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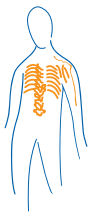

Infections of the Lower Respiratory Tract

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DEFINITION AND EPIDEMIOLOGY

Pneumonia, which is inflammation of the lung parenchyma, is usually caused by an acute infection. When the disease onset occurs outside of the hospital, it is referred to as *community-acquired* pneumonia. It ranges in severity from a mild, self-limited disease to one that is fatal. Community-acquired pneumonia is common, and most patients with pneumonia are treated in the outpatient setting.

Pneumonia is one of the most common reasons for hospitalization among all age groups and accounts for approximately 1 million hospitalizations per year. Each year, approximately 50,000 people in the United States die of influenza and pneumonia. Influenza or pneumonia is the leading cause of death due to infection and the ninth most common cause of death overall.

Numerous microorganisms cause pneumonia, including bacteria, viruses, mycobacteria, and fungi. These agents range from microorganisms that are part of the normal flora to exogenous microorganisms that are inhaled. Noninfectious diseases can mimic pneumonia. The incidence of pneumonia is lowest during early adulthood and increases with each decade of life (Fig. 92-1).

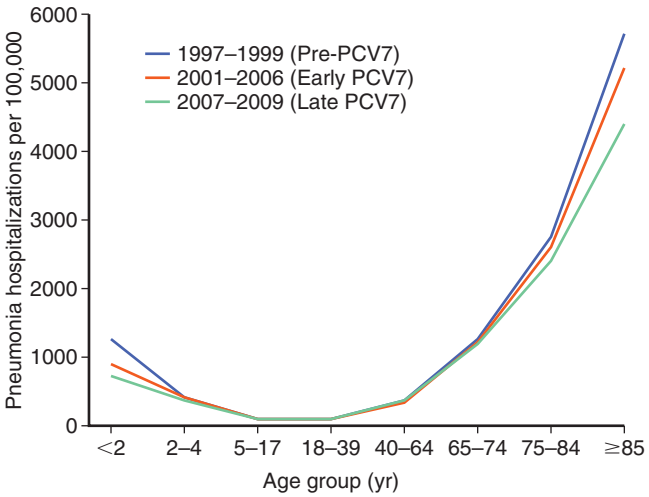

FIGURE 92-1 Rate of hospitalization for pneumonia by age group. PCV7, 7-valent pneumococcal conjugate vaccine. (From Griffin MR, Zhu Y, Moore MR, et al: U.S. hospitalizations for pneumonia after a decade of pneumococcal vaccination, N Engl J Med 369:155–163, 2013.)

PATHOLOGY

Bacterial pneumonia usually causes lobar pneumonia, which is consolidation of an entire lobe or a large portion of a lobe, or bronchopneumonia, which is patchy consolidation of the lung. Pneumococcal lobar pneumonia has four stages of the inflammatory response: consolidation, red hepatization, gray hepatization, and resolution. The initial congestion is characterized by fluid, with some neutrophils and bacteria, filling the alveoli. Red hepatization is characterized by red blood cells along with numerous neutrophils and fibrin filling the alveoli. With gray hepatization, there is breakdown of red blood cells and persistence of fibrin and neutrophils. The consolidated exudate within the alveolar spaces then undergoes resolution.

Pathophysiology

The lower respiratory tract is virtually sterile. Normal host defenses that protect against pneumonia include mucus production and cilia; in combination, they form the mucociliary escalator.

Impairment of host defenses leads to increased risk of pneumonia. Loss or suppression of the cough reflex due to stroke and other neurologic diseases, drugs, and alcohol increases the risk of developing pneumonia, as do aging and associated medical illnesses.

Environmental factors such as smoking and respiratory irritants impair ciliary function and increase the risk of developing pneumonia. Mechanical obstruction of an airway by a tumor or foreign body leads to decreased clearance of microorganisms and may produce postobstructive pneumonia. In addition to mechanical clearance, innate host defenses such as phagocytes and antibodies are essential after microorganisms reach the alveoli. Alveolar macrophages and other components of innate immunity are the first line of defense. Subsequently, opsonizing antibodies and neutrophils play an essential role. Impairment of these host defenses (e.g., alveolar macrophages by silica exposure, neutrophils by chemotherapy, antibodies by hypogammaglobulinemia) increases susceptibility to pneumonia. Those infected with human immunodeficiency virus (HIV) are at increased risk for pneumococcal pneumonia.

The two main mechanisms of entry of microorganism into the lung are microaspiration of organisms that colonize the upper respiratory tract and inhalation of airborne particles that contain a pathogenic microorganism. When a sufficient inoculum enters