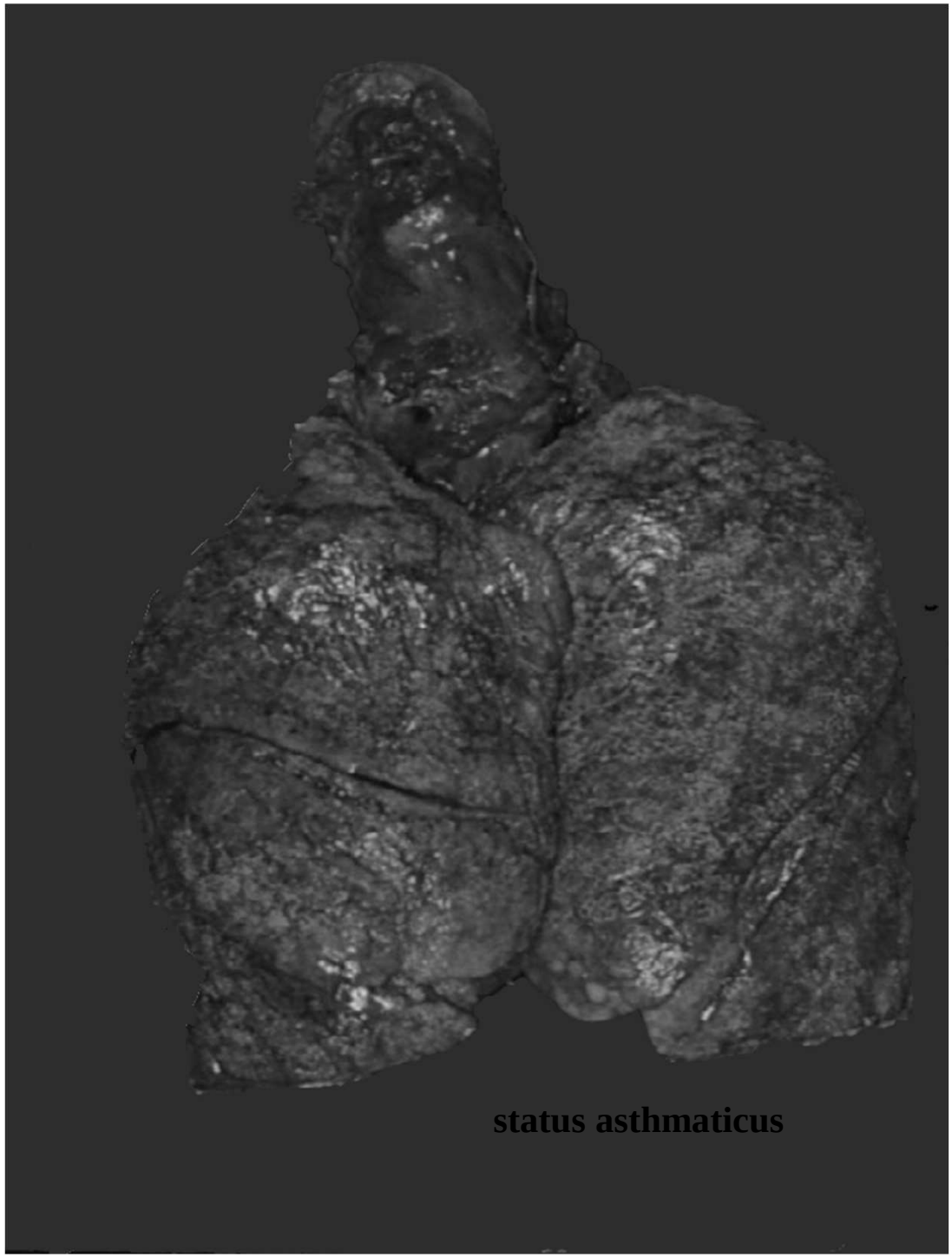







Normal lungs


status asthmaticus



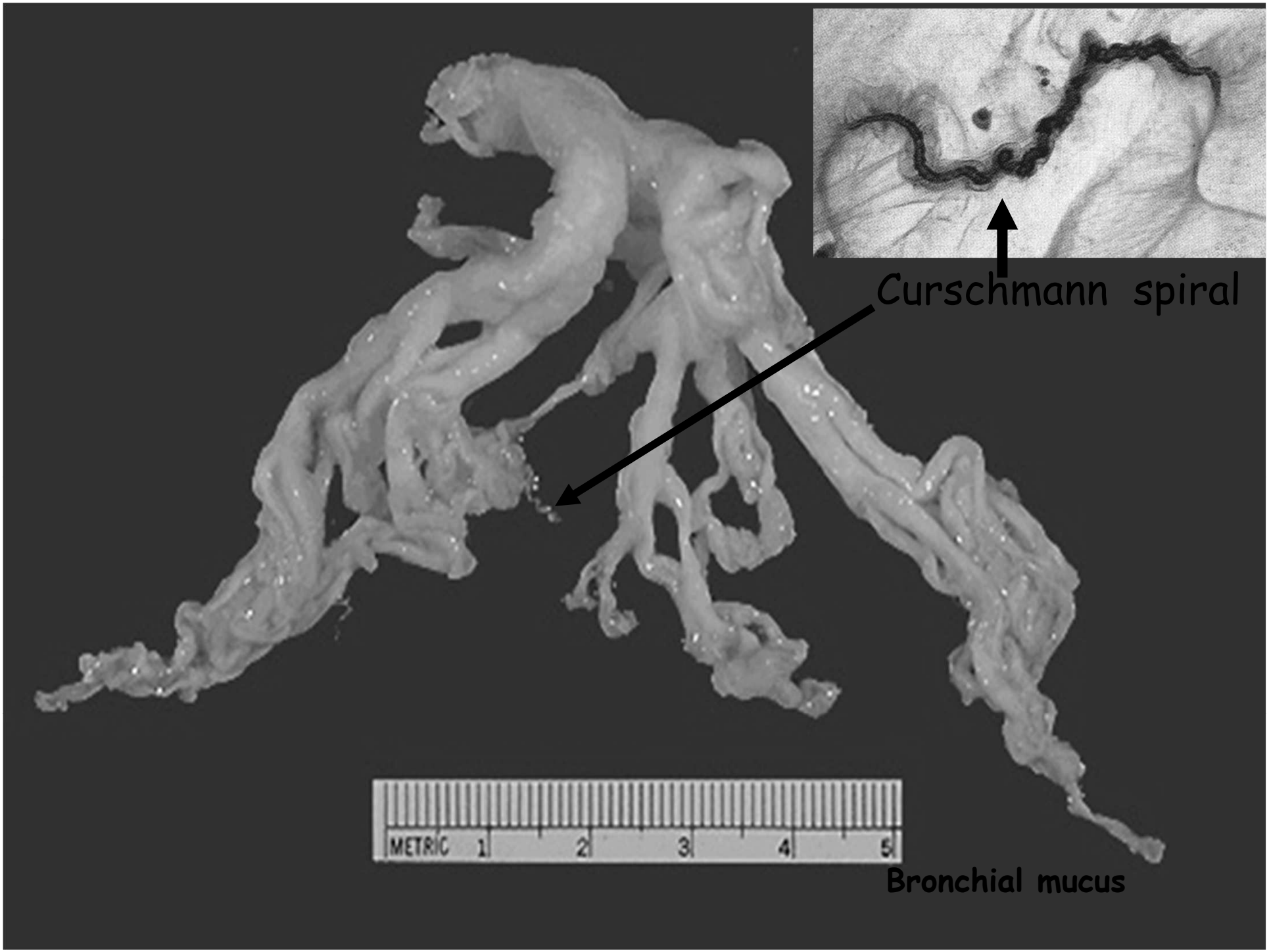
status asthmaticus

Status asthmaticus

Hypoxia

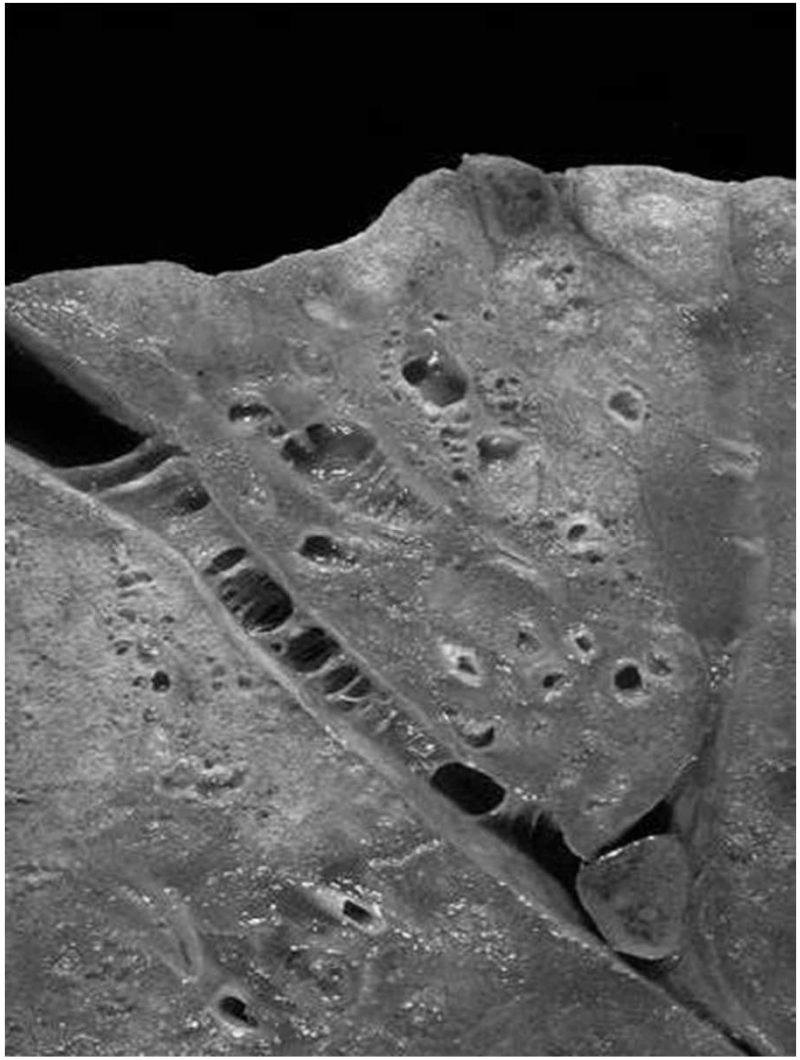
Acidosis

Death


Curschmann spiral
METRIC 1 2 3 4 5
Bronchial mucus

Bronchiectasis

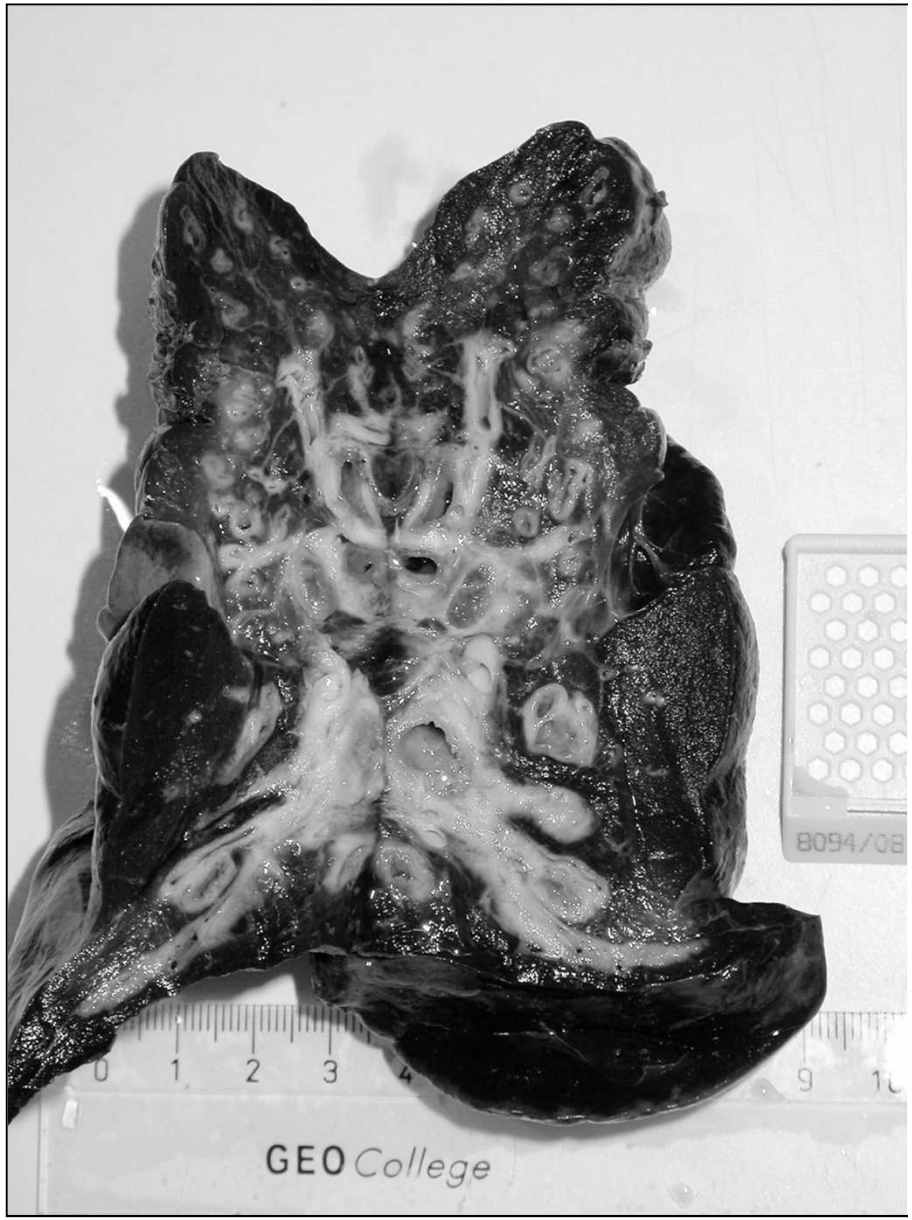
Permanent dilation of bronchi, caused by destruction of muscle and elastic tissue resulting from or associated with chronic necrotizing infections



Bronchiectasis

Symptoms:

- cough
- haemoptoe
- foul smelling sputum
- hypercapnia
- hypoxaemia

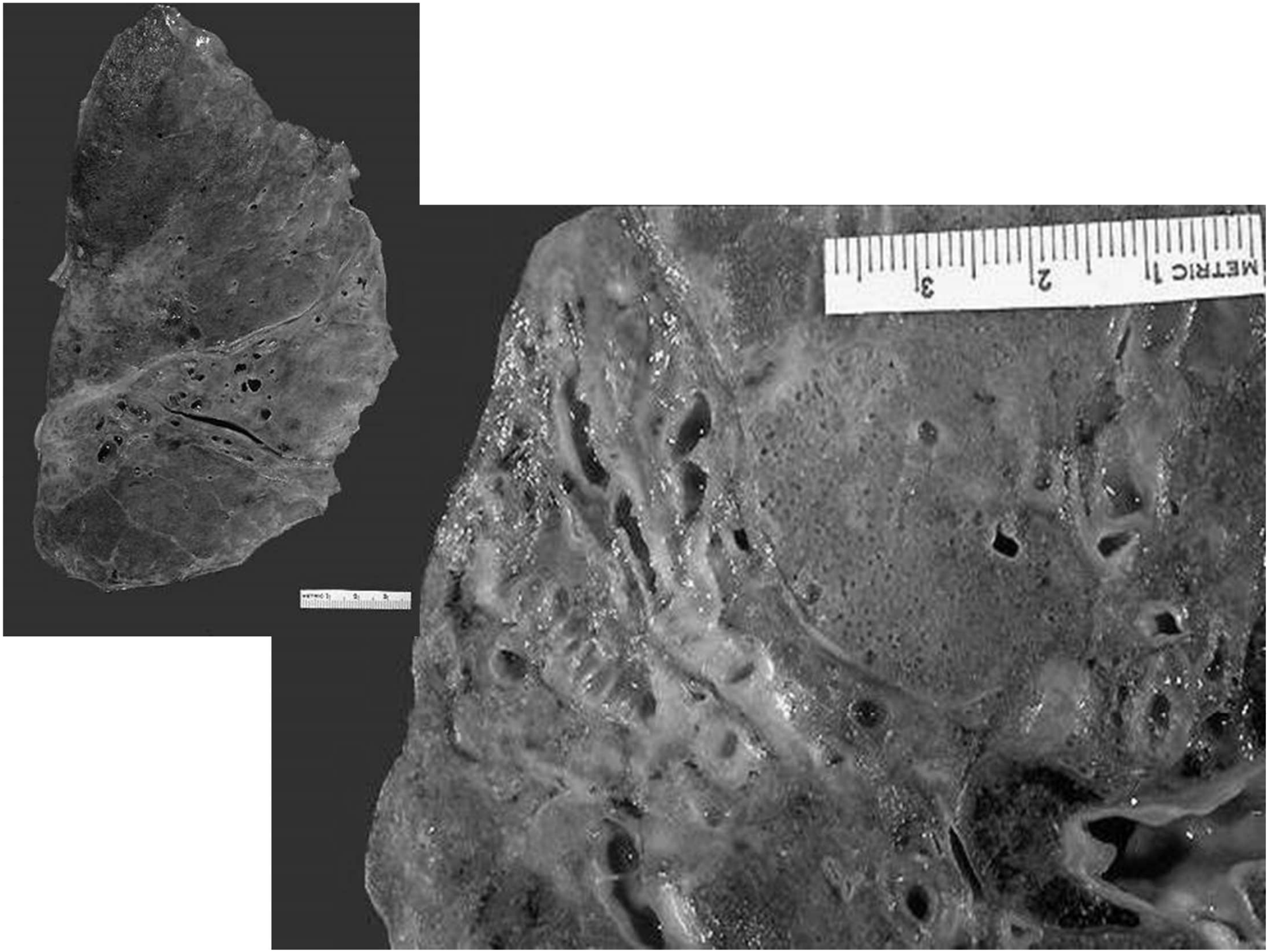


Bronchiectasis

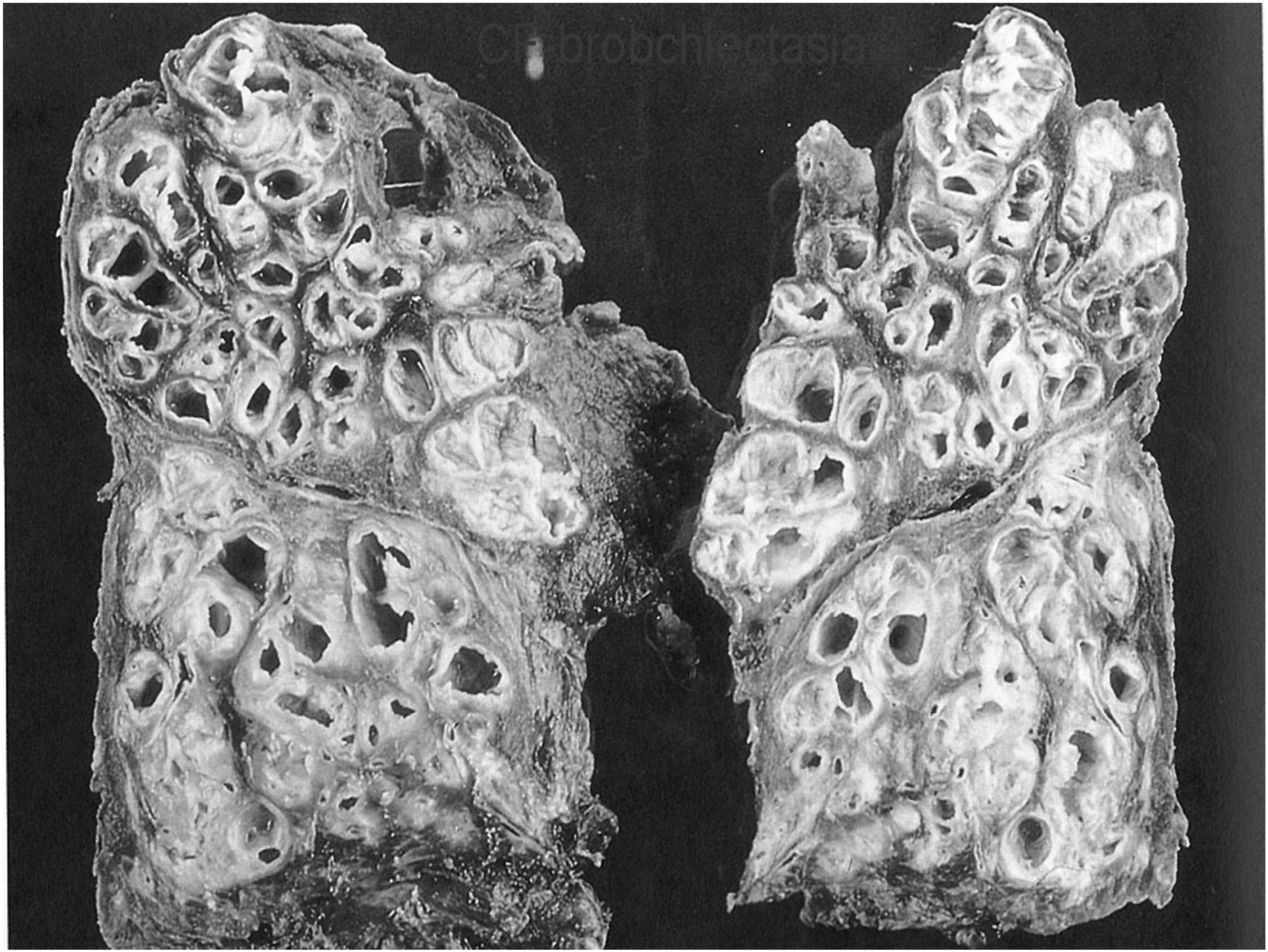
Pathogenesis

- Congenital (1%)
  - Ciliary dyskinesis,
  - Cystic fibrosis,
  - Intralobar sequestration,
  - Immunodeficiency states
- Post-infectious

- Bronchial obstruction


METRIC 1
2
3


7246


CF bronchiectasis

Bronchiectasis

Micr.:

intense acute and chronic inflammation in the bronchial wall,

necrotizing ulceration,

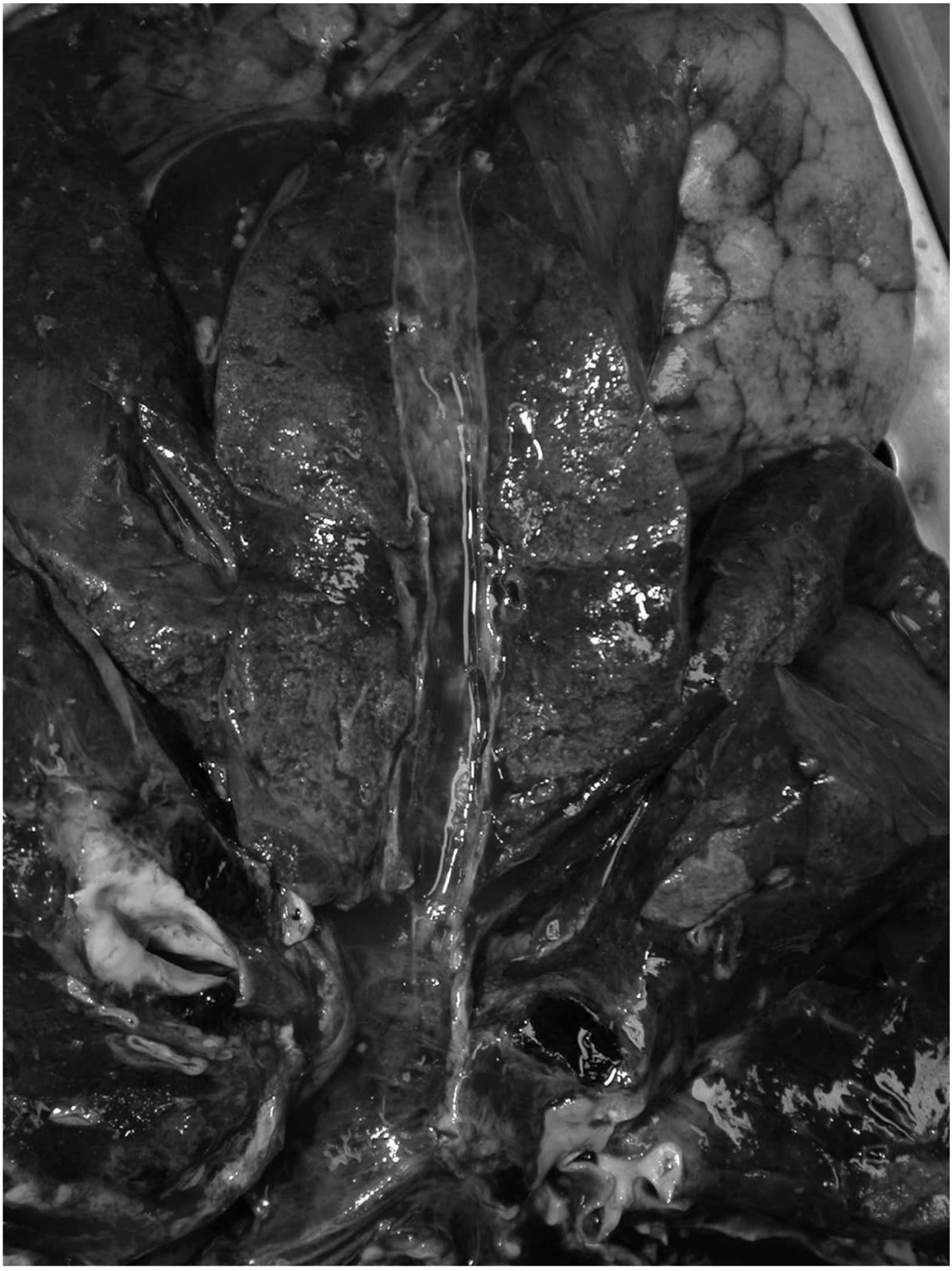
peribronchial fibrosis,

abscess formation

Bronchiectasis

Complications:

- Empyema
- Cor pulmonale
- Metastatic abscesses (brain)
- Amyloidosis
- SQCC



Restrictive Lung diseases

Diffuse interstitial (restrictive) lung diseases are a

heterogeneous group of disorders characterized predominantly *by*

diffuse and usually chronic involvement of the pulmonary connective tissue,

principally the *most peripheral* and *delicate interstitium* in the alveolar walls.

Restrictive Lung diseases

The hallmark of these disorders *is*

reduced compliance

(i.e., more pressure is required to expand the lungs because they are stiff),

which in turn necessitates **increased effort** of **breathing** (dyspnea).

Restrictive Lung diseases

Fibrosing

- Usual interstitial pneumonia (idiopathic pulmonary fibrosis)
- Nonspecific interstitial pneumonia
- Cryptogenic organizing pneumonia
- Associated with collagen vascular disease
- Pneumoconiosis
- Associated with therapies (drugs, radiation)

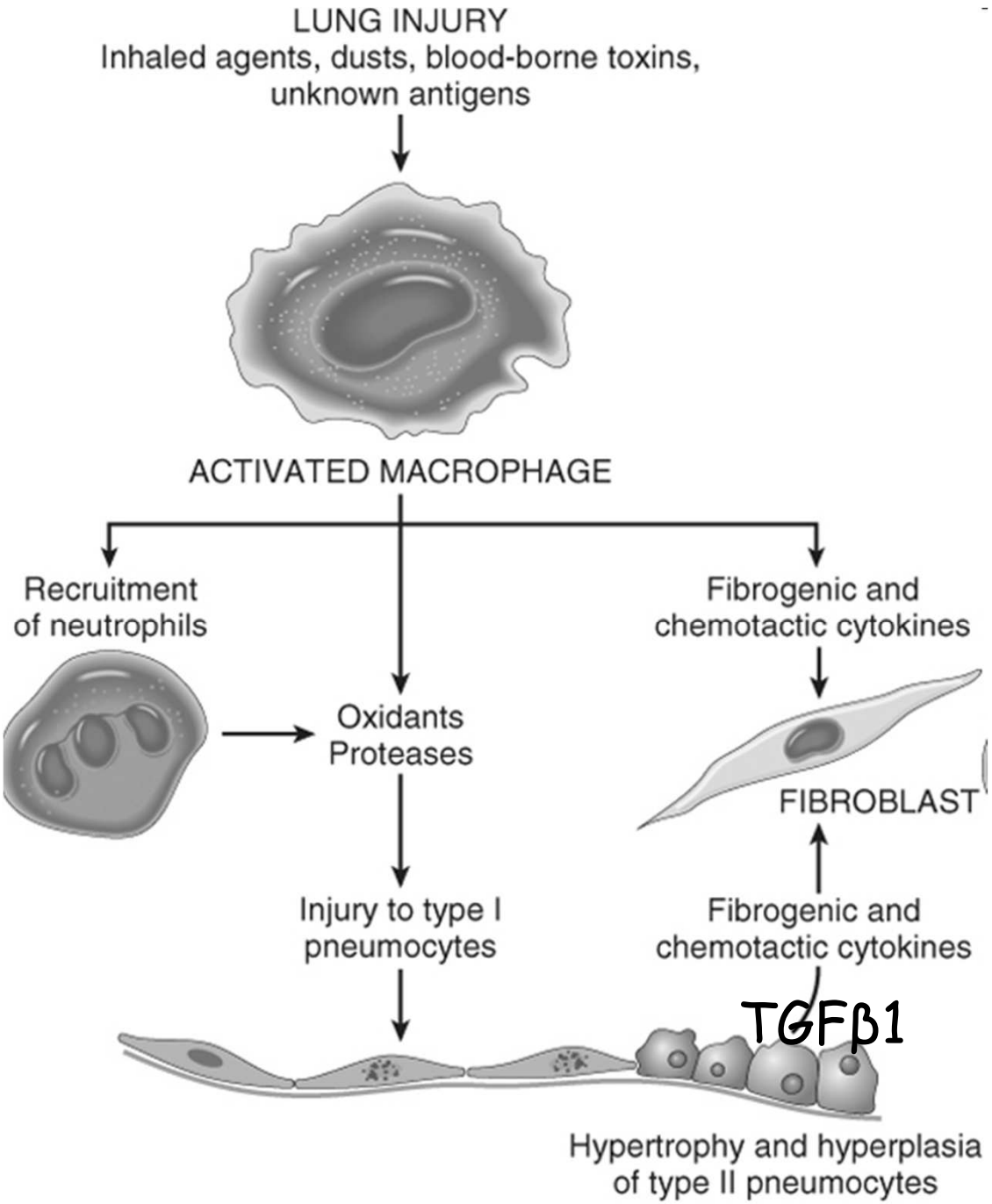
Granulomatous

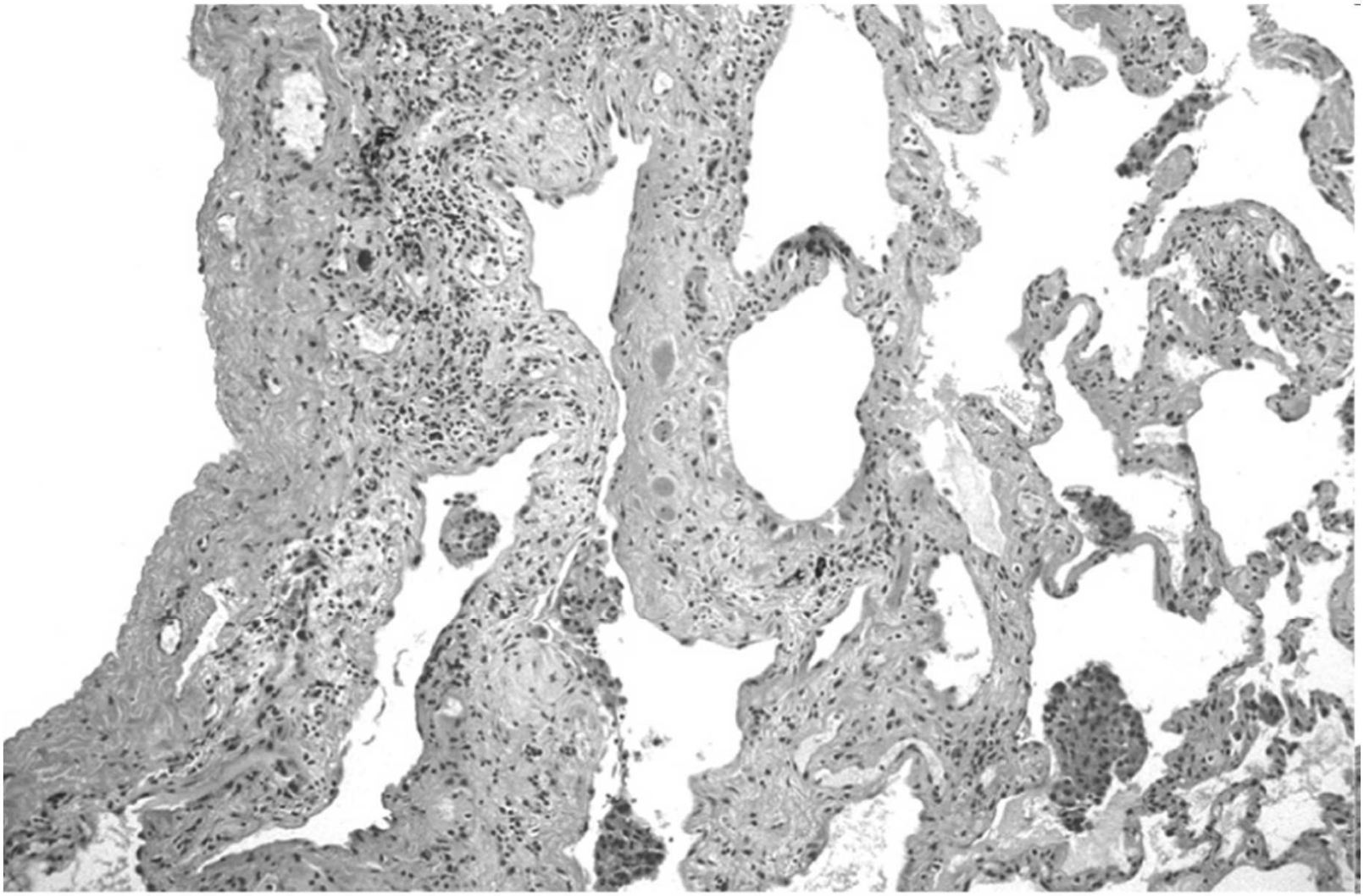
- Sarcoidosis
- Hypersensitivity pneumonia

Eosinophilic

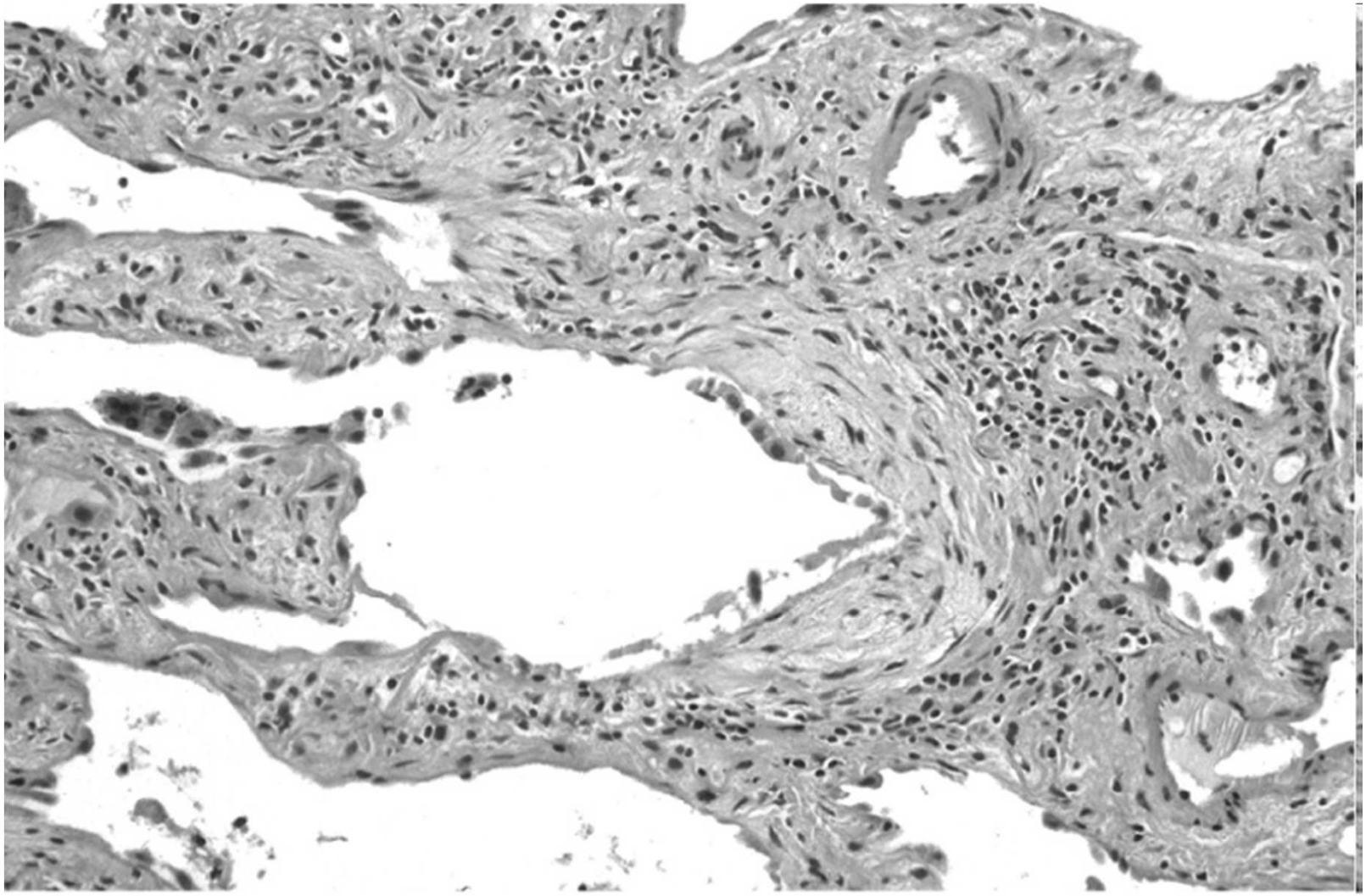
Smoking Related

- Desquamative interstitial pneumonia
- Respiratory bronchiolitis

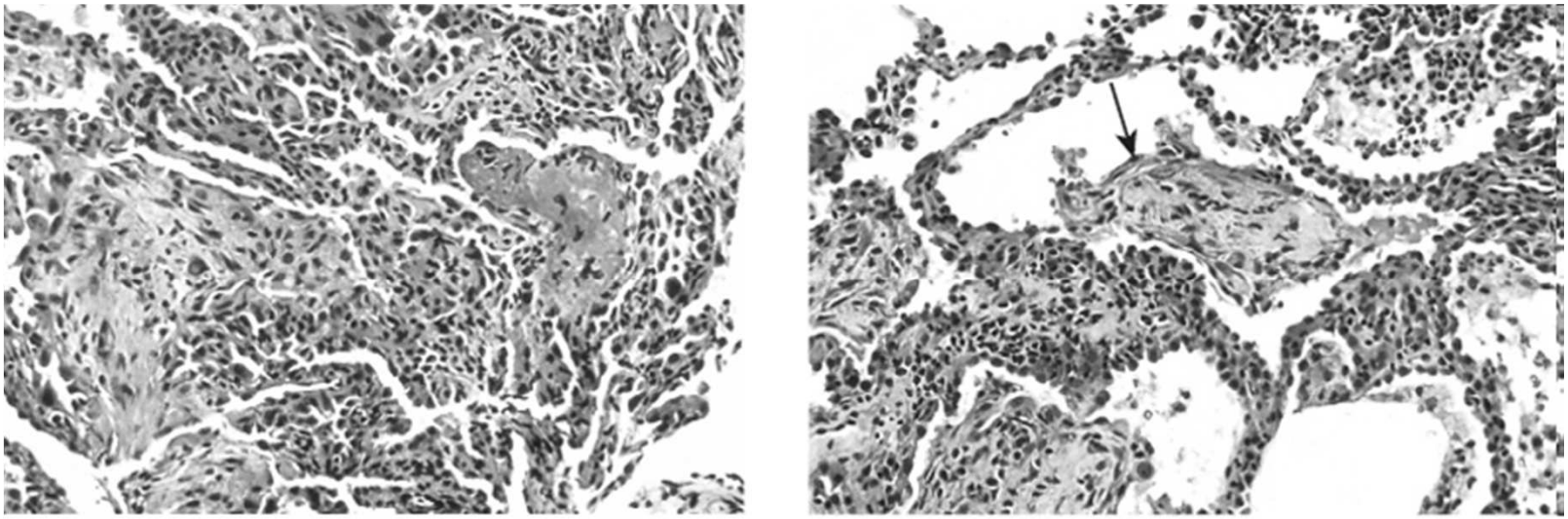

LUNG INJURY
Inhaled agents, dusts, blood-borne toxins, unknown antigens
ACTIVATED MACROPHAGE
Recruitment of neutrophils
Oxidants Proteases
Fibrogenic and chemotactic cytokines
FIBROBLAST
Injury to type I pneumocytes
Fibrogenic and chemotactic cytokines
TGFβ1
Hypertrophy and hyperplasia of type II pneumocytes
© Elsevier. Kumar et al: Robbins Basic Pathology 8e - www.studentconsult.com



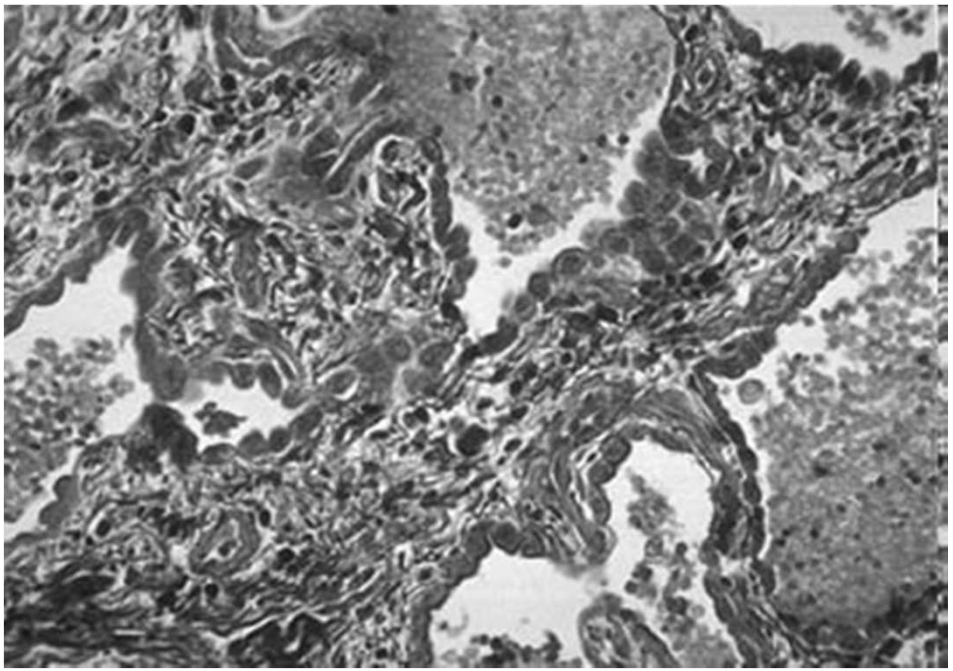
Usual interstitial pneumonia. The fibrosis, which varies in intensity, is more pronounced in the subpleural region.



Usual interstitial pneumonia. Fibroblastic focus with fibers running parallel to surface and bluish myxoid extracellular matrix.

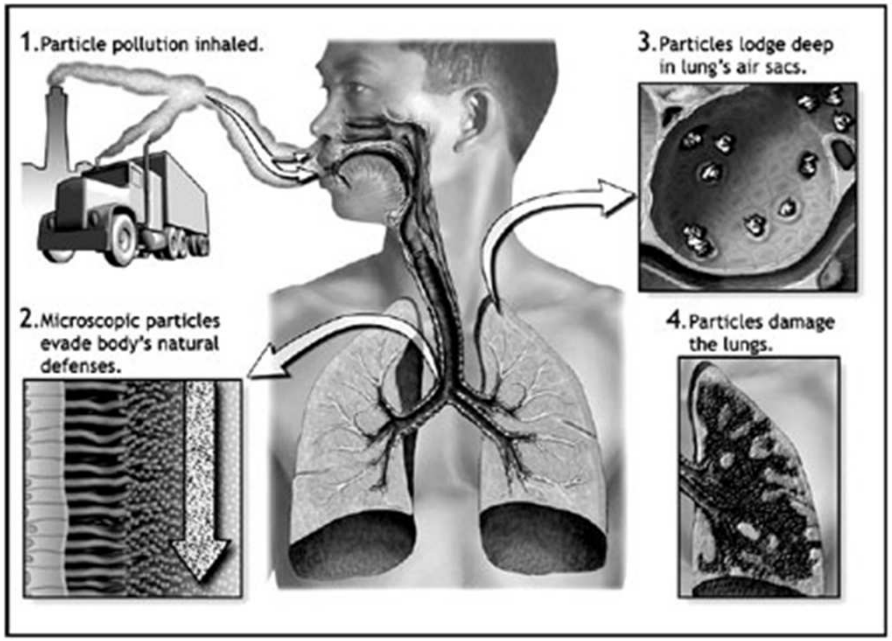


Cryptogenic organizing pneumonia. Alveolar spaces are filled with balls of fibroblasts (*arrow*).



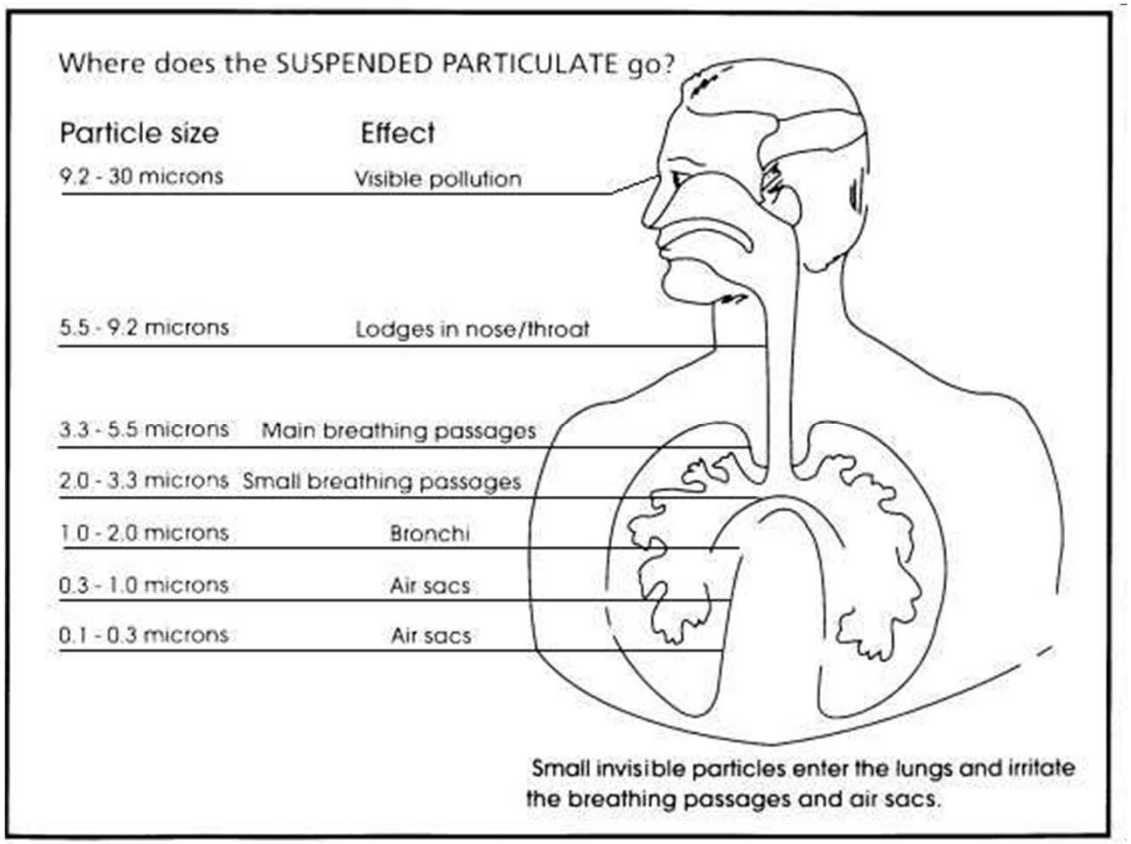
Pneumoconiosis

originally coined to describe the *non-neoplastic lung reaction* to inhalation of *mineral dusts*.



Pneumoconiosis

Particles that are 1 to 5 μm are the most dangerous



Coal dust

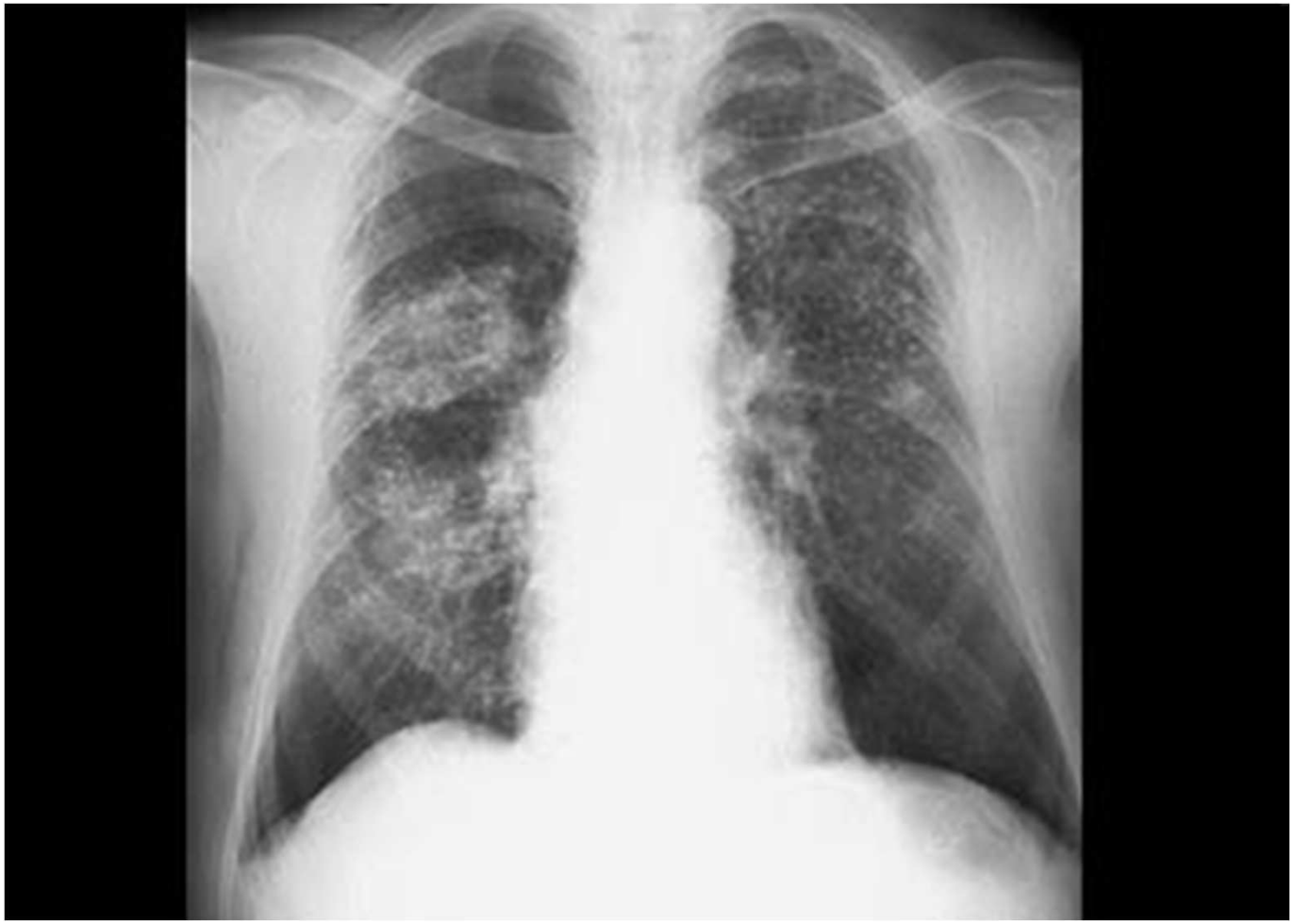
Coal workers

Silica Silicosis

(Sandblasting, quarrying, mining, stone cutting, foundry work, ceramics)

Asbestos Asbestosis

(Mining, milling, and fabrication of ores and materials; installation and removal of insulation)



Progressive massive fibrosis (PMF)

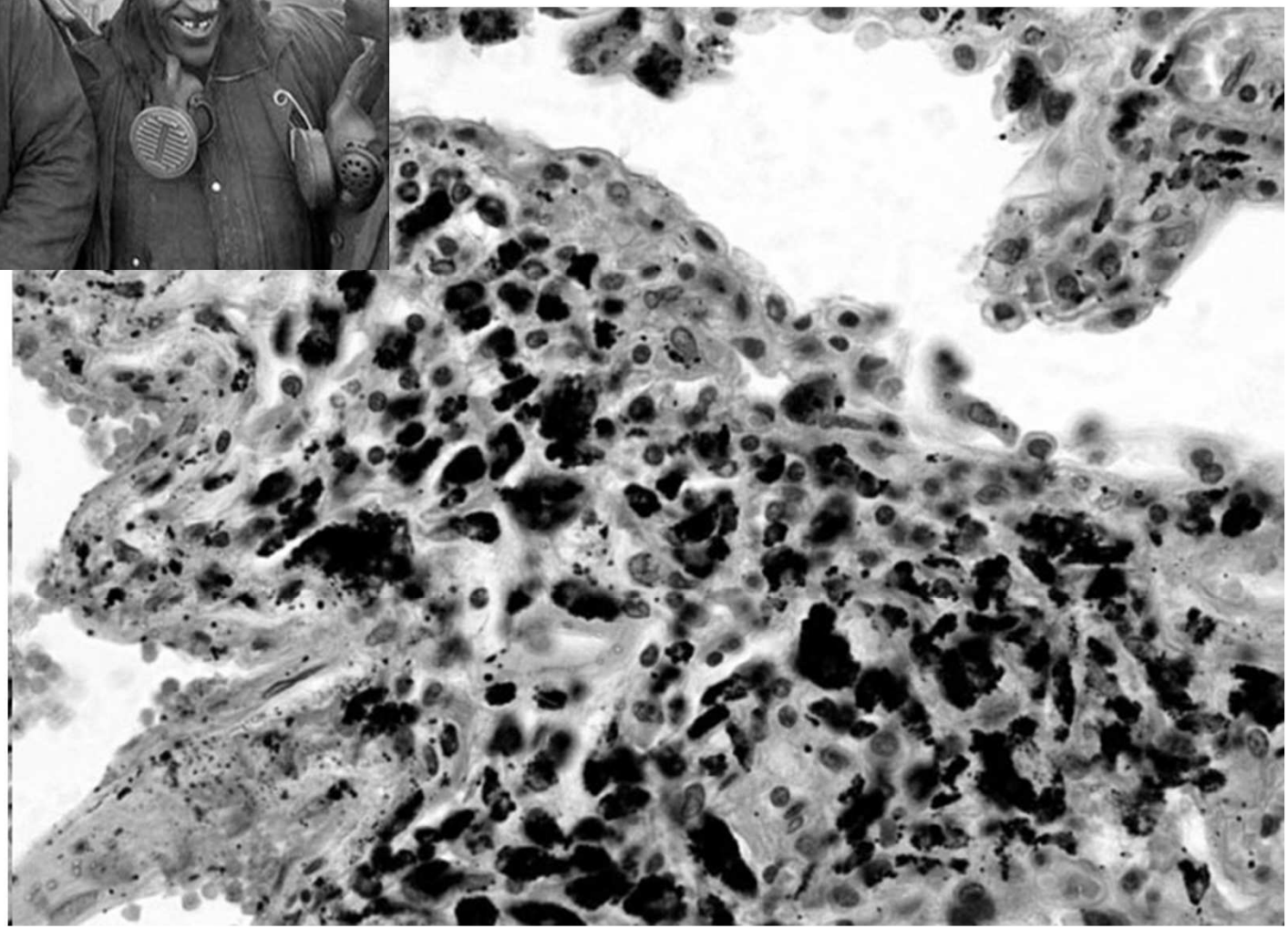
Coal dust

Simple coal workers' pneumoconiosis: macules and nodules

Complicated coal workers' pneumoconiosis: PMF – relatively rare, starts in the upper lobes

PMF progresses in the absence of further exposure

NO increased frequency of bronchogenic carcinoma



Silica Silicosis

(Sandblasting, quarrying, mining, stone cutting, foundry work, ceramics)

~ occurs in crystalline and amorphous forms,

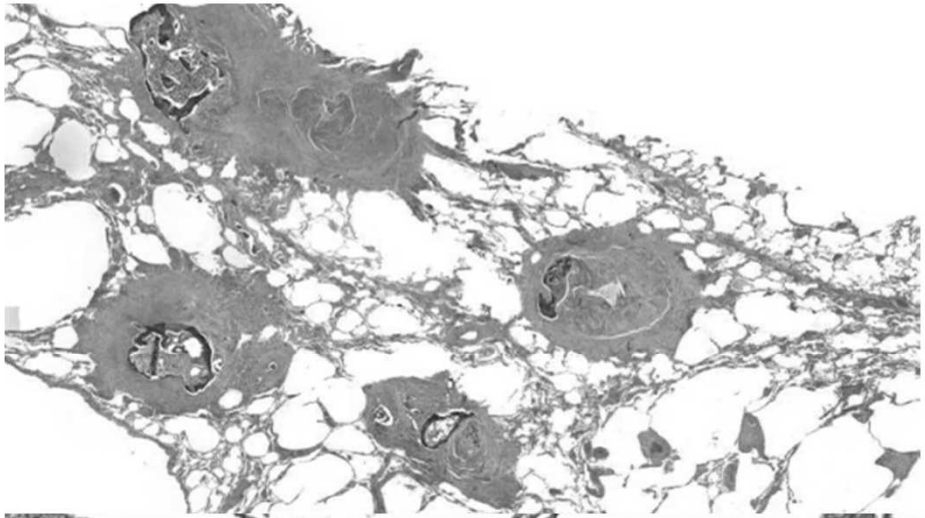
crystalline forms (**quartz**, cristobalite, tridymite) fibrogenic

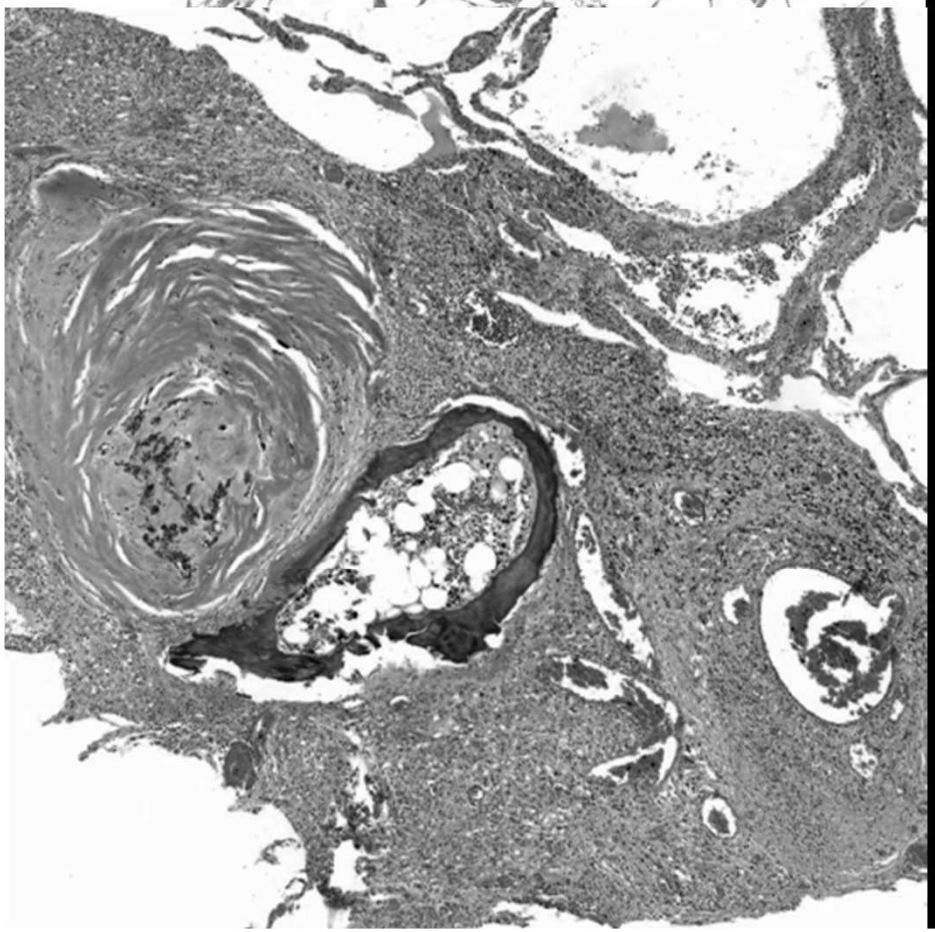
Inhalation, interaction with epithelial cells ,macrophages.

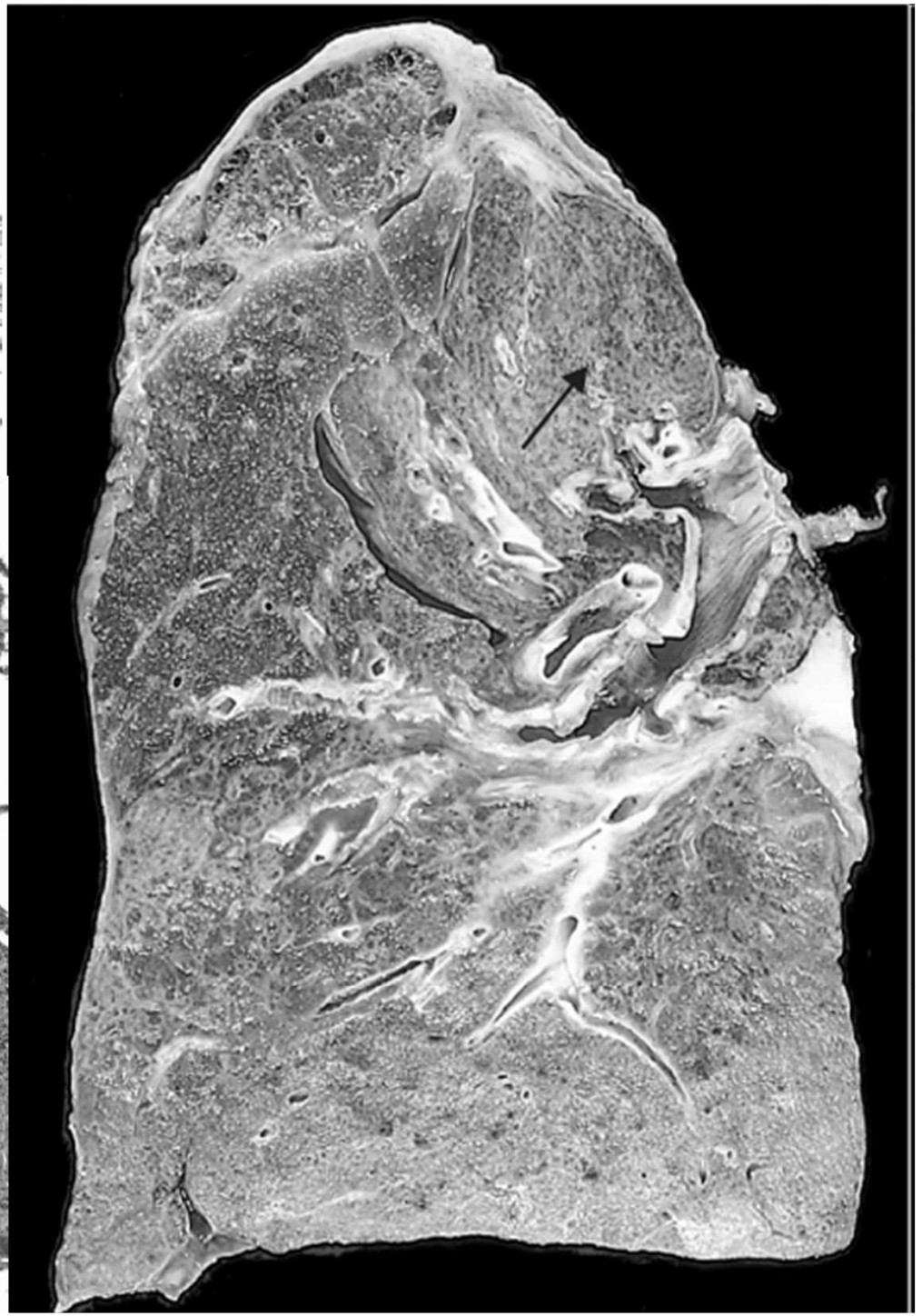
activation and release of mediators by pulmonary macrophages, including IL-1, **TNF**, fibronectin, lipid mediators, oxygen-derived free radicals, fibrogenic cytokines

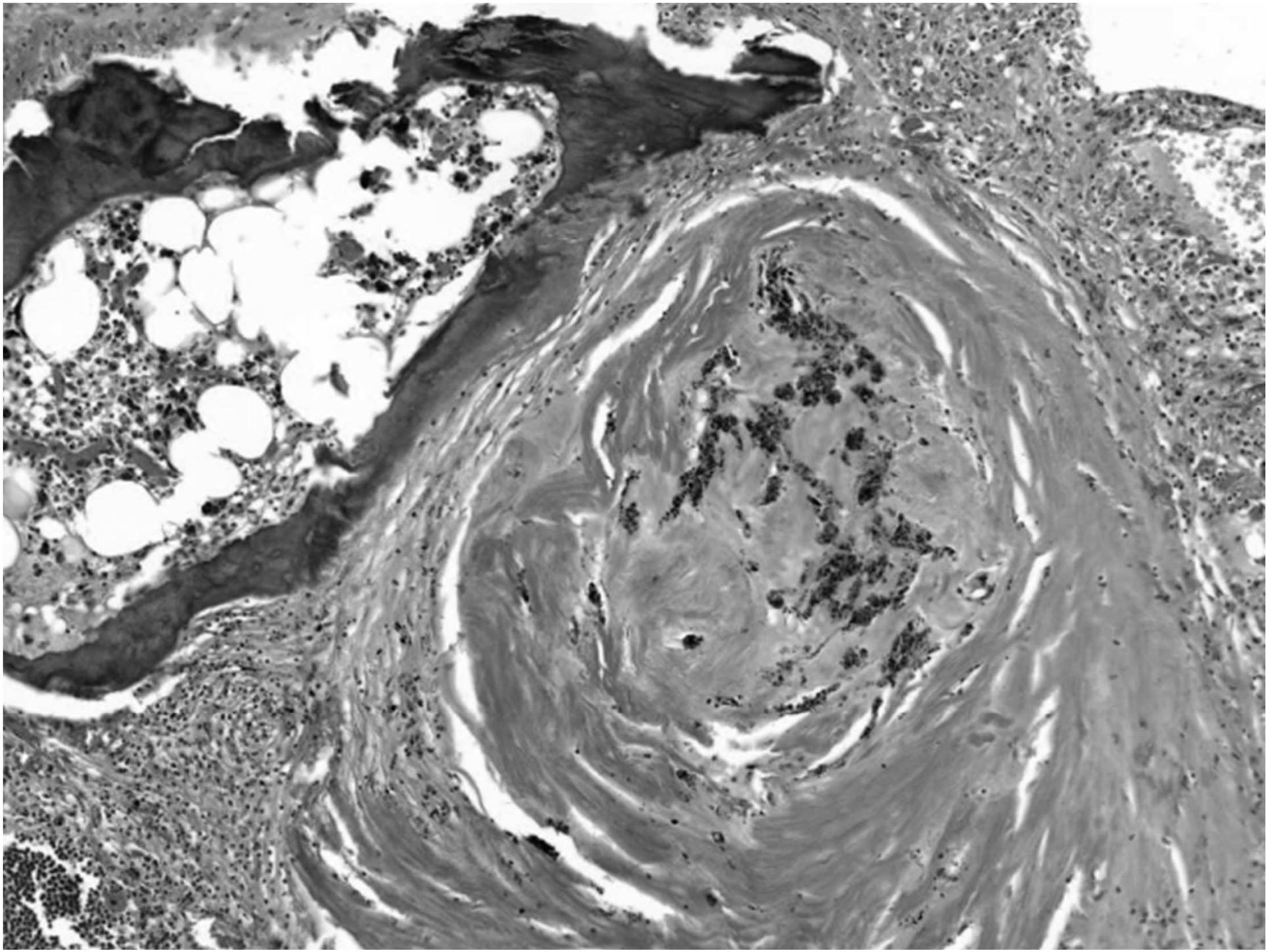
when mixed with other minerals, quartz has a reduced fibrogenic effect.

hematite (iron containig quartz) protective.









Silicosis

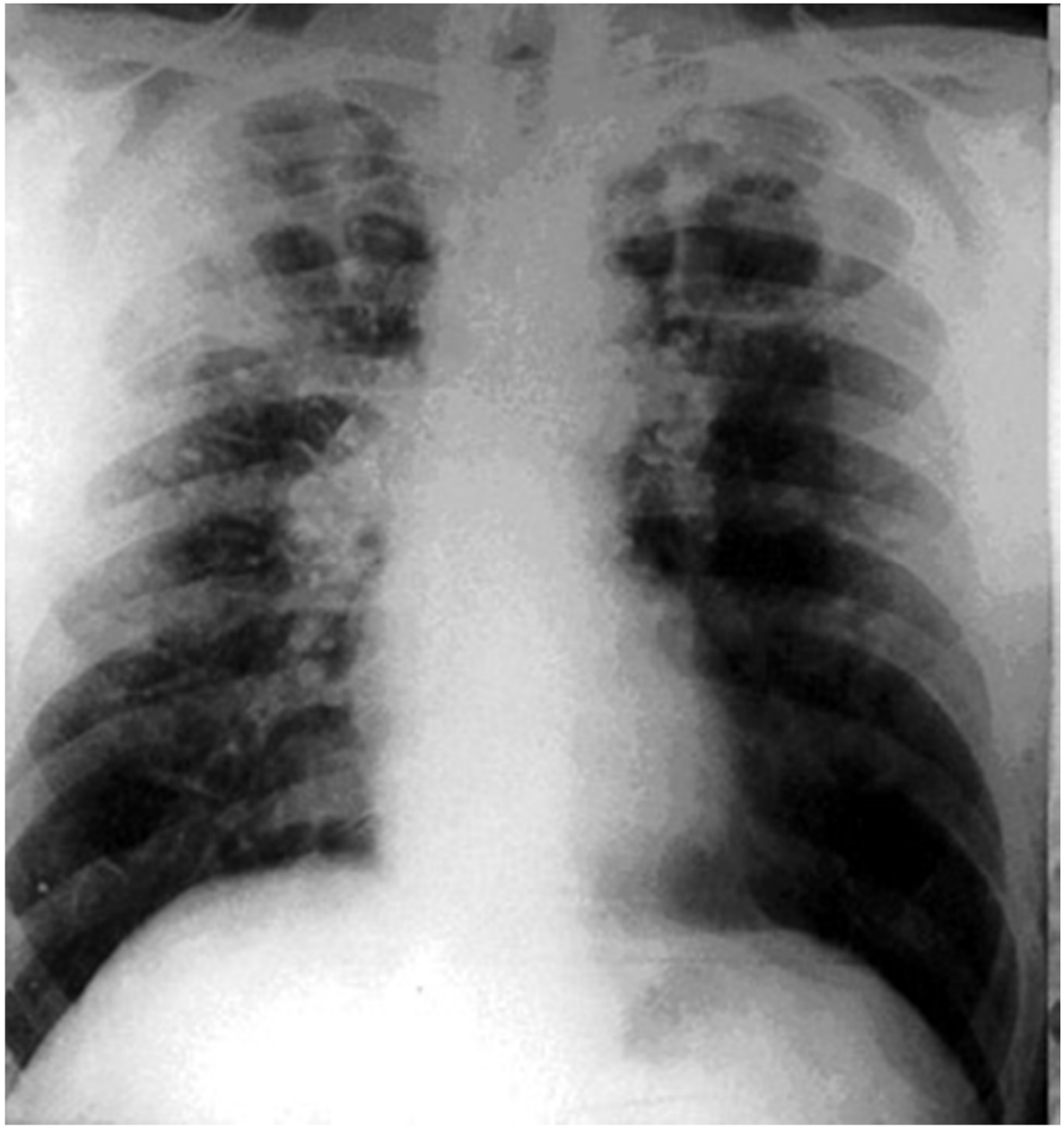
increased susceptibility to tuberculosis
(depression of cell-mediated immunity)

progression (even if the person is no longer exposed)
starts in the upper lobes

crystalline silica from occupational sources is carcinogenic (controversial)

Slow progression, CF, dyspnoe relatively late in the course

Silicotuberculosis



Multiple calcified 1 – 5 mm pulmonary nodules.
Eggshell calcifications in hilar lymph nodes bilaterally.
Cavitary lesions in both lung apices.

Asbestos Asbestosis

(Mining, milling, and fabrication of ores and materials; installation and removal of insulation)

Fibrosis starts in the lower lobes

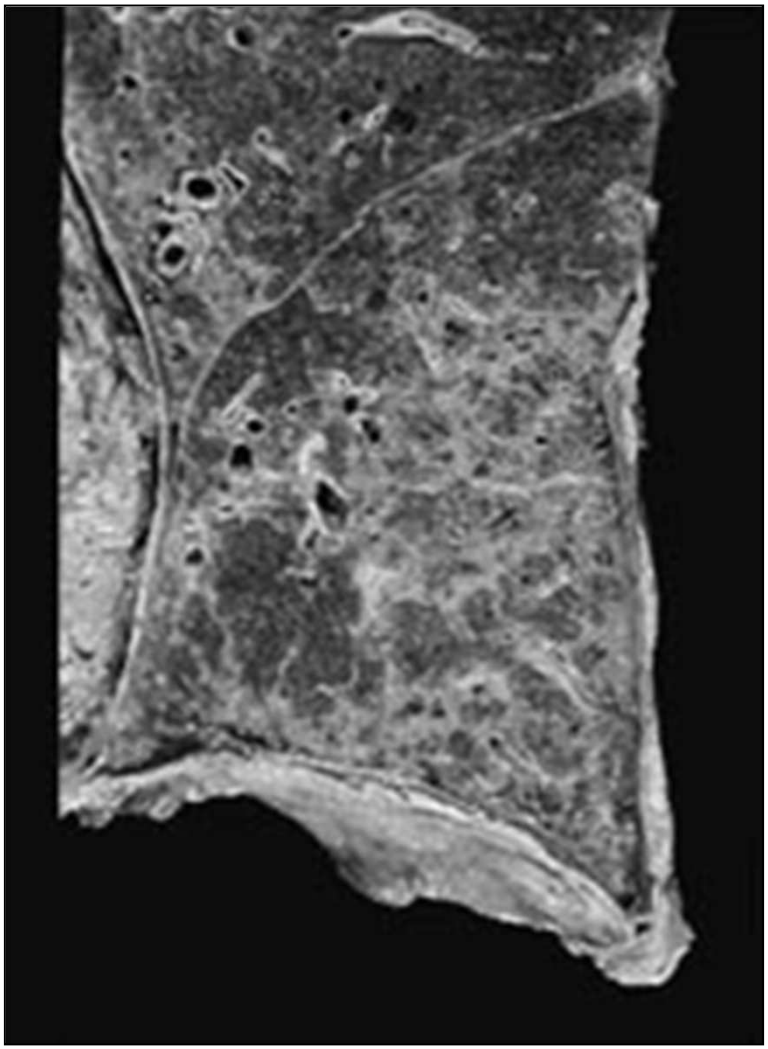
progressively worsening dyspnea , 10-20 years after exposure

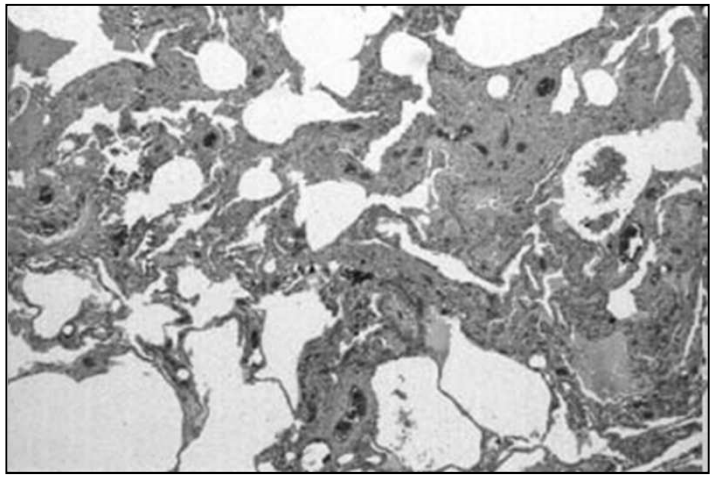
Asbestos Asbestosis

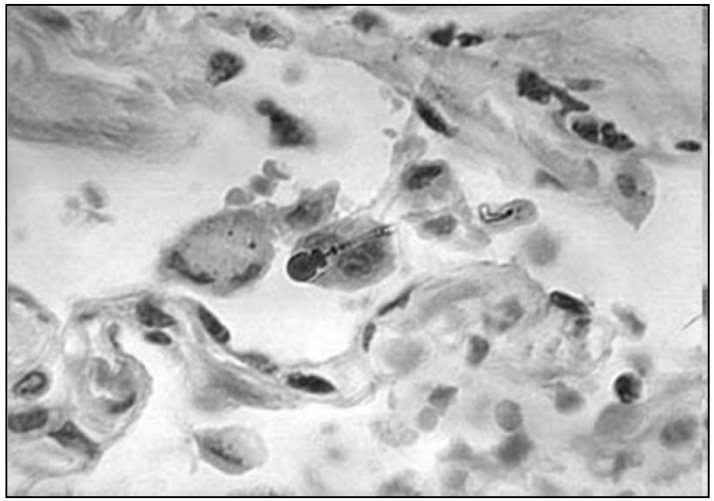
- Diffuse fibrosis,
- Pleural effusions,
- Pleural plaques;
- Mesothelioma;

Carcinoma of the lung and larynx
(cigarette smoking!)

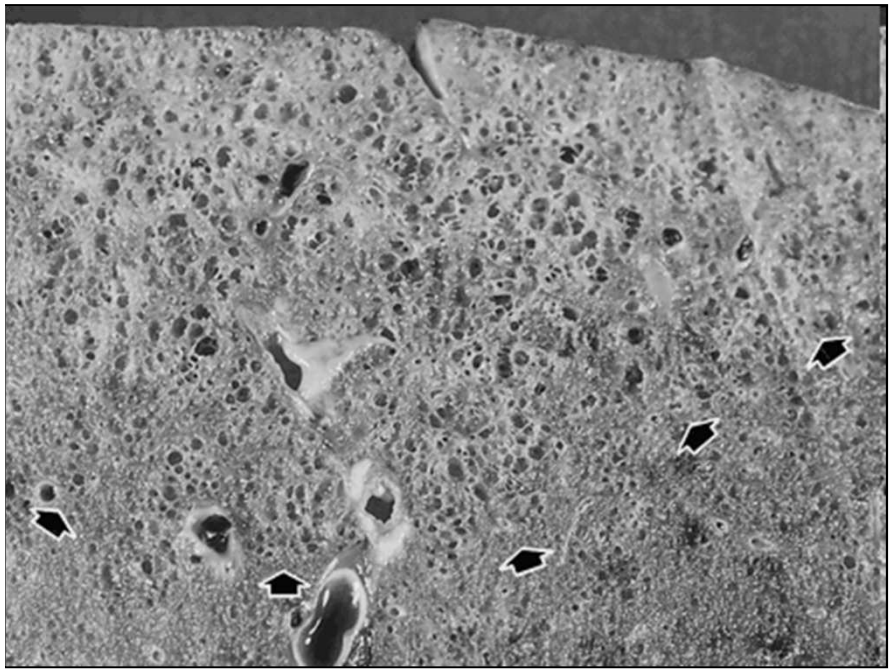
Asbestosis



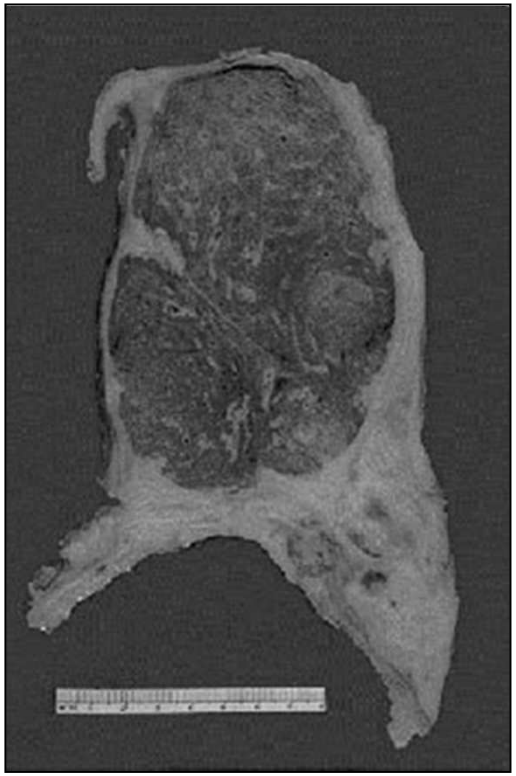




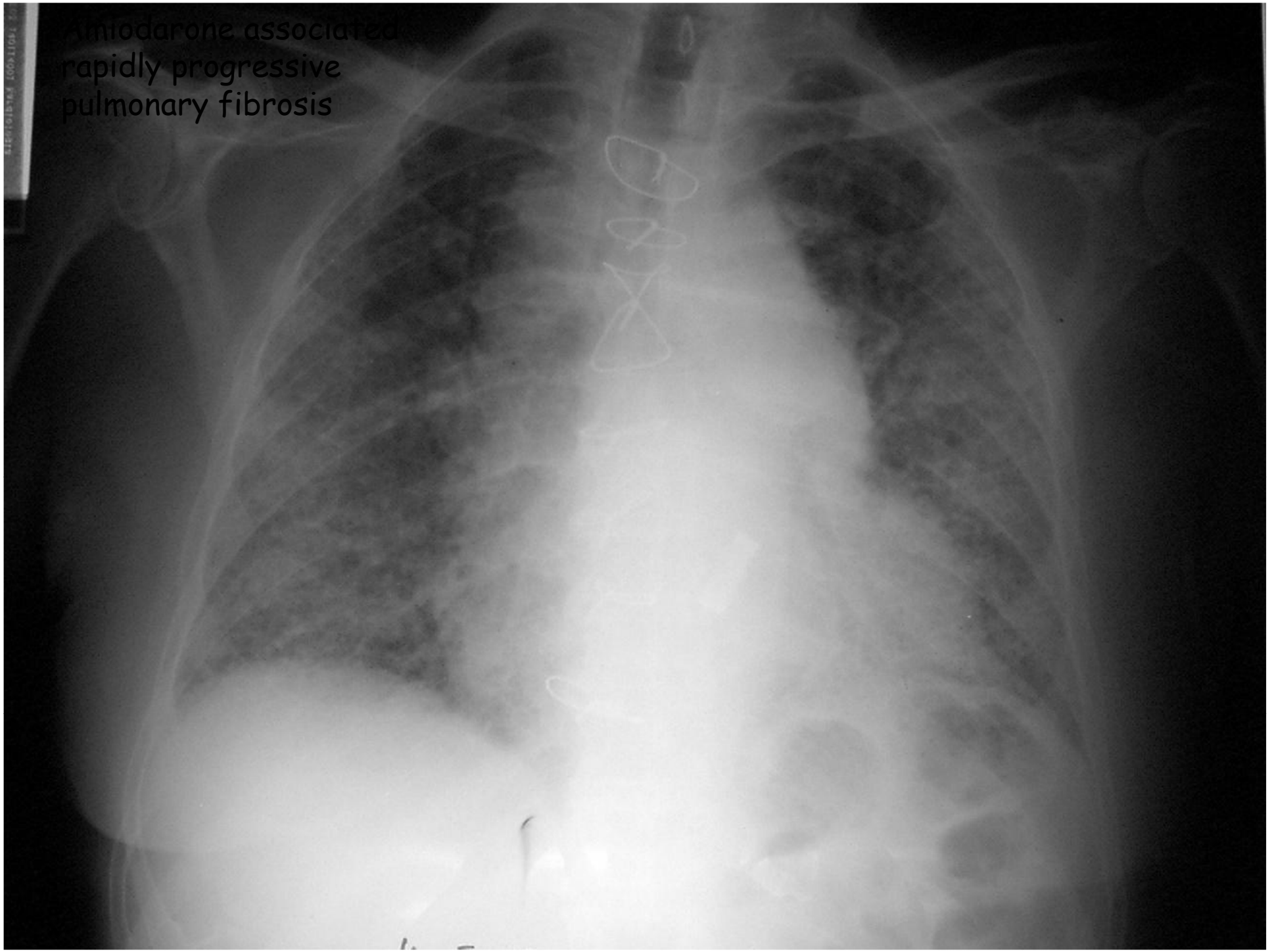
pleural thickening - localized interstitial pulmonary fibrosis.

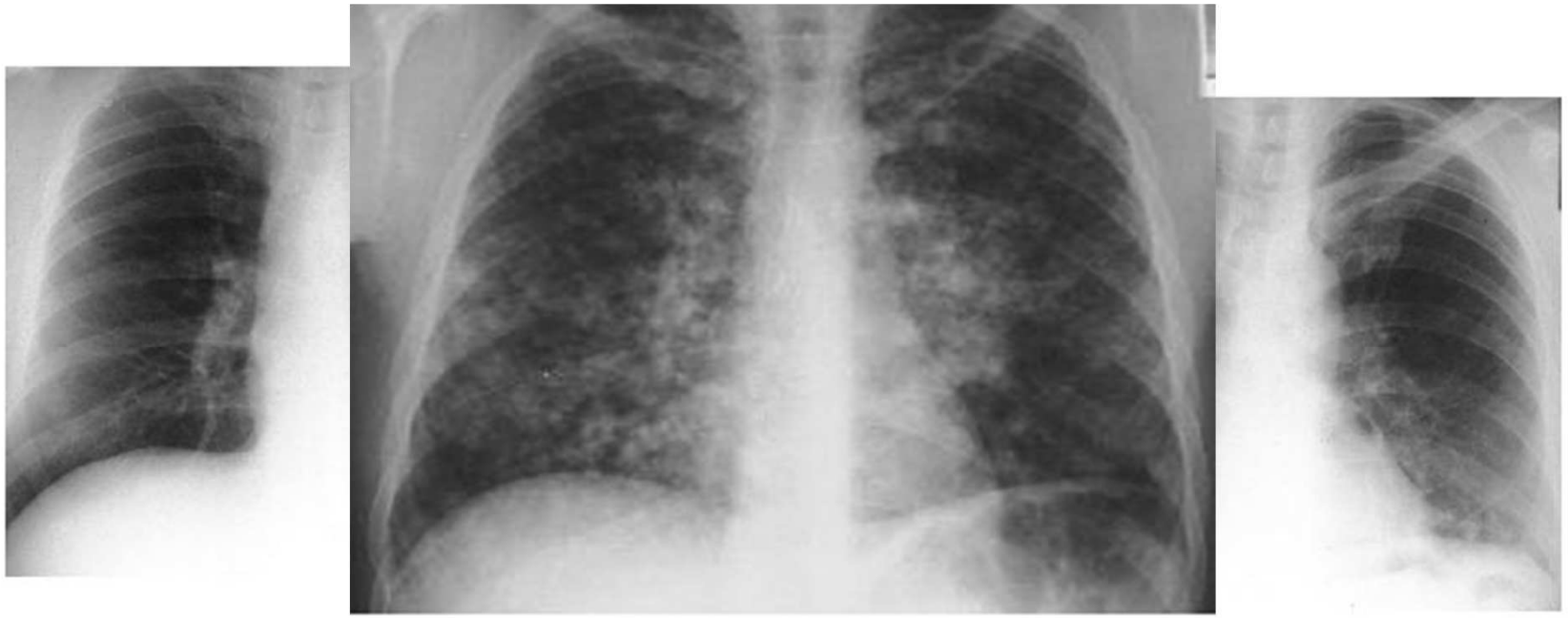


Honeycomb lung

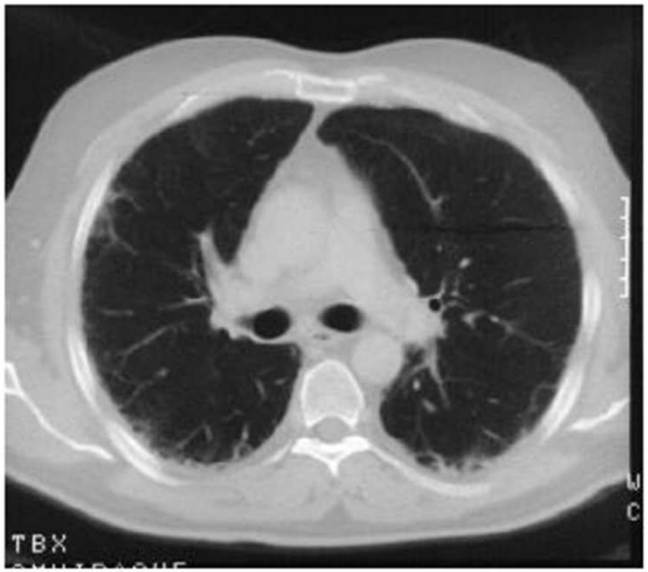


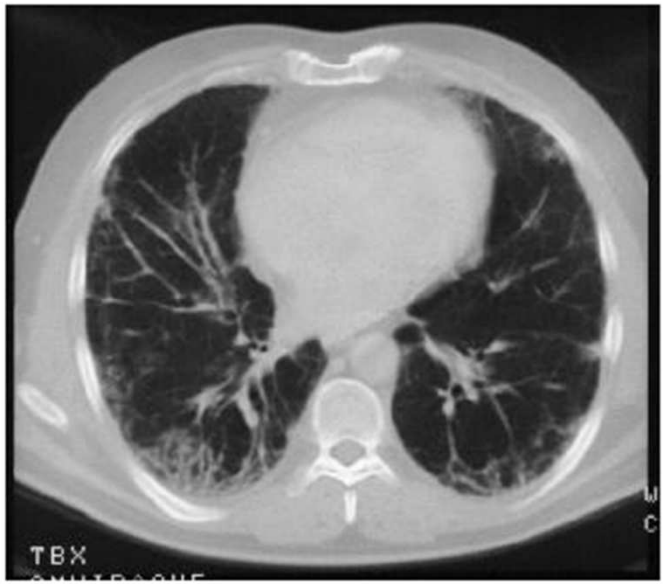
Mesothelioma


Amiodarone associated
rapidly progressive
pulmonary fibrosis



Result of chemotherapy





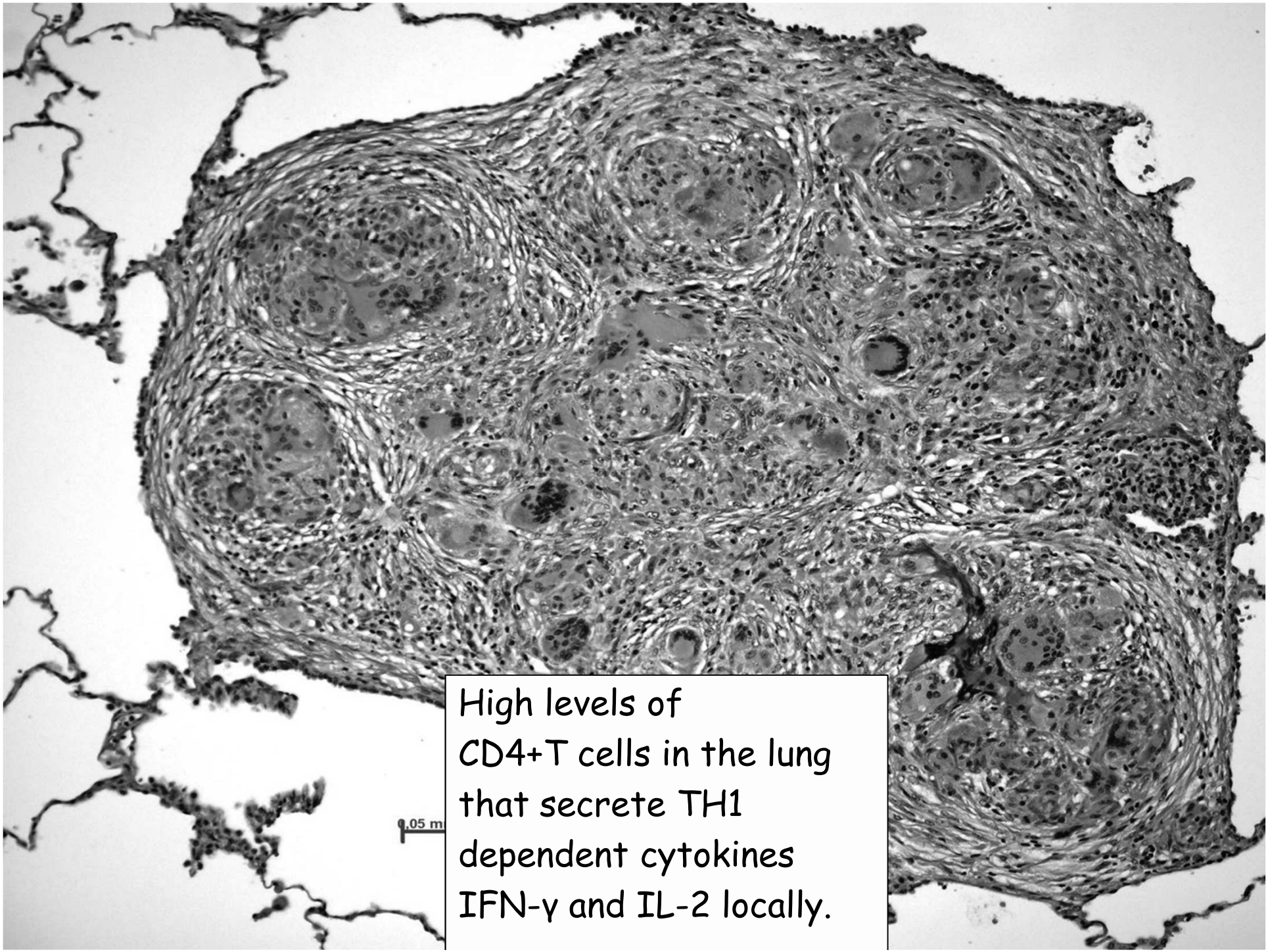
Sarcoidosis

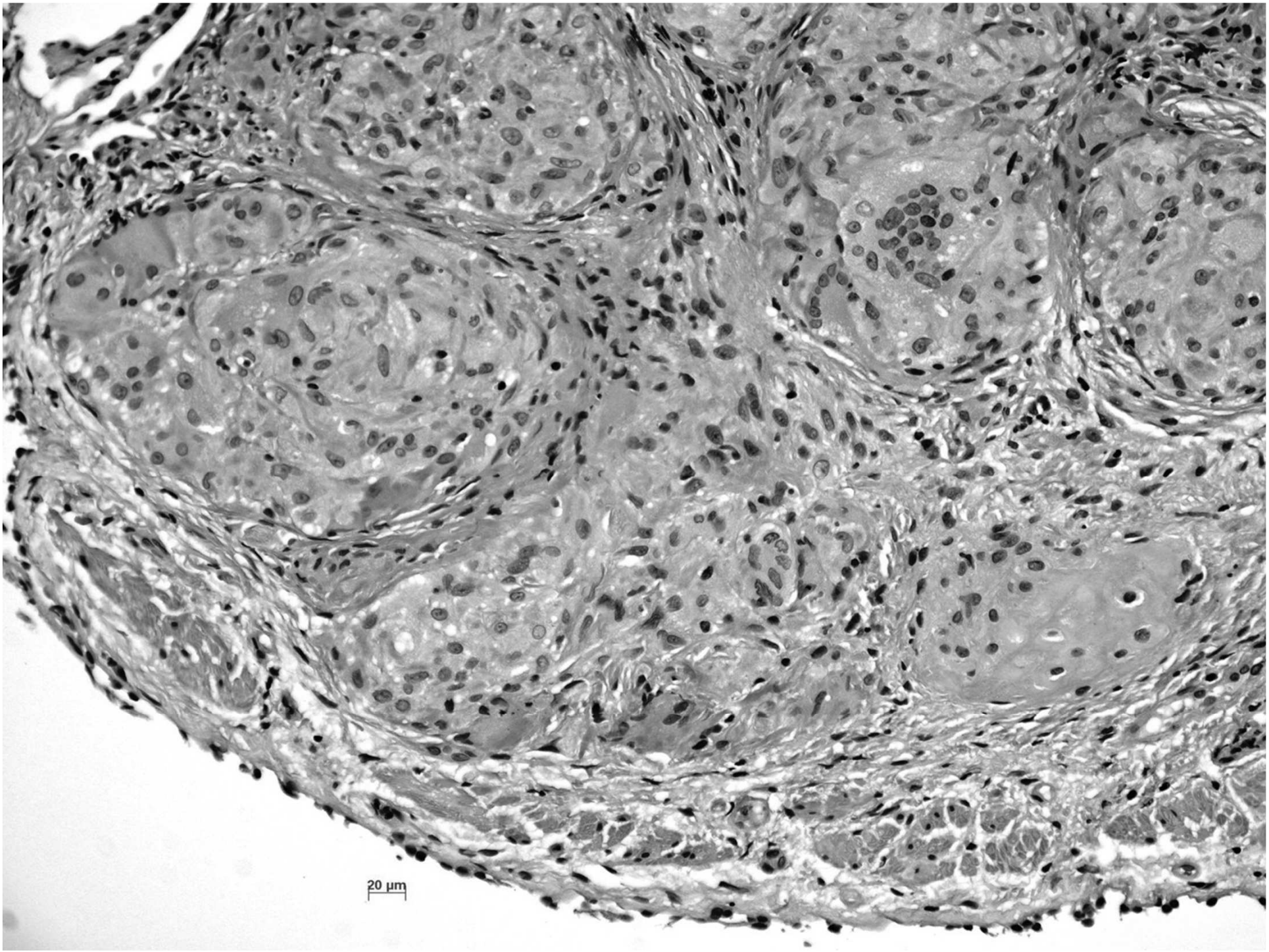
Multisystem disease of unknown etiology characterized by noncaseating granulomas in many tissues and organs

90% lung involvement

Restrictive pulmonary disease

Higher prevalence among *nonsmokers.*


High levels of
CD4+T cells in the lung
that secrete TH1
dependent cytokines
IFN-γ and IL-2 locally.


20 µm

Sarcoidosis
Clinical manifestations

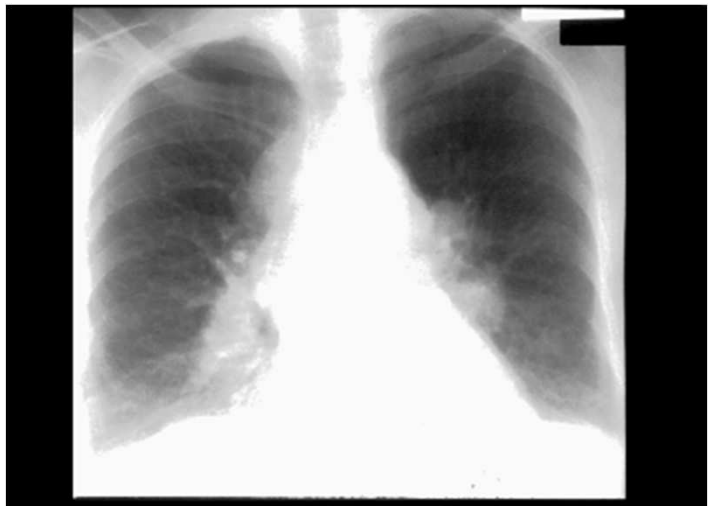
Lymph node enlargement (BHL)

Eye involvement (sicca syndrome [dry eyes], iritis, or iridocyclitis)

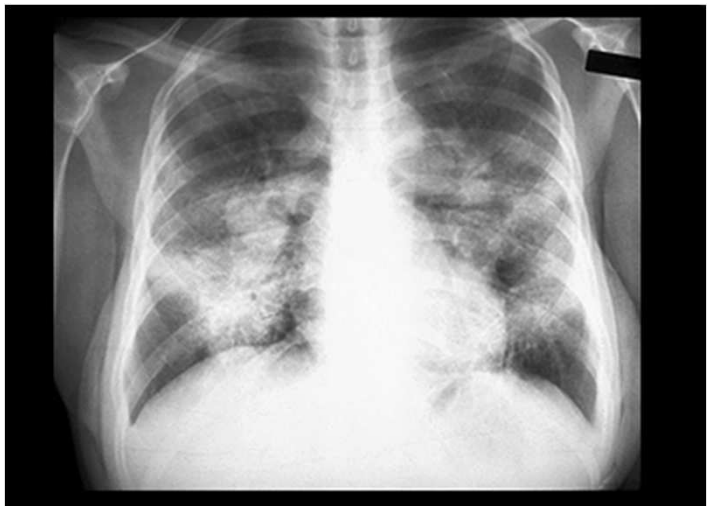
Skin lesions (erythema nodosum, lupus pernio)

Visceral (liver, skin, marrow)

Lung involvement: 90% of cases
granulomas and interstitial fibrosis



BHL



Pulmonary nodules

Hypersensitivity Pneumonitis

I*mmunologically mediated inflammatory lung disease* - primarily affects the alveoli - *allergic alveolitis.*

Occupational disease - heightened sensitivity to inhaled antigens (moldy hay)

Damage at the level of alveoli (Unlike bronchial asthma, in which *bronchi are the focus*)

Predominantly restrictive lung disease

with decreased diffusion capacity, lung compliance, and total lung volume

Hypersensitivity Pneumonitis

Morphology

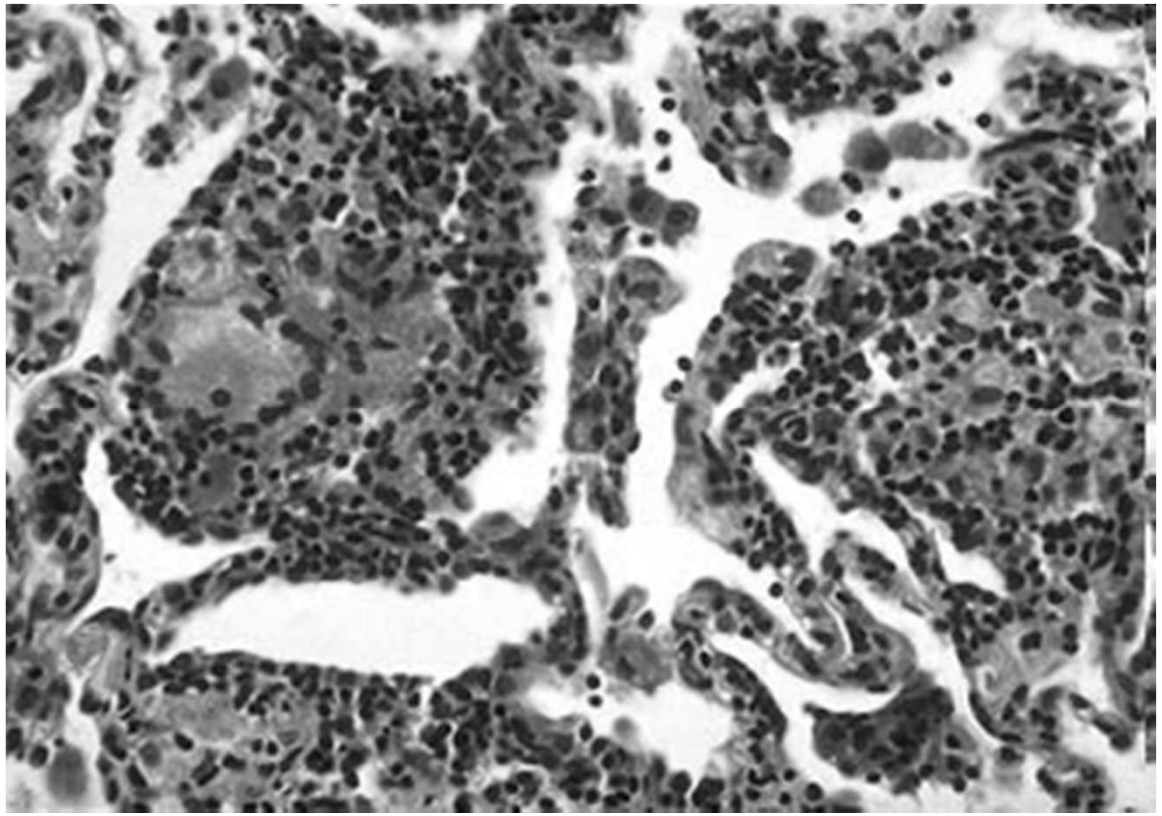
Patchy mononuclear cell infiltrates in the pulmonary interstitium, with characteristic **peribronchiolar** accentuation.

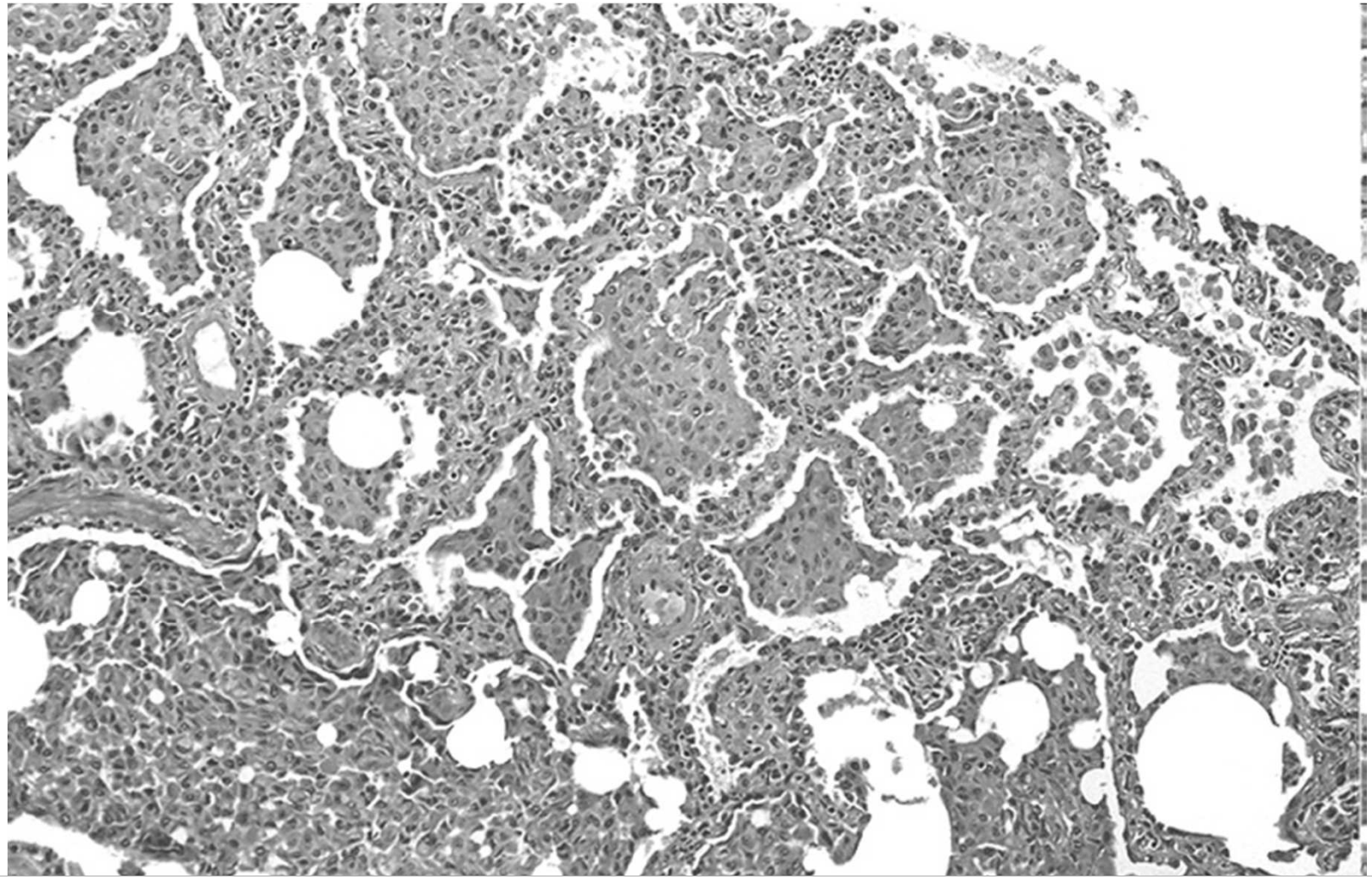
Lymphocytes predominate, plasma cells epithelioid cells.

Interstitial noncaseating granulomas peribronchiolar location.

Chronic cases: **diffuse interstitial fibrosis**

Hypersensitivity Pneumonitis





Desquamative interstitial pneumonia.

Accumulation of large numbers of mononuclear cells within the alveolar spaces with only mild fibrous thickening of the alveolar walls.