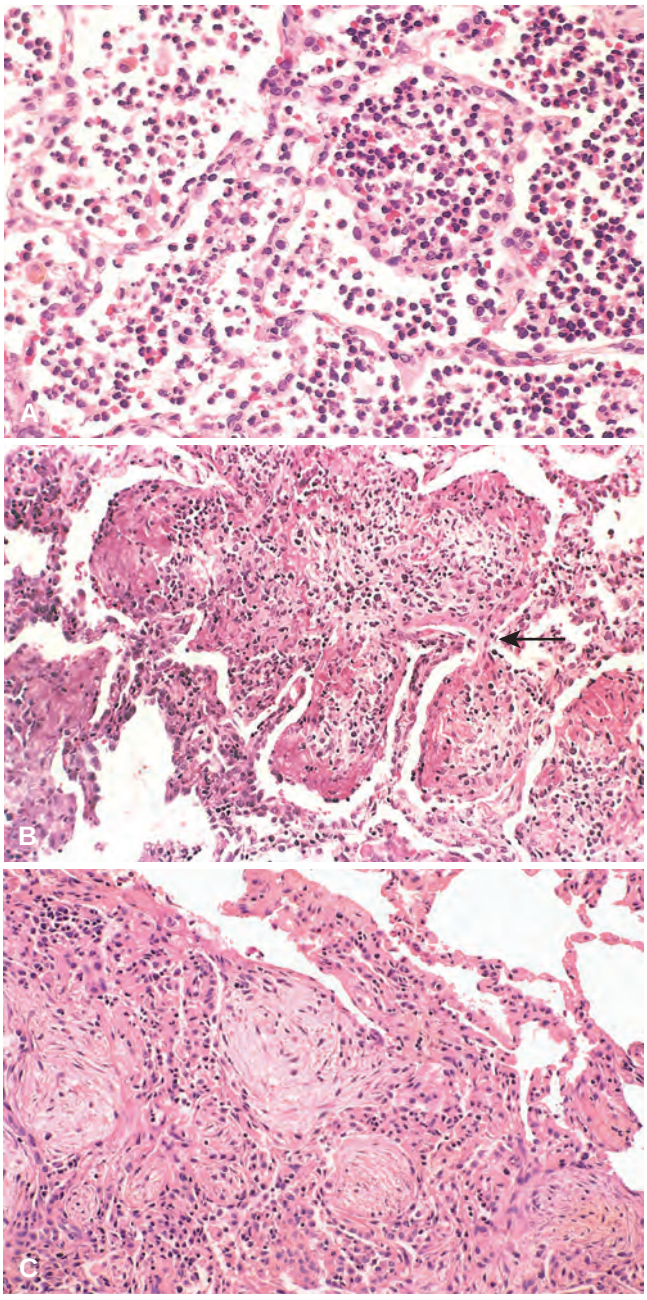

Figure 15-35 Stages of bacterial pneumonia. **A,** Acute pneumonia. The congested septal capillaries and numerous intra-alveolar neutrophils are characteristic of early red hepatization. Fibrin nets have not yet formed. **B,** Early organization of intra-alveolar exudate, seen focally to be streaming through the pores of Kohn *(arrow)*. **C,** Advanced organizing pneumonia. The exudates have been converted to fibromyxoid masses rich in macrophages and fibroblasts.

resulting in a color change to grayish-brown. In the final stage of **resolution** the exudate within the alveolar spaces is broken down by enzymatic digestion to produce granular, semifluid debris that is resorbed, ingested by macrophages, expectorated, or organized by fibroblasts growing into it (Fig. 15-35*C*). Pleural fibrinous reaction to the underlying inflammation, often present in the early stages if the consolidation extends to the surface **(pleuritis)**, may similarly resolve. More often it undergoes organization, leaving fibrous thickening or permanent adhesions.

Foci of **bronchopneumonia** are consolidated areas of acute suppurative inflammation. The consolidation may be confined to one lobe but is more often multilobar and frequently bilateral and basal because of the tendency of secretions to gravitate to the lower lobes. Well-developed lesions are slightly elevated, dry, granular, gray-red to yellow, and poorly delimited at their margins (Fig. 15-33). Histologically, the reaction usually elicits a neutrophil-rich exudate that fills the bronchi, bronchioles, and adjacent alveolar spaces (Fig. 15-35*A*).

Complications of pneumonia include (1) tissue destruction and necrosis, causing **abscess formation** (particularly common with type 3 pneumococci or *Klebsiella* infections); (2) spread of infection to the pleural cavity, causing the intrapleural fibrinosuppurative reaction known as **empyema**; and (3) **bacteremic dissemination** to the heart valves, pericardium, brain, kidneys, spleen, or joints, causing metastatic abscesses, endocarditis, meningitis, or suppurative arthritis.

Clinical Course. The major symptoms of community-acquired acute bacterial pneumonia are abrupt onset of high fever, shaking chills, and cough producting mucopurulent sputum; occasional patients may have hemoptysis. When pleuritis is present it is accompanied by pleuritic pain and pleural friction rub. The whole lobe is radiopaque in lobar pneumonia, whereas there are focal opacities in bronchopneumonia.

The clinical picture is markedly modified by the administration of antibiotics. Treated patients may be relatively afebrile with few clinical signs 48 to 72 hours after the initiation of antibiotics. The identification of the organism and the determination of its antibiotic sensitivity are the keystones to appropriate therapy. Fewer than 10% of patients with pneumonia severe enough to merit hospitalization now succumb, and in most such instances death results from a complication, such as empyema, meningitis, endocarditis, or pericarditis, or to some predisposing influence, such as debility or chronic alcoholism.

Community-Acquired Viral Pneumonia

Common viral infections include influenza virus types A and B, the respiratory syncytial viruses, human metapneumovirus, adenovirus, rhinoviruses, rubeola, and varicella viruses. Any of these agents can cause merely an upper respiratory tract infection, recognized as the common cold, or a more severe lower respiratory tract infection. Factors that favor such extension of the infection include extremes of age, malnutrition, alcoholism, and underlying debilitating illnesses.

Although the molecular details vary, all of the viruses that cause pneuomia produce disease through similar general mechanisms. These viruses have tropisms that allow them to attach to and enter respiratory lining cells. Viral replication and gene expression leads to cytopathic changes, inducing cell death and secondary inflammation. The resulting damage and impairment of local pulmonary defenses, such as mucociliary clearance, may predisopose to bacterial superinfections, which are often more serious than the viral infection itself.