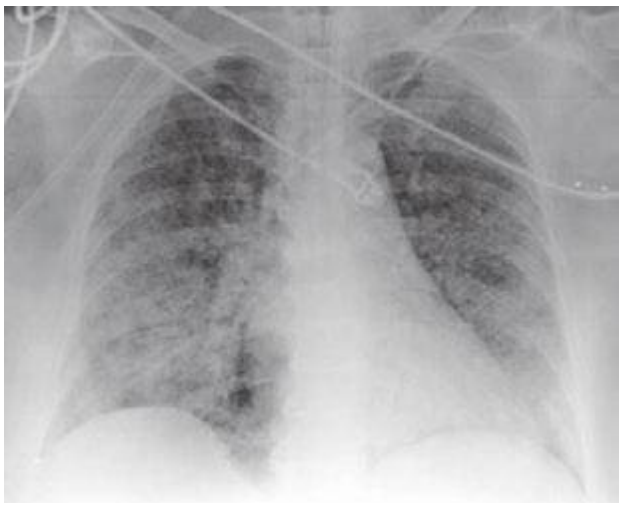

FIGURE 322-2 A representative anteroposterior chest x-ray in the exudative phase of ARDS shows diffuse interstitial and alveolar infiltrates that can be difficult to distinguish from left ventricular failure.

and an inability to get enough air. Tachypnea and increased work of breathing result frequently in respiratory fatigue and ultimately in respiratory failure. Laboratory values are generally nonspecific and are primarily indicative of underlying clinical disorders. The chest radiograph usually reveals alveolar and interstitial opacities involving at least three-quarters of the lung fields (Fig. 322-2). While characteristic for ARDS, these radiographic findings are not specific and can be indistinguishable from cardiogenic pulmonary edema (Chap. 326). Unlike the latter, however, the chest x-ray in ARDS rarely shows cardiomegaly, pleural effusions, or pulmonary vascular redistribution. Chest CT in ARDS reveals extensive heterogeneity of lung involvement (Fig. 322-4).

Because the early features of ARDS are nonspecific, alternative diagnoses must be considered. In the differential diagnosis of ARDS, the most common disorders are cardiogenic pulmonary edema, diffuse pneumonia, and alveolar hemorrhage. Less common diagnoses to consider include acute interstitial lung diseases (e.g., acute interstitial pneumonitis; Chap. 315), acute immunologic injury (e.g., hypersensitivity pneumonitis; Chap. 310), toxin injury (e.g., radiation pneumonitis; Chap. 263), and neurogenic pulmonary edema (Chap. 47e).

Proliferative Phase This phase of ARDS usually lasts from day 7 to day 21. Most patients recover rapidly and are liberated from mechanical

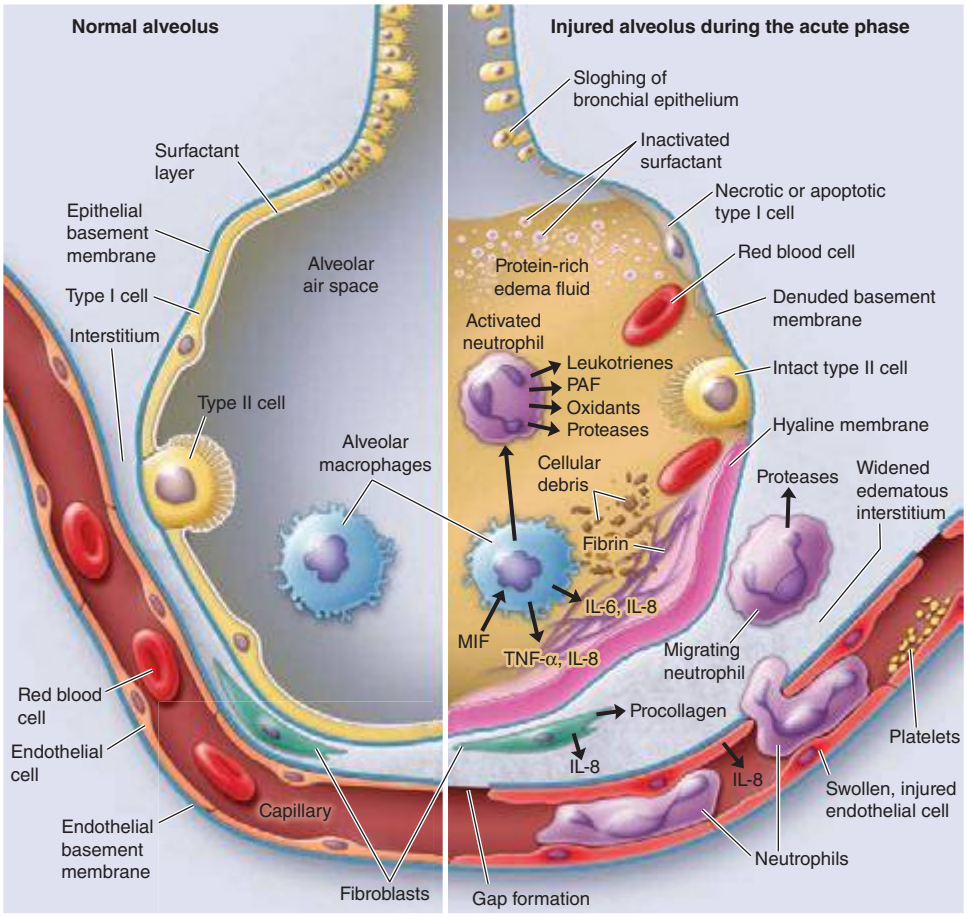

FIGURE 322-3 The normal alveolus (*left*) and the injured alveolus in the acute phase of acute lung injury and the acute respiratory distress syndrome (*right*). In the acute phase of the syndrome (*right*), there is sloughing of both the bronchial and alveolar epithelial cells, with the formation of protein-rich hyaline membranes on the denuded basement membrane. Neutrophils are shown adhering to the injured capillary endothelium and transmigrating through the interstitium into the air space, which is filled with protein-rich edema fluid. In the air space, an alveolar macrophage is secreting cytokines—i.e., interleukins 1, 6, 8, and 10 (IL-1, -6, -8, and -10) and tumor necrosis factor α (TNF-α)—that act locally to stimulate chemotaxis and activate neutrophils. Macrophages also secrete other cytokines, including IL-1, -6, and -10. IL-1 can also stimulate the production of extracellular matrix by fibroblasts. Neutrophils can release oxidants, proteases, leukotrienes, and other proinflammatory molecules, such as platelet-activating factor (PAF). A number of antiinflammatory mediators are also present in the alveolar milieu, including the IL-1-receptor antagonist, soluble TNF-α receptor, autoantibodies to IL-8, and cytokines such as IL-10 and IL-11 (not shown). The influx of protein-rich edema fluid into the alveolus has led to the inactivation of surfactant. MIF, macrophage inhibitory factor. *(From LB Ware, MA Matthay: N Engl J Med 342:1334, 2000, with permission.)*