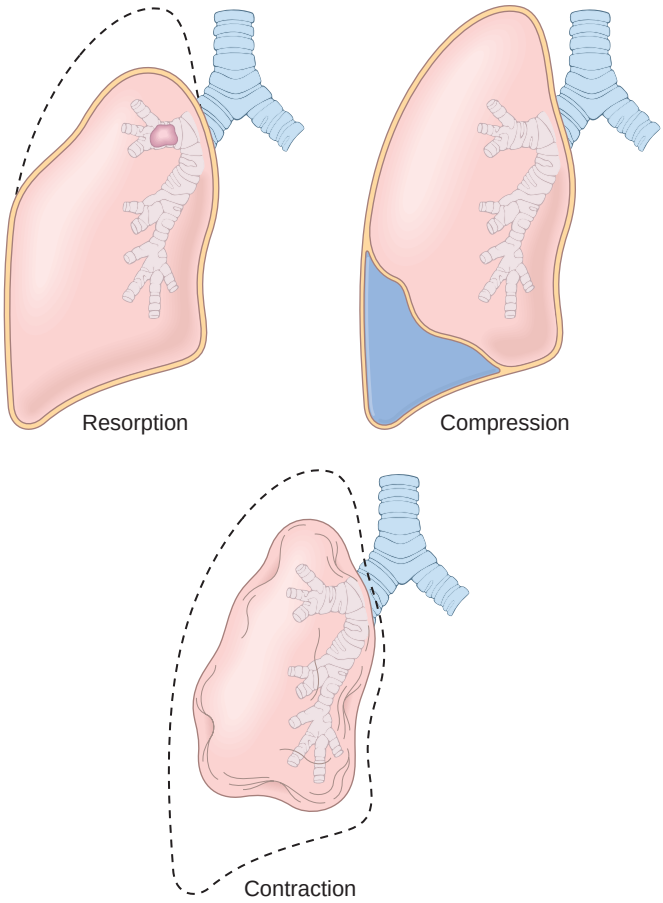

Figure 15-2 Various forms of acquired atelectasis. Dashed lines indicate normal lung volume.

inflated lung, producing areas of relatively airless pulmonary parenchyma. The main types of acquired atelectasis, which is encountered principally in adults, are the following (Fig. 15-2).

- *Resorption atelectasis* stems from complete obstruction of an airway. Over time, air is resorbed from the dependent alveoli, which collapse. Since lung volume is diminished, the mediastinum shifts *toward* the atelectatic lung. Airway obstruction is most often caused by excessive secretions (e.g., mucus plugs) or exudates within smaller bronchi, as may occur in bronchial asthma, chronic bronchitis, bronchiectasis, and postoperative states. Aspiration of foreign bodies and, rarely, fragments of bronchial tumors may also lead to airway obstruction and atelectasis.
- *Compression atelectasis* results whenever significant volumes of fluid (transudate, exudate or blood), tumor, or air (*pneumothorax*) accumulate within the pleural cavity. With compression atelectasis, the mediastinum shifts *away* from the affected lung.
- *Contraction atelectasis* occurs when focal or generalized pulmonary or pleural fibrosis prevents full lung expansion.

Significant atelectasis reduces oxygenation and predisposes to infection. Except in cases caused by contraction, atelectasis is a reversible disorder.

Pulmonary Edema

Pulmonary edema (leakage of excessive interstitial fluid which accumulates in alveolar spaces) can result from hemodynamic disturbances (hemodynamic or cardiogenic pulmonary edema) or from direct increases in capillary permeability, as a result of microvascular injury (Table 15-1). A general consideration of edema is given in Chapter 3, and pulmonary congestion and edema are described briefly in the context of congestive heart failure (Chapter 11). Whatever the clinical setting, pulmonary congestion and edema produce heavy, wet lungs. Therapy and outcome depend on the underlying etiology.

Hemodynamic Pulmonary Edema

Hemodynamic pulmonary edema is due to *increased hydrostatic pressure*, as occurs most commonly in left-sided congestive heart failure. Fluid accumulates initially in the basal regions of the lower lobes because hydrostatic pressure is greatest in these sites (dependent edema). Histologically, the alveolar capillaries are engorged, and an intra-alveolar transudate appears as finely granular pale pink material. Alveolar microhemorrhages and *hemosiderin-laden macrophages ("heart failure" cells)* may be present. In long-standing pulmonary congestion (e.g., as seen in mitral stenosis), hemosiderin-laden macrophages are abundant, and fibrosis and thickening of the alveolar walls cause the soggy lungs to become firm and brown *(brown induration)*. These changes not only impair normal respiratory function but also predispose to infection.

Table 15-1 Classification and Causes of Pulmonary Edema

Hemodynamic Edema
Increased hydrostatic pressure (increased pulmonary venous pressure)
Left-sided heart failure (common)
Volume overload
Pulmonary vein obstruction
Decreased oncotic pressure (less common)
Hypoalbuminemia
Nephrotic syndrome
Liver disease
Protein-losing enteropathies
Lymphatic obstruction (rare)
Edema Due to Alveolar Wall Injury (Microvascular or Epithelial Injury)
Direct Injury
Infections: bacterial pneumonia
Inhaled gases: high concentration oxygen, smoke
Liquid aspiration: gastric contents, near-drowning
Radiation
Indirect Injury
Septicemia
Blood transfusion related
Burns
Drugs and chemicals: chemotherapeutic agents (bleomycin), other medications (methadone, amphotericin B), heroin, cocaine, kerosene, paraquat
Shock, trauma
Edema of Undetermined Origin
High altitude
Neurogenic (central nervous system trauma)