edema suggests pulmonary hypertension. Right ventricular heart failure is usually the result of chronic hypoxemia and is often related to end-stage lung disease.

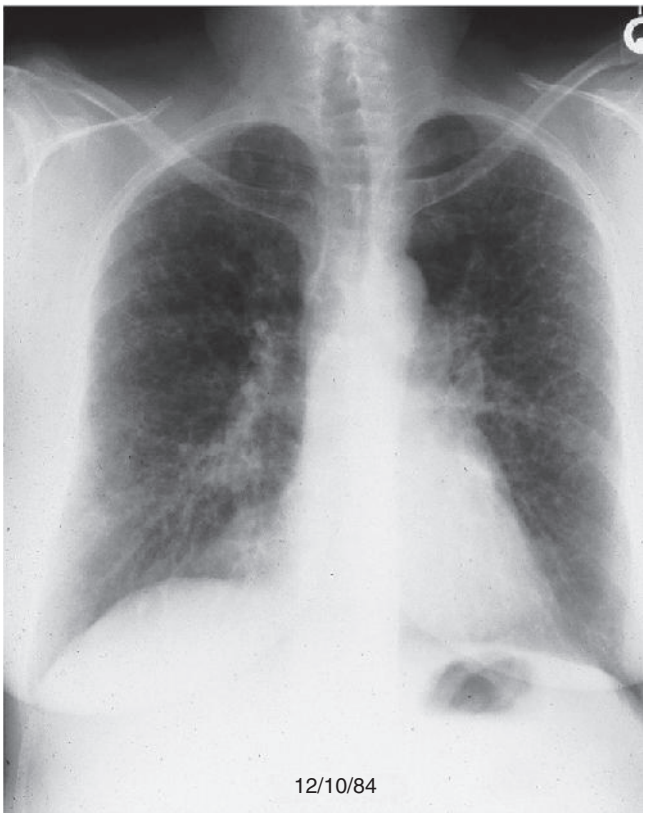
Laboratory studies may be of benefit. For example, eosinophilia suggests the particular group of disorders associated with pulmonary infiltrates and peripheral blood eosinophilia.

A chest radiograph can narrow the diagnosis based on the distribution of the typical reticulonodular changes found in ILD. For example, sarcoidosis, lymphangioleiomyomatosis, silicosis, HP, eosinophilic granuloma, and ankylosing spondylitis most often affect the upper- and mid-level lung fields, whereas IPF, asbestosis, and many connective tissue–related ILDs typically involve the lower-level lung fields.

Disease patterns are best analyzed with the use of HRCT of the chest, a test considered essential in the evaluation of patients thought to have ILD. HRCT can reveal patterns of disease that allow the diagnostic considerations to be significantly narrowed. For example, upper lobe–predominant cystic lung disease on HRCT suggests LCH, sarcoidosis, or lymphangioleiomyomatosis. Lower lobe and peripheral reticular opacities with associated traction bronchiectasis and honeycombing suggest IPF, asbestosis, or certain connective tissue disease–related ILDs. Visualization of abnormalities of the mediastinum or pleura associated with parenchymal lung disease is helpful. Sarcoidosis, for example, typically exhibits hilar and mediastinal lymphadenopathy associated with beadlike septal nodules, whereas pleural plaques associated with lower lobe fibrosis are consistent with asbestosis. Incorporation of HRCT imaging data with clinical history sometimes may be sufficient for diagnosis.

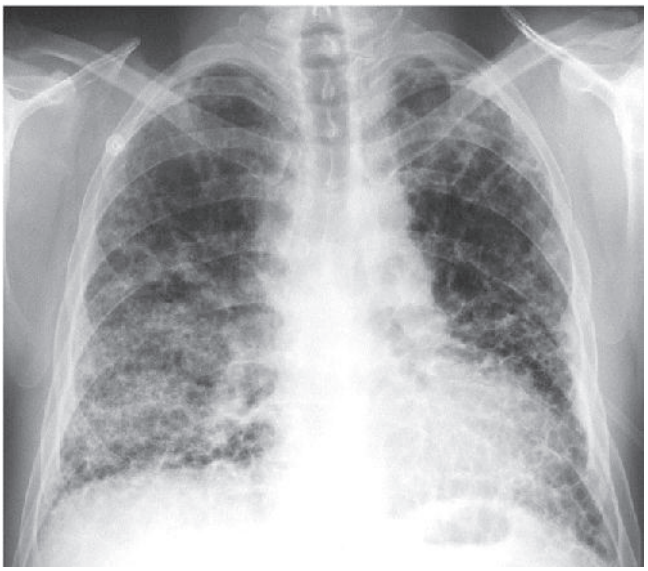
Pulmonary function tests for ILD typically reveal a restrictive pattern characterized by proportionately decreased airflow with a preserved ratio of forced expiratory volume in 1 second to forced vital capacity (FEV_1/FVC) (i.e., no obstruction to airflow) and decreased lung volumes as highlighted by decreased total lung capacity and functional residual capacity. The diffusion capacity of the lung for carbon dioxide (D_{LCO}) is often decreased and may be the earliest change seen in drug-induced ILD or connective tissue disease–related ILD. The restrictive abnormality found in most forms of ILD results from decreased compliance of the lung in the setting of fibrosis.

Histologically, ILD affects the interstitium of the lung, the space located between the basement membrane of the vascular structures in the distal air spaces and the basement membranes of the epithelial cells that line the alveoli (Fig. 17-1). This space extends proximally toward the alveolar ducts and respiratory bronchioles. Normally, the interstitium of the lung contains a few fibroblasts and connective tissue components within a very thin wall that allows efficient diffusion of gases. In ILD, however, this space expands with the accumulation of fibroblasts or other



Preserved lung volumes

Sarcoidosis
Hypersensitivity pneumonitis



Small lung volumes

Idiopathic pulmonary fibrosis
Asbestosis

FIGURE 17-1 Radiographic manifestations of interstitial lung diseases. Well-preserved lung volumes with bilateral interstitial reticulonodular infiltrates *(left)* are seen in diseases similar to sarcoidosis and hypersensitivity pneumonitis. Reduced lung volumes with bilateral basilar infiltrates *(right)* are seen in idiopathic pulmonary fibrosis.