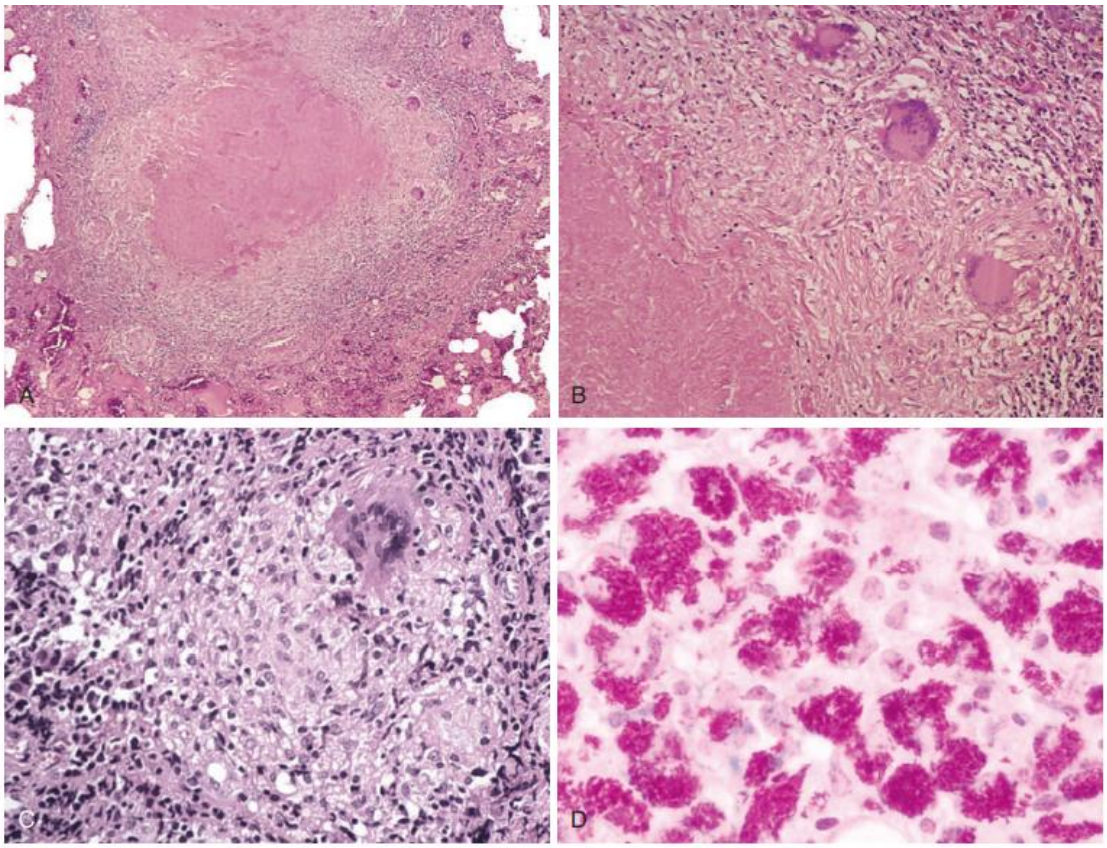

Fig. 13.35 The morphologic spectrum of tuberculosis. A characteristic tubercle at low magnification (A) and at higher power (B) shows central granular caseation surrounded by epithelioid and multinucleate giant cells. This is the usual response in individuals who develop cell-mediated immunity to the organism. (C) Occasionally, even in immunocompetent patients, tubercular granulomas may not show central caseation; hence, irrespective of the presence or absence of caseous necrosis, use of special stains for acid-fast organisms is indicated when granulomas are present. (D) In this specimen from an immunosuppressed patient, sheets of macrophages packed with mycobacteria are seen (acid-fast stain).

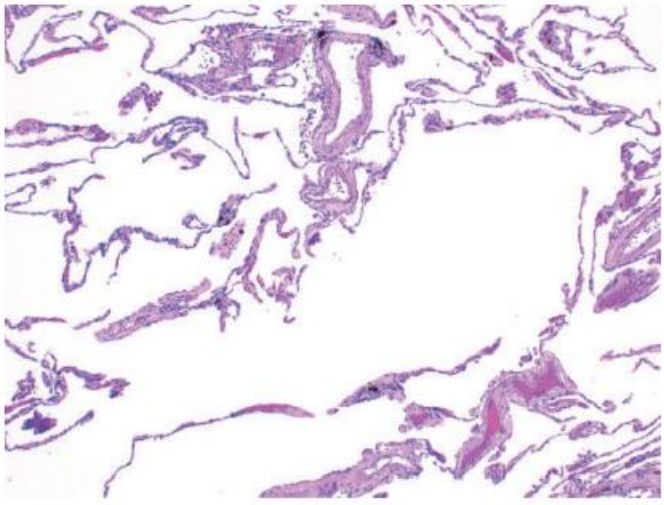

Fig. 13.7 Pulmonary emphysema. There is marked enlargement of the air spaces, with destruction of alveolar septa but without fibrosis. Note the presence of black anthracotic pigment.

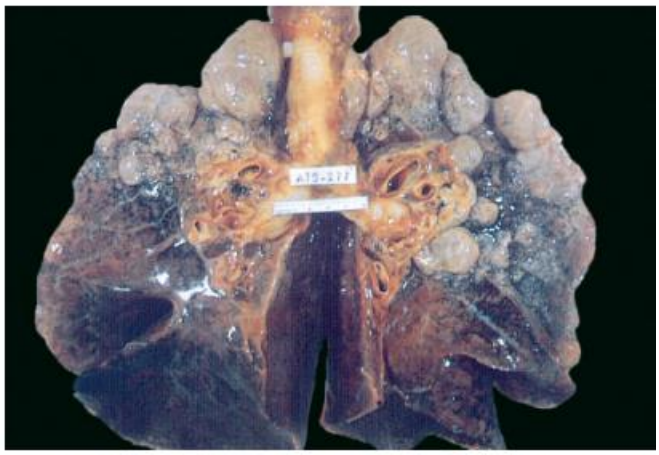

Fig. 13.8 Bullous emphysema with large apical and subpleural bullae. *(From the Teaching Collection of the Department of Pathology, University of Texas Southwestern Medical School, Dallas, Texas.)*

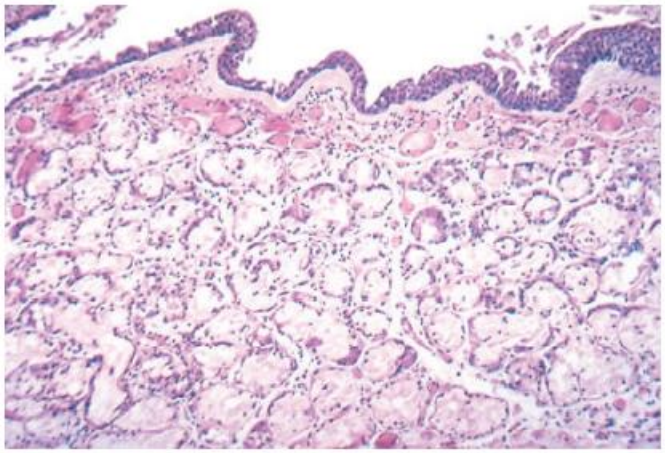

Fig. 13.9 Chronic bronchitis. The lumen of the bronchus is above. Note the marked thickening of the mucous gland layer (approximately twice-normal) and squamous metaplasia of lung epithelium. *(From the Teaching Collection of the Department of Pathology, University of Texas, Southwestern Medical School,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

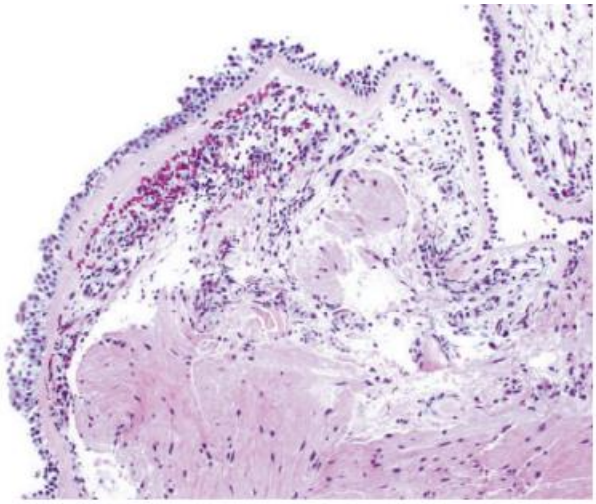

Fig. 13.11 Bronchial biopsy specimen from an asthmatic patient showing sub–basement membrane fibrosis, eosinophilic inflammation, and smooth muscle hyperplasia.

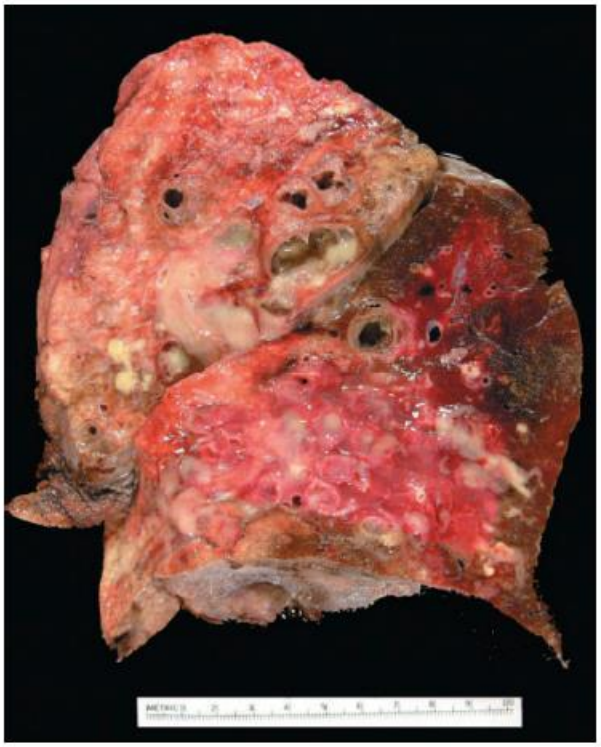

Fig. 13.12 Bronchiectasis in a patient with cystic fibrosis who underwent lung resection for transplantation. Cut surface of lung shows markedly dilated bronchi filled with purulent mucus that extend to subpleural regions.

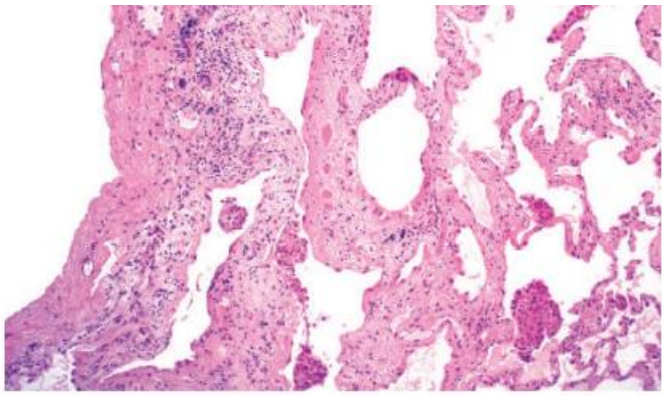

Fig. 13.14 Usual interstitial pneumonia. The fibrosis, which varies in intensity, is more pronounced in the subpleural region.

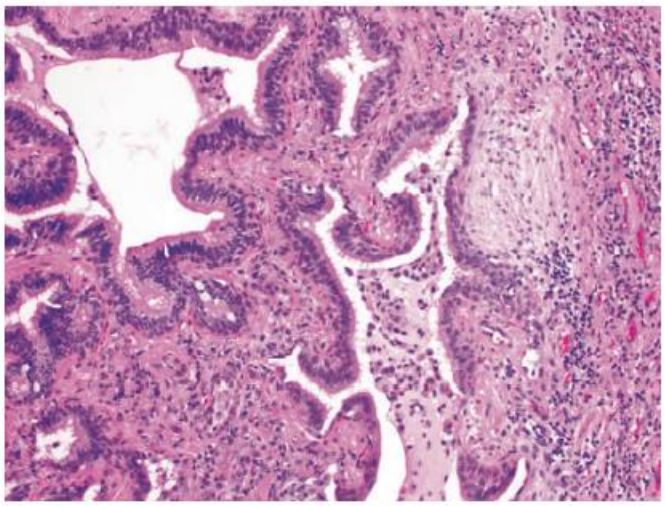

Fig. 13.15 Usual interstitial pneumonia. Fibroblastic focus with fibers running parallel to surface and bluish myxoid extracellular matrix. Honeycombing is present to the left.

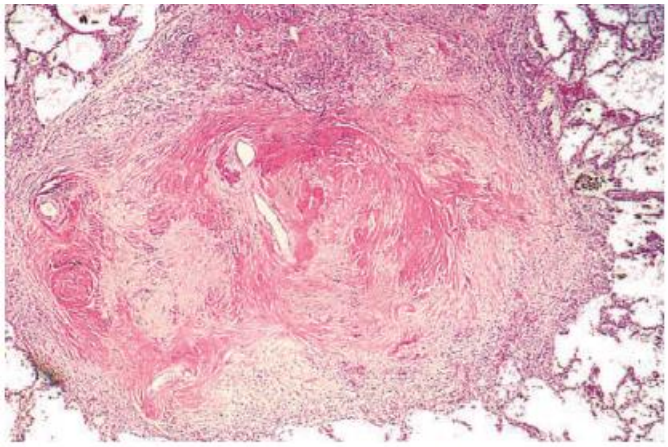

Fig. 13.18 Coalescent collagenous silicotic nodules. *(Courtesy of Dr. John Godleski, Brigham and Women's Hospital, Boston, Massachusetts.)*

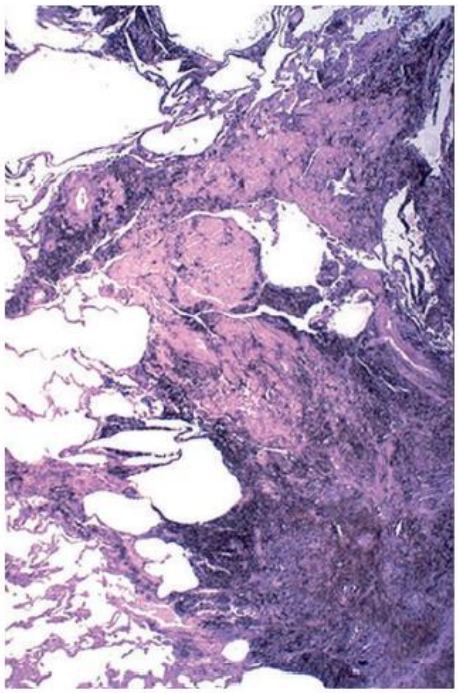

Fig. 13.16 Progressive massive fibrosis in a coal worker. A large amount of black pigment is associated with fibrosis. *(From Klatt EC:* Robbins and Cotran atlas of pathology, *ed 2, Elsevier, Philadelphia, p 121.)*

Fig. 13.17 Advanced silicosis, seen in a transected lung. Scarring has contracted the upper lobe into a small dark mass *(arrow)*. Note the dense pleural thickening. *(Courtesy of Dr. John Godleski, Brigham and Women's Hospital, Boston, Massachusetts.)*

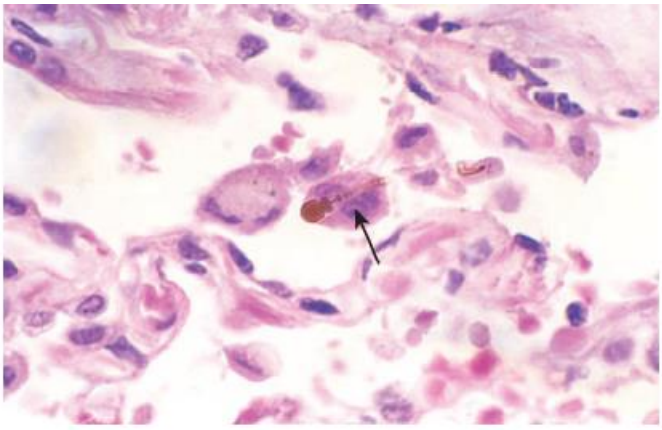

Fig. 13.19 High-power detail of an asbestos body, revealing the typical beading and knobbed ends *(arrow)*.

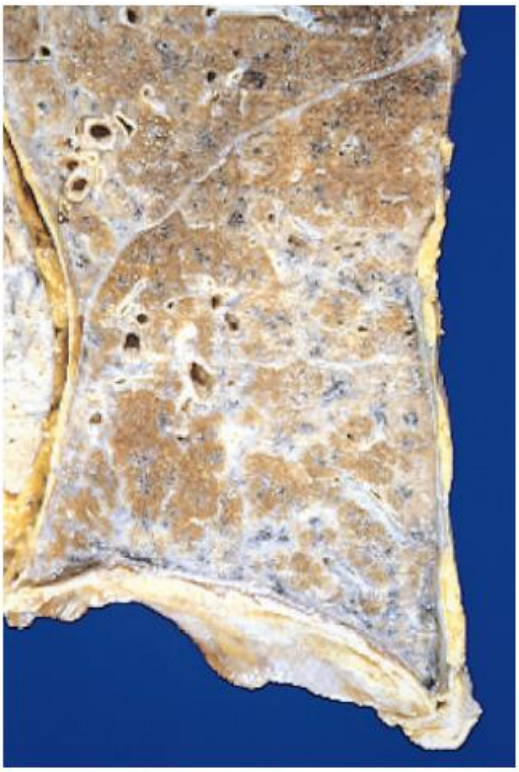

Fig. 13.20 Asbestosis. Markedly thickened visceral pleura covers the lateral and diaphragmatic surface of the lung. Note also severe interstitial fibrosis diffusely affecting the lower lobe of the lung.

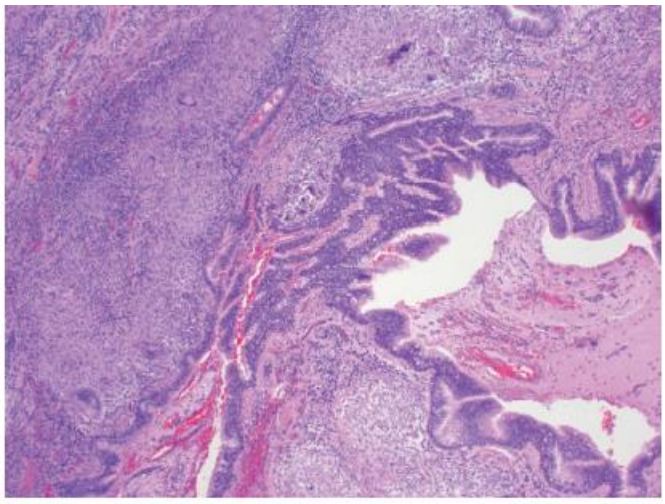

Fig. 13.21 Sarcoid. Characteristic peribronchial noncaseating granulomas with many giant cells are present.

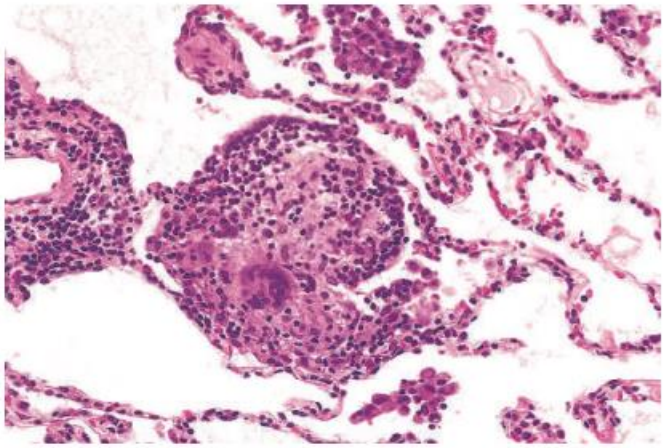

Fig. 13.22 Hypersensitivity pneumonitis. histologic appearance. Loosely formed interstitial granulomas and chronic inflammation are characteristic.

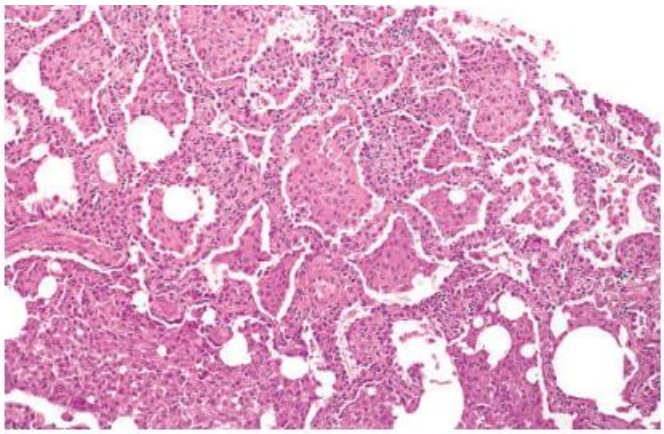

Fig. 13.23 Desquamative interstitial pneumonia. There is accumulation of large numbers of macrophages within the alveolar spaces with only slight fibrous thickening of the alveolar walls.

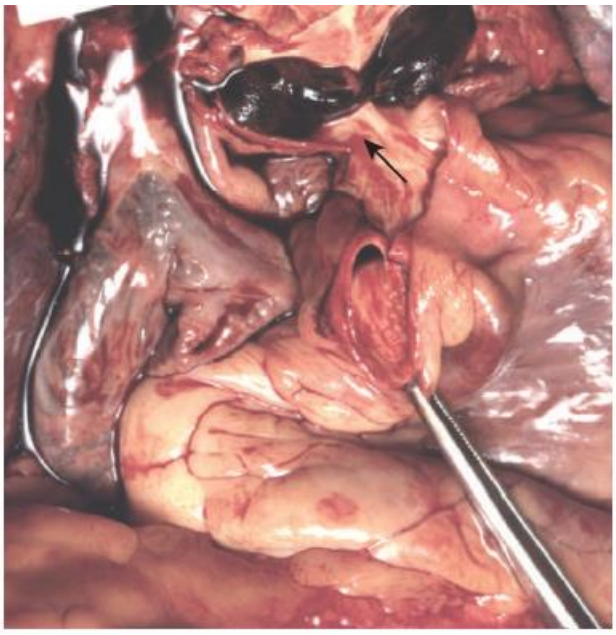

Fig. 13.24 Large saddle embolus from the femoral vein lying astride the main left and right pulmonary arteries. *(Courtesy of Dr. Linda Margraf, Department of Pathology, University of Texas Southwestern Medical School, Dallas, Texas.)*

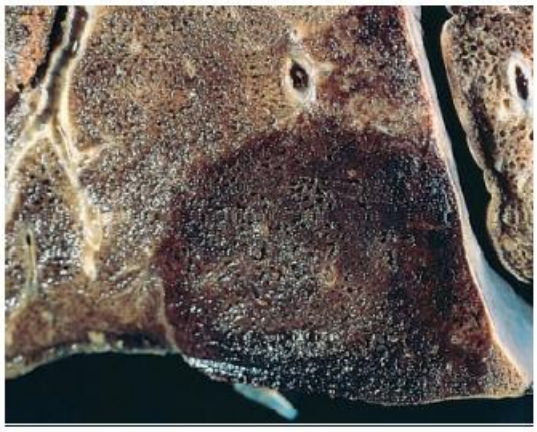

Fig. 13.25 A small, roughly wedge-shaped hemorrhagic pulmonary infarct of recent occurrence.

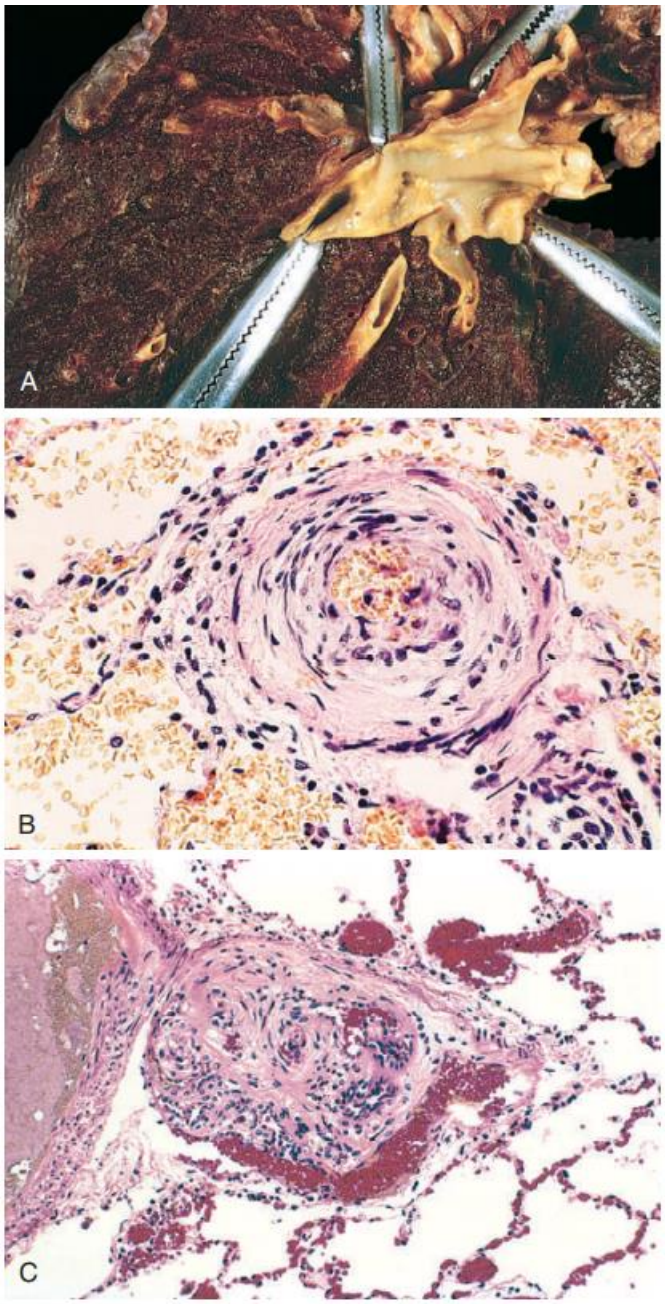

Fig. 13.26 Vascular changes in pulmonary hypertension. (A) Atheroma formation, a change usually limited to large pulmonary arteries. (B) Marked medial hypertrophy. (C) Plexiform lesion characteristic of advanced pulmonary hypertension seen in small arteries.

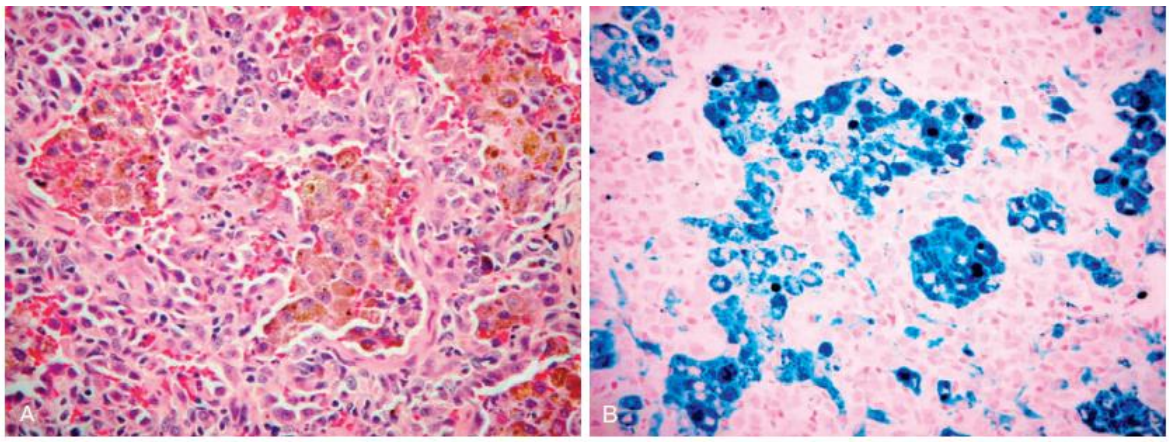

Fig. 13.27 Diffuse alveolar hemorrhage syndrome. (A) Lung biopsy specimen demonstrates large numbers of intraalveolar hemosiderin-laden macrophages on a background of thickened fibrous septa. (B) The tissue has been stained with Prussian blue, an iron stain that highlights the abundant intracellular hemosiderin. *(From the Teaching Collection of the Department of Pathology, Children's Medical Center, Dallas, Texas.)*

Fig. 13.30 Lobar pneumonia with gray hepatization. The lower lobe is uniformly consolidated.

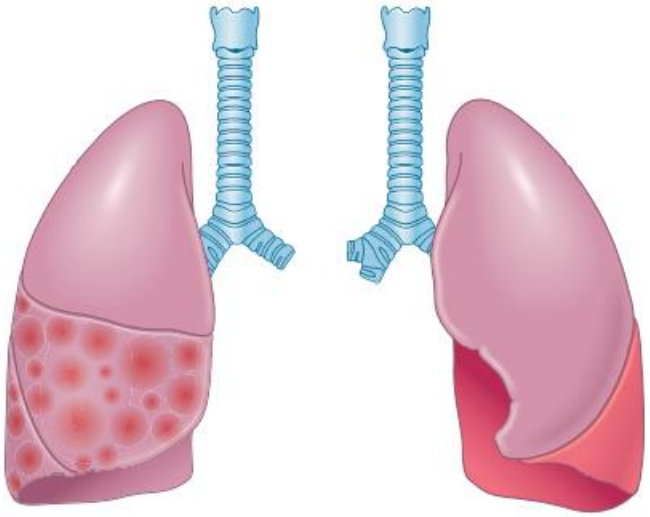

Fig. 13.29 The anatomic distribution of bronchopneumonia and lobar pneumonia affecting the lower lobes of the lung.

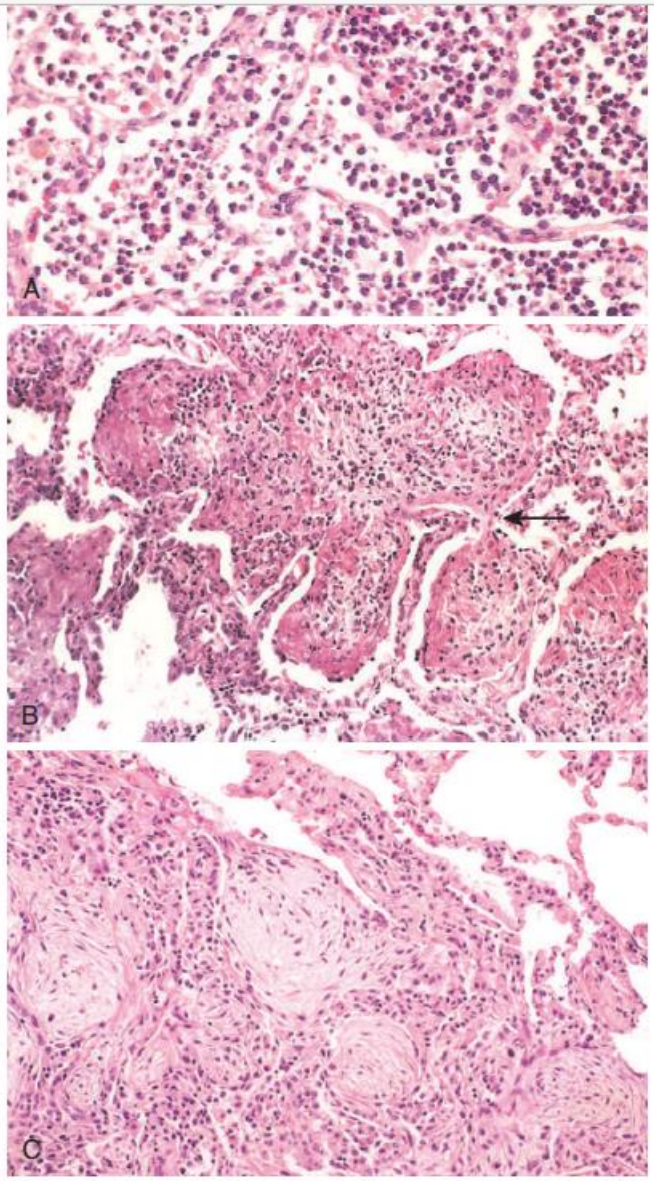

Fig. 13.31 (A) Acute pneumonia. The congested septal capillaries and extensive neutrophil exudation into alveoli correspond to early red hepatization. Fibrin nets have not yet formed. (B) Early organization of intraalveolar exudates, seen in areas to be streaming through the pores of Kohn *(arrow)*. (C) Advanced organizing pneumonia, featuring transformation of exudates to fibromyxoid masses richly infiltrated by macrophages and fibroblasts.

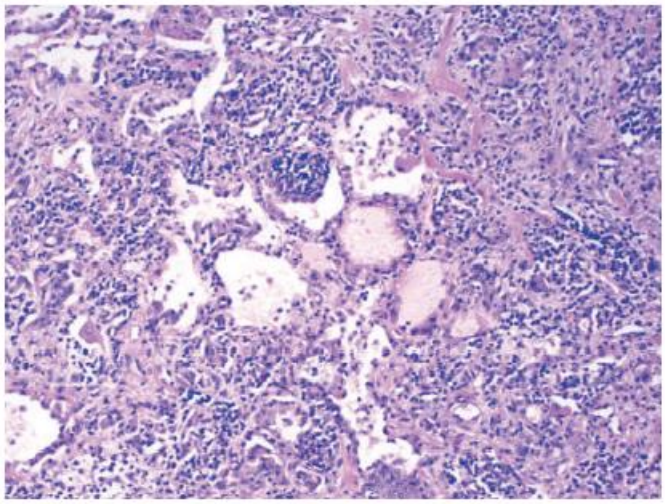

Fig. 13.32 Viral pneumonia. The thickened alveolar walls are infiltrated with lymphocytes and some plasma cells, which are spilling over into alveolar spaces. Note the focal alveolar edema *(center)* and early fibrosis *(upper right)*.

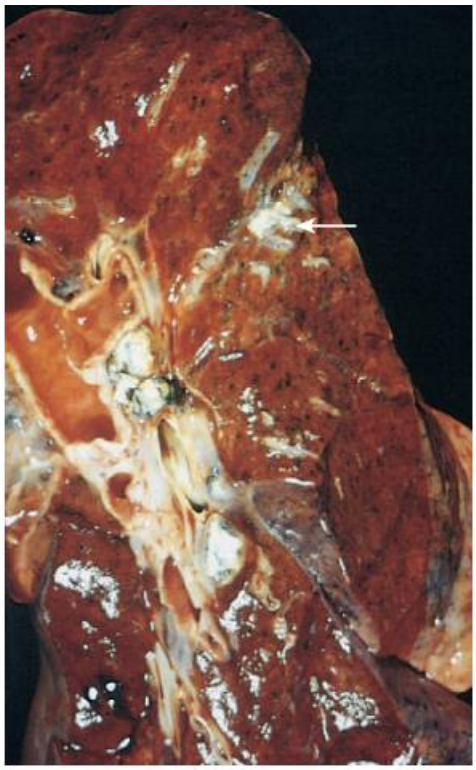

Fig. 13.34 Primary pulmonary tuberculosis, Ghon complex. The gray-white parenchymal focus *(arrow)* is under the pleura in the lower part of the upper lobe. Hilar lymph nodes with caseation are seen *(left)*.

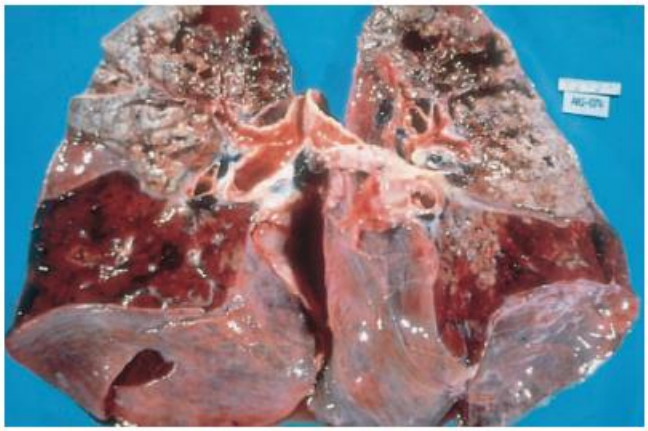

Fig. 13.36 Secondary pulmonary tuberculosis. The upper parts of both lungs are riddled with gray-white areas of caseation and multiple areas of softening and cavitation.

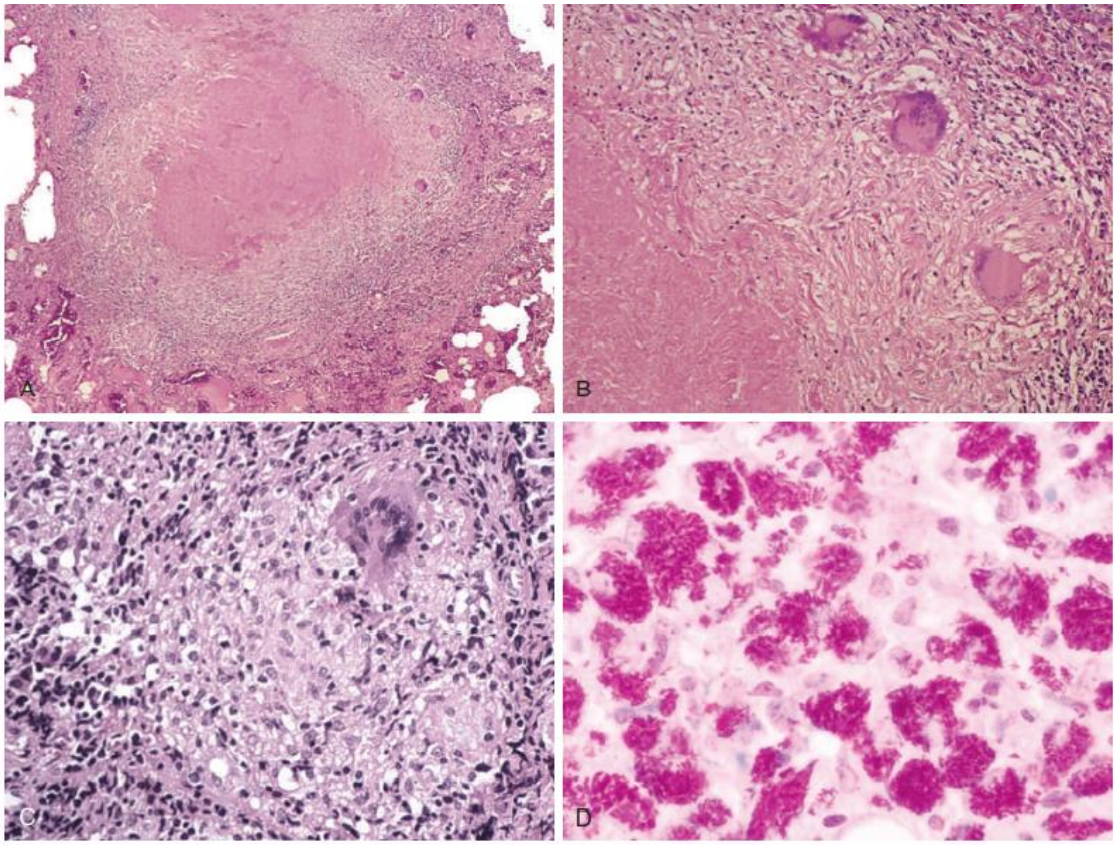

Fig. 13.35 The morphologic spectrum of tuberculosis. A characteristic tubercle at low magnification (A) and at higher power (B) shows central granular caseation surrounded by epithelioid and multinucleate giant cells. This is the usual response in individuals who develop cell-mediated immunity to the organism. (C) Occasionally, even in immunocompetent patients, tubercular granulomas may not show central caseation; hence, irrespective of the presence or absence of caseous necrosis, use of special stains for acid-fast organisms is indicated when granulomas are present. (D) In this specimen from an immunosuppressed patient, sheets of macrophages packed with mycobacteria are seen (acid-fast stain).

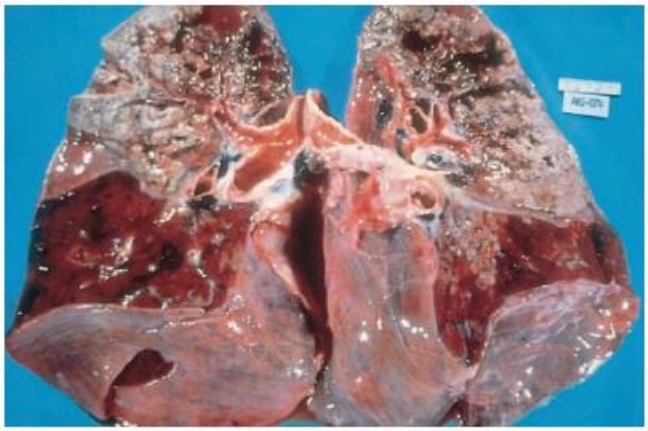

Fig. 13.36 Secondary pulmonary tuberculosis. The upper parts of both lungs are riddled with gray-white areas of caseation and multiple areas of softening and cavitation.

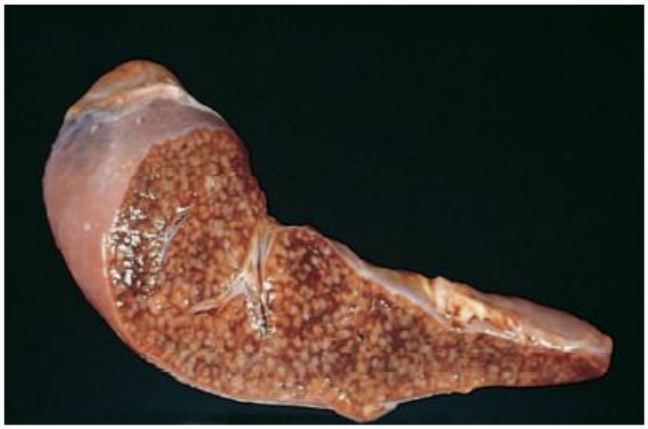

Fig. 13.37 Miliary tuberculosis of the spleen. The cut surface shows numerous gray-white granulomas.

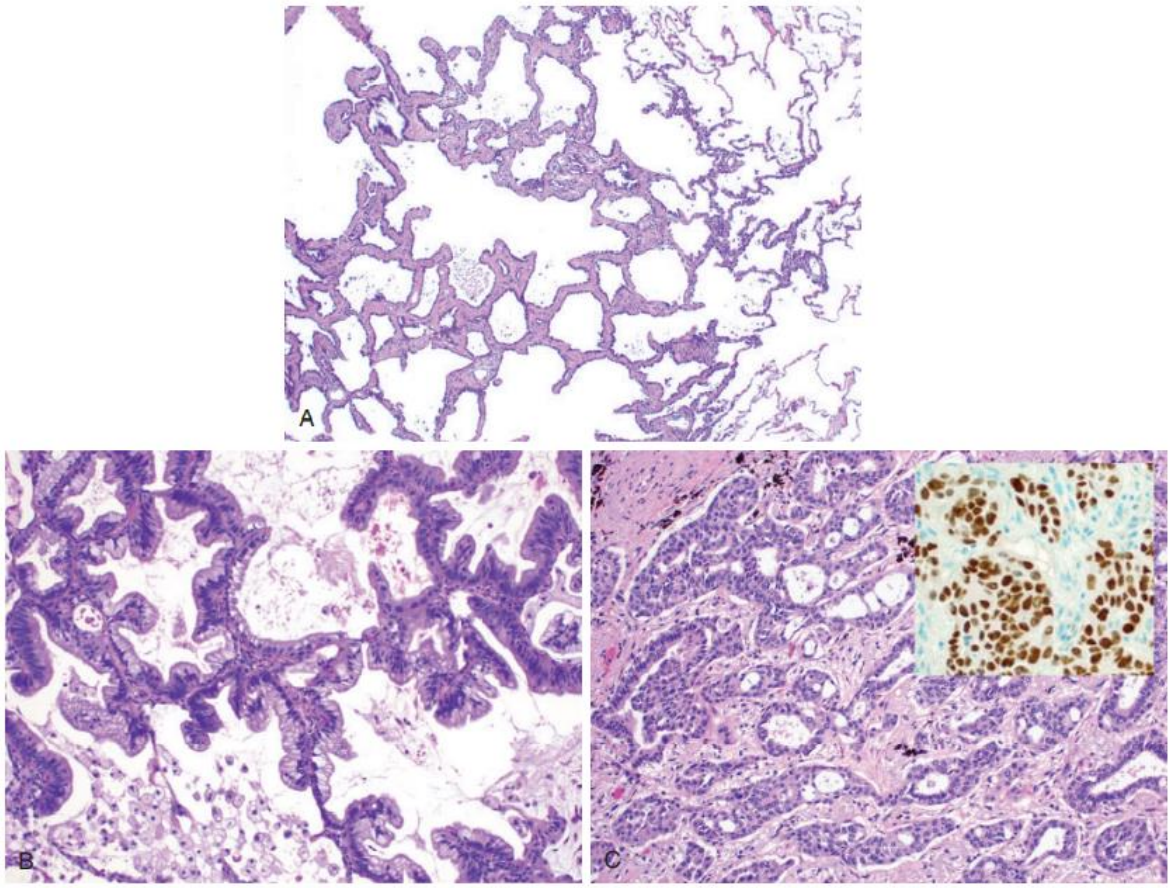

Fig. 13.43 Adenocarcinoma and associated lesions. (A) Atypical adenomatous hyperplasia with cuboidal epithelium and mild interstitial fibrosis. (B) Adenocarcinoma in situ, mucinous subtype, with characteristic growth along preexisting alveolar septa, without invasion. (C) Gland-forming adenocarcinoma; inset shows thyroid transcription factor 1 (TTF-1) positivity, which is seen in a majority of pulmonary adenocarcinomas.

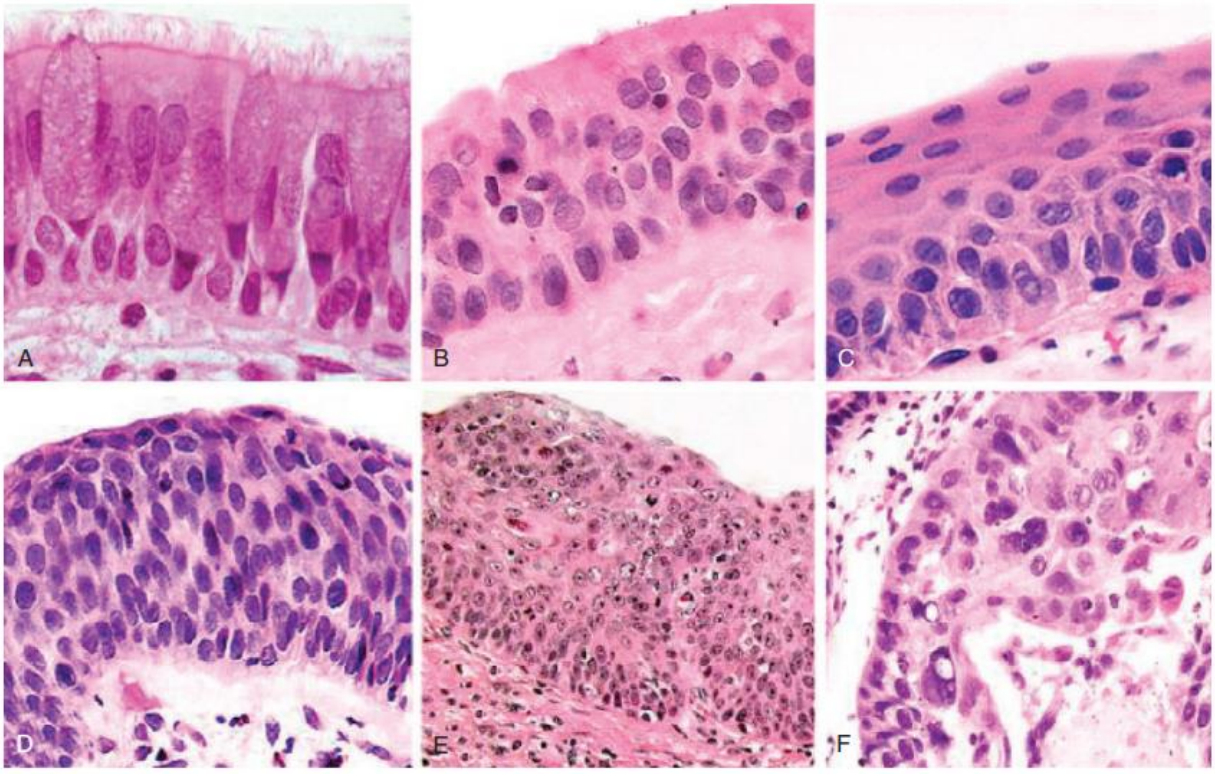

Fig. 13.44 Precursor lesions of squamous cell carcinomas. (A to C) Some of the earliest (and "mild") changes in smoking-damaged respiratory epithelium include goblet cell hyperplasia (A), basal cell (or reserve cell) hyperplasia (B), and squamous metaplasia (C). (D) More ominous changes include the appearance of squamous dysplasia, characterized by the presence of disordered squamous epithelium, with loss of nuclear polarity, nuclear hyperchromasia, pleomorphism, and mitotic figures. (E and F) Squamous dysplasia may, in turn, progress through the stages of mild, moderate, and severe dysplasia. Carcinoma in situ (CIS) (E) is the stage that immediately precedes invasive squamous cell carcinoma (F). Apart from the lack of basement membrane disruption in CIS, the cytologic features of CIS are similar to those in frank carcinoma. *(A to E, Courtesy of Dr. Adi Gazdar, Department of Pathology, University of Texas Southwestern Medical School, Dallas, Texas. F, Reproduced with permission from Travis WD, Colby TV, Corrin B, et al, editors:* World Health Organization histological typing of lung and pleural tumors, *Heidelberg, 1999, Springer.)*

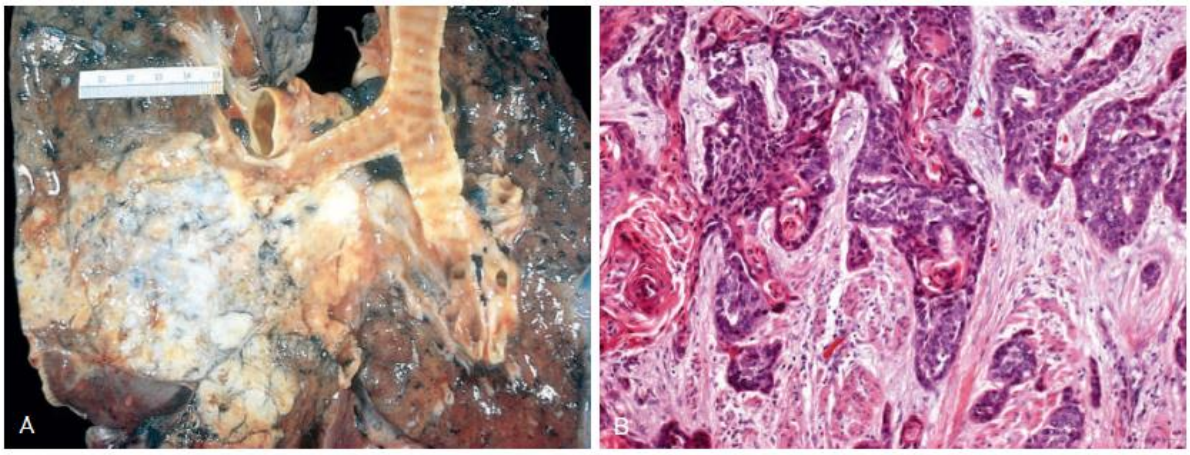

Fig. 13.45 Squamous cell carcinoma. (A) Squamous cell carcinoma appearing as a central (hilar) mass that is invading contiguous parenchyma. (B) Well-differentiated squamous cell carcinoma, showing keratinization and pearls.

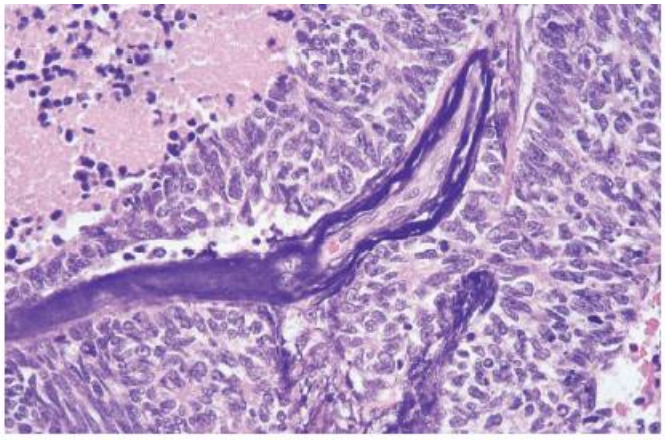

Fig. 13.46 Small cell carcinoma with small deeply basophilic cells and areas of necrosis *(top left)*. Note basophilic staining of vascular walls due to encrustation by DNA from necrotic tumor cells (Azzopardi effect).

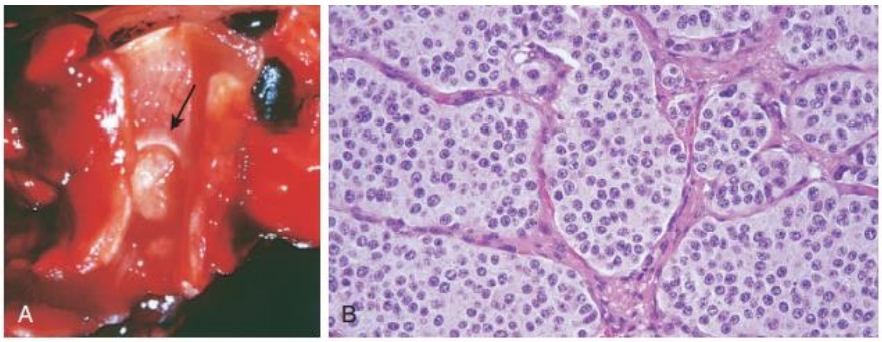

Fig. 13.47 Bronchial carcinoid. (A) Carcinoid growing as a spherical, pale mass *(arrow)* protruding into the lumen of the bronchus. (B) Histologic appearance demonstrating small, rounded, uniform nuclei and moderate cytoplasm. *(Courtesy of Dr. Thomas Krausz, Department of Pathology, University of Chicago Pritzker School of Medicine, Chicago, Illinois.)*