more vertical right main bronchus) and are most often single. Abscesses that develop in the course of pneumonia or bronchiectasis are usually multiple, basal, and diffusely scattered. Septic emboli and pyemic abscesses are multiple and may affect any region of the lungs.

The **cardinal histologic change in all abscesses is suppurative destruction of the lung parenchyma within the central area of cavitation.** The abscess cavity may be filled with suppurative debris or, if there is communication with an air passage, may be partially drained to create an air-containing cavity. Superimposed saprophytic infections are prone to develop within the necrotic debris. Continued infection leads to large, poorly demarcated, fetid, green-black, multilocular cavities designated gangrene of the lung. In chronic cases considerable fibroblastic proliferation produces a fibrous wall.

Clinical Course. The manifestations of pulmonary abscesses are much like those of bronchiectasis and are characterized principally by cough, fever, and copious amounts of foul-smelling purulent or sanguineous sputum. Fever, chest pain, and weight loss are common. Clubbing of the fingers and toes may appear within a few weeks after the onset of an abscess. The diagnosis can be only suspected from the clinical findings and must be confirmed radiologically. Whenever an abscess is discovered in older individuals, it is important to rule out an underlying carcinoma, which is present in 10% to 15% of cases.

The course of abscesses is variable. With antimicrobial therapy, most resolve leaving behind a scar. Complications include extension of the infection into the pleural cavity, hemorrhage, the development of *brain abscesses* or *meningitis* from septic emboli, and (rarely) secondary amyloidosis (type AA).

Chronic Pneumonia

Chronic pneumonia is most often a localized lesion in the immunocompetent patient, with or without regional lymph node involvement. Typically, the inflammatory reaction is granulomatous, and is caused by bacteria (e.g., *M. tuberculosis*) or fungi (e.g., *Histoplasma capsulatum*). Tuberculosis of the lung and other organs was described in Chapter 8. Chronic pneumonias caused by fungi are discussed here.

Histoplasmosis

Histoplasma capsulatum infection is acquired by inhalation of dust particles from soil contaminated with bird or bat droppings that contain small spores (microconidia), the infectious form of the fungus. It is endemic along the Ohio and Mississippi rivers and the Caribbean. It is also found in Mexico, Central and South America, parts of eastern and southern Europe, Africa, eastern Asia and Australia. Like *M. tuberculosis, H. capsulatum* is an intracellular pathogen that is found mainly in phagocytes. The clinical presentations and morphologic lesions of histoplasmosis also strikingly resemble those of tuberculosis, including (1) a self-limited and often latent primary pulmonary involvement, which may result in coin lesions on chest radiography; (2) chronic, progressive, secondary lung disease, which is localized to the lung apices and causes cough, fever, and night sweats; (3) spread to extrapulmonary sites, including mediastinum, adrenals, liver, or meninges; and (4) widely disseminated disease in immunocompromised patients. Histoplasmosis can occur in immunocompetent individuals but as expected it is more severe in those with depressed cell mediated immunity.

The pathogenesis of histoplasmosis is incompletely understood. It is known that macrophages are the major target of infection. *H. capsulatum* may be internalized into macrophages after opsonization with antibody. *Histoplasma* yeasts can multiply within the phagosome, and lyse the host cells. *Histoplasma* infections are controlled by helper T cells that recognize fungal cell wall antigens and heat-shock proteins and subsequently secrete IFN-γ, which activates macrophages to kill intracellular yeasts. In addition, *Histoplasma* induces macrophages to secrete TNF, which recruits and stimulates other macrophages to kill *Histoplasma.*

MORPHOLOGY

In the lungs of otherwise healthy adults, *Histoplasma* infections produce **granulomas**, which usually undergo caseation necrosis and coalesce to produce large areas of consolidation, but may also liquefy to form cavities (particularly in patients with COPD). With spontaneous resolution or effective treatment, these lesions undergo fibrosis and concentric calcification (tree-bark appearance) (Fig. 15-37*A*). Histologic differentiation from tuberculosis, sarcoidosis, and coccidioidomycosis requires identification of the 3- to 5-μm thin-walled yeast forms, which may persist in tissues for years.

In **fulminant disseminated histoplasmosis**, which occurs in immunosuppressed individuals, granulomas do not form; instead, there are focal accumulations of mononuclear phagocytes filled with fungal yeasts throughout the body (Fig. 15-37*B*).

The diagnosis of histoplasmosis is established by culture or identification of the fungus in tissue lesions. In addition, serologic tests for antibodies and antigen are also available. Antigen detection in body fluids is most useful in the early stages, because antibodies are formed 2 to 6 weeks after infection.

Blastomycosis

Blastomyces dermatitidis is a soil-inhabiting dimorphic fungus. It causes disease in the central and southeastern United States; infection also occurs in Canada, Mexico, the Middle East, Africa, and India. There are three clinical forms: *pulmonary blastomycosis, disseminated blastomycosis,* and a rare *primary cutaneous form* that results from direct inoculation of organisms into the skin. Pulmonary blastomycosis most often presents as an abrupt illness with productive cough, headache, chest pain, weight loss, fever, abdominal pain, night sweats, chills, and anorexia. Chest radiographs reveal lobar consolidation, multilobar infiltrates, perihilar infiltrates, multiple nodules, or miliary infiltrates. The upper lobes are most frequently involved. The pneumonia most often resolves spontaneously, but it may persist, or progress to a chronic lesion.