PATHOLOGY

Classic pneumonia evolves through a series of stages. The initial stage is edema with a proteinaceous exudate and often bacteria in the alveoli. Next is a rapid transition to the red hepatization phase. Erythrocytes in the intraalveolar exudate give this stage its name. In the third phase, gray hepatization, no new erythrocytes are extravasating, and those already present have been lysed and degraded. The neutrophil is the predominant cell, fibrin deposition is abundant, and bacteria have disappeared. This phase corresponds with the successful containment of the infection and improvement in gas exchange. In the final phase, resolution, the macrophage reappears as the dominant cell in the alveolar space and the debris of neutrophils, and bacteria and fibrin have been cleared, as has the inflammatory response.

This pattern has been described best for lobar pneumococcal pneumonia but may not apply to pneumonia of all etiologies. In VAP, respiratory bronchiolitis may precede the development of a radiologically apparent infiltrate. A bronchopneumonia pattern is most common in nosocomial pneumonias, whereas a lobar pattern is more common in bacterial CAP. Despite the radiographic appearance, viral and *Pneumocystis* pneumonias represent alveolar rather than interstitial processes.

COMMUNITY-ACQUIRED PNEUMONIA

■ ETIOLOGY

The list of potential etiologic agents of CAP includes bacteria, fungi, viruses, and protozoa. Newer viral pathogens include metapneumoviruses, the coronaviruses responsible for severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS), and the recently discovered coronavirus that originated in Wuhan, China, and is designated SARS-CoV-2. First described in December 2019, SARS-CoV-2 and its associated clinical disease, COVID-19, have reached pandemic proportions and are a cause of significant morbidity and mortality. The virus and the disease are discussed in detail in **Chap. 199**.