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ROSEN'S Emergency Medicine

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Volume 1

10th Edition

ROSEN'S Emergency Medicine

Concepts and Clinical Practice

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Philadelphia, PA 19103-2899

ROSEN'S EMERGENCY MEDICINE: CONCEPTS AND CLINICAL PRACTICE, TENTH EDITION
ISBN: 978-0-323-75789-8
VOLUME 1
ISBN: 978-0-323-75847-5
VOLUME 2
ISBN: 978-0-323-75848-2

Senior Content Strategist: Kayla Wolfe
Content Development Specialist: Kristen Helm
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Printed in Canada

Last digit is the print number: 9 8 7 6 5 4 3 2 1

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Pneumonia

Matthew A. Waxman and Gregory J. Moran

KEY CONCEPTS

- Empirical antimicrobial therapy should be started in the emergency department (ED) for patients admitted with pneumonia.
- *Streptococcus pneumoniae* is the most commonly encountered pathogen in hospitalized patients, especially those requiring the intensive care unit.
- No characteristic radiographic pattern is pathognomonic for a specific pneumonia pathogen.
- *Legionella* should be suspected in patients with gastrointestinal or neurologic symptoms presenting with pneumonia.
- As part of the evaluation of patients with pneumonia, the patient's immune status should be considered. Patients with HIV and other immunosuppressive conditions are at risk for opportunistic infections, such as *Pneumocystis jiroveci.*
- Empirical therapy should treat the most likely pathogens for the clinical situation, such as *S. pneumoniae, Haemophilus influenzae, Mycoplasma pneumoniae,* and *Chlamydia pneumoniae,* and should be consistent with current national treatment guidelines, such as those from the American Thoracic Society/Infectious Disease Society of America (ATS/IDSA).
- Community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA) is an uncommon cause of community-acquired pneumonia (CAP), but empirical coverage of MRSA should be strongly considered for patients with severe pneumonia and sepsis, with concomitant influenza, contact with someone infected with MRSA, or radiographic evidence of necrotizing pneumonia.
- Patients with prior use of intravenous antibiotics, neutropenia, or underlying bronchiectasis are at increased risk of infection with *Pseudomonas aeruginosa.* Empirical therapy for high-risk or critically ill patients should cover for *P. aeruginosa.*
- Disposition is dictated by the patient's underlying medical conditions, severity of illness, likelihood of clinical deterioration, and feasibility of home care and outpatient follow-up.

PRINCIPLES

Background and Importance

Pneumonia is the leading infectious cause of death worldwide, with over 3.1 million deaths annually. In the United States, there are over 4 million adult cases of community-acquired pneumonia (CAP) annually. The economic burden associated with CAP annually in the United States is over 17 billion dollars. Most cases of CAP are managed in the outpatient setting, and the mortality is low. Pneumonia necessitating hospitalization is associated with a mortality rate as high as 20%. Pneumonia remains challenging because of an expanding spectrum of pathogens including SARS-CoV-2, changing antibiotic resistance patterns, continued introduction of newer antimicrobial agents, and increasing emphasis on cost-effectiveness and outpatient management.

As the percentage of the population older than 65 years continues to increase, the incidence of pneumonia is expected to increase. An increasing number of patients are taking immunosuppressive drugs related to the treatment of malignancy, transplantation, or autoimmune disease, resulting in more cases of pneumonia from opportunistic pathogens. *Streptococcus pneumoniae* is the most frequently identified pathogen and is also associated with increasing antimicrobial resistance. In addition, the threat exists of respiratory infections caused by biologic terrorism or newly recognized pathogens such as COVID-19 that have the potential to spread globally through international travel.

Anatomy and Physiology

Despite the constant presence of potential pathogens in the respiratory tract, the lungs are remarkably resistant to infection. The alveolar surface of the lungs covers an area of approximately 140 m^2, about 10,000 L of air passes through the respiratory tract each day, and typical ambient air can contain hundreds to thousands of microorganisms per cubic meter. Although the cough and laryngeal reflexes prevent most large particulate matter from entering the lower respiratory tract, aspiration of oropharyngeal contents may be a common occurrence during normal sleep. Despite these hazards, the lower airway tract is a virtually sterile environment.

Pathophysiology

The development of clinical pneumonia requires a defect in host defenses, presence of a particularly virulent organism, or introduction of a large inoculum of organisms. Pneumonia commonly results from microaspiration of upper respiratory pathogens into the sterile lower respiratory tract. If the challenge of invading organisms overwhelms host defenses, microbial proliferation leads to inflammation, an immune response, and clinical pneumonia. If host defenses are weak, a minimal challenge may lead to the development of pneumonia. The challenge with pneumonia is identifying the causative agent rather than making the diagnosis. A careful history, including foreign travel, recent antibiotic use, and exposure to the health care system, such as dialysis or living in a nursing home, can help inform empiric therapy. Empiric therapy should be chosen with activity against the spectrum of likely pathogens based on the patient's overall clinical presentation.

In the emergency department (ED), it is often difficult to determine the specific cause of pneumonia because routine microbiologic and serologic testing is not available in the time frame of ED evaluation, although rapid polymerase chain reaction (PCR) testing to determine the pathogen is increasingly available. Even in hospitalized patients, the specific microbiologic cause of pneumonia is usually not identified. When identified, *S. pneumoniae* is the most commonly encountered pathogen in hospitalized patients, with *Haemophilus influenzae* a distant second.[1,2] *Legionella, Mycoplasma,* and *Chlamydophila* spp., referred to as atypical pathogens, are also prevalent in hospitalized

patients. Improved molecular testing has shown that viral pathogens such as rhinoviruses, influenza, parainfluenza, and adenoviruses account for up to one-quarter of etiologies in hospitalized patients.[3,4]

Among adults requiring intensive care unit (ICU) admission, *S. pneumoniae* is the most common pathogen, with even higher prevalence among fatal cases. *Legionella* spp., *Staphylococcus aureus* (including methicillin-resistant *S. aureus* [MRSA]), and aerobic gram-negative bacilli also appear to be relatively more common among adults with severe CAP.[4] Atypical organisms, such as *Mycoplasma* species or viruses, account for a higher proportion of pneumonia in patients with milder illness amenable to outpatient therapy. Atypical organisms can also occur with significant frequency in patients with severe illness requiring hospitalization, particularly because of *Legionella* infection. Coinfection, such as with *Chlamydophila pneumoniae* and *S. pneumoniae,* is also well recognized.

S. pneumoniae is a gram-positive coccus that colonizes the nasopharynx in 40% of healthy adults. Although this organism can cause pneumonia in healthy people, patients with a history of diabetes, cardiovascular disease, alcoholism, sickle cell disease, splenectomy, and malignancy or other immunosuppressive illness are at increased risk. A vaccine containing the capsular polysaccharides of 23 pneumococcal types most commonly associated with pneumonia reduces the likelihood of serious pneumococcal infection. It is recommended for adults at increased risk because of underlying illness or age older than 65 years and others who smoke or have comorbidities such as chronic lung disease.[5] Despite this recommendation, many ED patients have not received the pneumococcal vaccine, and vaccinating eligible patients in this setting seems to be feasible and effective. A 13-valent protein-conjugate pneumococcal vaccine effectively reduces invasive pneumococcal disease and pneumonia in infants and young children. Although underutilized in the adult population, the vaccine has resulted in a marked decrease in the incidence of pneumococcal pneumonia.[6]

H. influenzae, the second most frequently isolated organism in CAP among adults, is a pleomorphic gram-negative rod. It is a common pathogen in adults with chronic obstructive pulmonary disease (COPD), alcoholism, malnutrition, malignancy, or diabetes.

S. aureus may be emerging as a more common cause of CAP and has been found more frequently than *H. influenzae* in some series. Community-associated strains of methicillin-resistant *S. aureus* (CA-MRSA) are uncommon in CAP but are more likely to cause severe disease. Often associated with influenza, staphylococcal pneumonias are often necrotizing, with cavitation and pneumatocele formation. Intravenous (IV) drug users may develop hematogenous spread of *S. aureus* that involves both lungs, with multiple small infiltrates or abscesses (e.g., tricuspid endocarditis resulting in septic pulmonary emboli).

Klebsiella pneumoniae is a gram-negative rod that rarely causes disease in a normal host and accounts for a small percentage of cases of CAP. It may cause severe pneumonia in debilitated patients with alcoholism, diabetes, or other chronic illness. There is a high incidence of antibiotic resistance because the organism is often hospital-acquired.

Mycoplasma pneumoniae is one of the most common causes of CAP in previously healthy patients younger than 40 years. Another important organism in CAP is *C. pneumoniae,* an intracellular parasite transmitted between humans by respiratory secretions or aerosols. Seroprevalence studies have indicated that virtually everyone is infected with *C. pneumoniae* at some time and that reinfection is common, particularly in older adults. It accounts for at least 10% of outpatient CAP cases, although this is underestimated due to difficulty in diagnosing infection with this organism.

At least 30 species of *Legionella* have been isolated since the 1976 convention-related outbreak in Philadelphia, from which the organism derives its name. *Legionella* is an intracellular organism that lives in aquatic or soil environments. There is no person-to-person transmission. Although it is implicated in point outbreaks related to cooling towers and similar aquatic sources, the organism also lives in ordinary tap water and is underdiagnosed as a cause of CAP. *Legionella* prevalence seems to vary greatly by geographic region with a high prevalence in Australia.

Lower respiratory infections caused by anaerobic organisms generally result from the aspiration of oropharyngeal contents with large amounts of bacteria. These infections are typically polymicrobial with oral flora such as *Peptostreptococcus, Bacteroides, Fusobacterium,* and *Prevotella* spp. Presentation is often subacute or chronic and may be difficult to distinguish clinically from other causes of pneumonia. Clinical factors that suggest an anaerobic infection include risk factors for aspiration, such as central nervous system depression or swallowing dysfunction, severe periodontal disease, fetid sputum, and presence of a pulmonary abscess or empyema.

Viral pneumonias are common in infants and young children and are recognized as an important cause of pneumonia in adults. Respiratory syncytial virus and parainfluenza viruses are the most common causes of pneumonia in infants and small children, occurring mostly during autumn and winter. Influenza viruses are historically the most common cause of viral pneumonia in adults. Winter influenza outbreaks, usually of influenza type A, may cause up to 40,000 deaths annually in the United States. More than 90% occur in people aged 65 years or older. Updated influenza epidemiology is available from the United States Centers for Disease Control (CDC).[7] Metapneumovirus is a paramyxovirus that is an important cause of viral pneumonia in children and adults. SARS-CoV2 is now the most common viral pneumonia requiring hospitalization. During epidemics, clinicians should suspect a viral agent in patients with hypoxia, fever, and cough.

Fungal infections caused by organisms such as *Histoplasma capsulatum, Blastomyces dermatitidis,* and *Coccidioides immitis* commonly manifest as pulmonary disease. These organisms are present in the soil in various US geographic areas—*H. capsulatum* in the Mississippi and Ohio River valleys, *C. immitis* in desert areas of the Southwest, and *B. dermatitidis* in a poorly defined area extending beyond that of *H. capsulatum*. These infections should be considered in people in appropriate geographic areas, especially in those who are near activities that disturb the soil, such as construction or dirt bike riding, and in patients who do not respond to antibacterial antibiotics. The clinical presentation varies from an acute or chronic pneumonia to asymptomatic granulomas and hilar adenopathy.

Pneumocystis pneumonia (PCP) occurs in immunocompromised hosts, principally those with acquired immunodeficiency syndrome (AIDS) or malignancy. *Pneumocystis jiroveci* is one of the most common opportunistic infections leading to a diagnosis of HIV infection (see Chapter 121). Patients with pulmonary complaints should be questioned about HIV risk factors, and emergency clinicians should search for signs of HIV-related immunosuppression, such as weight loss, lymphadenopathy, and oral thrush. PCP typically manifests subacutely with fatigue, exertional dyspnea, nonproductive cough, pleuritic chest pain, and fever.

Mycobacterium tuberculosis is a slow-growing bacterium transmitted between people by droplet nuclei produced from coughing and sneezing. *M. tuberculosis* survives within macrophages as a facultative intracellular parasite and may remain dormant in the body for many years. Active tuberculosis (TB) develops within 2 years of infection in approximately 5% of patients, and another 5% develop reactivation disease at some later time. Reactivation is more likely to occur in people with impaired cell-mediated immunity, such as patients with diabetes, renal failure, immunosuppressive therapy, malnutrition, or HIV. Approximately one-third of the world's population is infected with

M. tuberculosis. About 9 million new cases of active disease develop annually, resulting in 1.5 million deaths worldwide. Approximately 2.7 per 100,000 individuals in the United States develop TB each year.[8] Multidrug-resistant strains of *M. tuberculosis* have been found in increasing numbers, especially among patients with HIV and in immigrants from Southeast Asia.

Clinical Features

The ED evaluation should focus on establishing the diagnosis of pneumonia and determining the presence of epidemiologic and clinical features that would influence decisions regarding hospitalization and antibiotics. Fundamental components of the history include character of symptoms, setting in which the pneumonia is acquired, geographic or animal exposures, and host factors that predispose to certain types of infections.

Pneumonia generally manifests as a cough productive of purulent sputum, shortness of breath, and fever. In most healthy older children and adults, the diagnosis can be reasonably excluded on the basis of history and physical examination, with suspected cases confirmed by chest radiography. The absence of any abnormalities in vital signs or chest auscultation substantially reduces the likelihood of pneumonia as demonstrated by radiography. No single isolated clinical finding, however, is highly reliable in establishing or excluding a diagnosis of pneumonia.[6]

Older or debilitated patients with pneumonia often have nonspecific complaints, such as acute confusion or a deterioration of baseline function, without classic symptoms. Similarly, older patients may not present with a well-defined infiltrate on radiography. Older patients are more likely to have advanced illness at the time of presentation and may have sepsis in the absence of a previous syndrome suggestive of pneumonia. Occasionally, patients with lower lobe pneumonia have abdominal or back pain as a presenting symptom.

Classic teaching divides pneumonia based on clinical patterns into typical pneumonia caused by pyogenic bacteria, such as *S. pneumoniae* or *H. influenzae,* and atypical pneumonia caused by organisms such as *Mycoplasma* and *Chlamydophila* spp. This classic teaching is artificial, and a clear differentiation between these two types of pneumonia on clinical grounds alone is impossible. Clinical factors, including timing of onset, viral prodrome, absence of rigors, nonproductive cough, lower degree of fever, absence of pleurisy or consolidation, normal leukocyte count, and an ill-defined infiltrate on a chest radiograph, cannot reliably differentiate atypical pneumonias from those with pyogenic bacterial causes. Although it is impossible to determine the specific cause of pneumonia with certainty without microbiologic or serologic tests, certain clinical factors can suggest that a specific pathogen should be considered.

Clinical factors suggesting pneumococcal pneumonia include the abrupt onset of a single shaking chill, followed by fever, cough productive of rust-colored sputum, and pleuritic chest pain. Patients with a history of asplenia, sickle cell disease, HIV, multiple myeloma, or agammaglobulinemia are at increased risk of pneumococcal bacteremia and sepsis, with high mortality rates. Adults with chronic lung disease who develop pneumonia caused by *H. influenzae* typically demonstrate an insidious worsening of baseline cough and sputum production, and bacteremia is rare. *K. pneumoniae* may cause severe pneumonia in older or debilitated patients with so-called currant jelly sputum from the necrotizing nature of the infection. Abscess formation, empyema, and bacteremia are common with this organism, and mortality is high.

Mycoplasmal infection usually begins as a flulike illness with headache, malaise, fever, and nonproductive cough. Skin lesions, including maculopapular, vesicular, urticarial, or erythema multiforme–type rashes, are common, especially in younger patients. Although bullous myringitis is described as a classic finding, it is not specific for mycoplasmal infection and is seldom encountered. Patients generally do not have a toxic appearance, and most can be treated on an outpatient basis. Although mucopurulent sputum generally indicates the presence of pyogenic bacterial pneumonia or bronchitis, it may also be present with mycoplasmal or viral pneumonia. Viral pneumonia in adults is often preceded by symptoms of upper respiratory infection, such as rhinitis or sore throat. Most *C. pneumoniae* infections in young adults cause a minor, self-limited, upper respiratory illness that is subacute in onset. This organism is also associated with bronchitis, wheezing, sinusitis, and pharyngitis. Development of radiographically evident pneumonia is more common in older adults with C. *pneumoniae*. Some patients with *Legionella* infection have a mild, self-limited atypical pneumonia presentation. Older patients, smokers, and those with chronic disease or immunosuppression are more prone to develop the more acute and severe systemic illness of Legionnaires disease. Gastrointestinal symptoms, such as diarrhea and abdominal cramping, confusion, and muscle aches are sometimes prominent.

In addition to age, the presence of underlying illness, and presenting symptoms, the setting of acquisition of pneumonia may provide clues to likely causes. CAP that occurs in otherwise healthy individuals is likely to be caused by viruses, *Mycoplasma* spp., or *S. pneumoniae. S. aureus,* including MRSA, can cause severe pneumonia associated with influenza. Recently hospitalized and long-term care patients may develop pneumonia from agents that are uncommon in CAP, such as Enterobacteriaceae, *Pseudomonas aeruginosa,* and *S. aureus.* Healthy patients in an institutional setting, such as a dormitory or military barracks, are more likely to have pneumonia caused by *Mycoplasma* spp. or viruses.

Patients with underlying lung disease, especially COPD, constitute an important group likely to develop pneumonia. The lower respiratory tract of these patients is commonly colonized with organisms such as *S. pneumoniae, H. influenzae,* and *Moraxella catarrhalis.* Cystic fibrosis patients are prone to pneumonia caused by *P. aeruginosa* or *S. aureus*. Defective mucociliary clearance in both these groups makes them highly susceptible to repeated episodes of pneumonia.

Patients with immunosuppression as a result of hematologic malignancy, patients receiving chemotherapy for malignancy, and transplant recipients are prone to pulmonary infections with a wide variety of organisms. In addition to the usual pathogens, these patients may develop pneumonia secondary to viruses such as cytomegalovirus (CMV), varicella, or herpes simplex virus. They are also more likely to develop pneumonia caused by aerobic gram-negative bacilli, *Aspergillus* and geographic fungi, and *P. jiroveci.*

Although the use of highly active antiretroviral therapy has decreased the incidence of opportunistic infections among HIV-infected patients, emergency physicians are likely to encounter patients presenting with opportunistic infections who are undiagnosed or not under regular HIV care. In addition to *P. jiroveci*, there is also an increased incidence of *M. tuberculosis* and common bacterial pathogens such as *S. pneumoniae. S. pneumoniae* and *S. aureus* pneumonia remain the most common pathogens in HIV-infected patients. Other less common causes of pneumonia in HIV-infected patients include *Mycobacterium avium* complex, CMV, aerobic gram-negative bacilli, and *Cryptococcus neoformans.* PCP usually has a gradual presentation characterized by malaise, nonproductive cough, exertional dyspnea, and weight loss. Hypoxemia, hypocapnia, and absence of pulmonary effusions are common with PCP pneumonia. PCP pneumonia often presents in the ED with a decreased oxygen saturation with ambulation in the setting of HIV disease or risk factors.

The potential for opportunistic pulmonary infection can be predicted by a recent absolute CD4 lymphocyte count less than 200/mm^3.

Patients with recognized HIV infection often know their CD4 count. A total lymphocyte count less than 1000/mm^3 on a complete blood count is also suggestive and can be used as a guide for patients in which a CD4 count is not known. In patients who do not know their HIV status, the presence of findings such as weight loss, oral hairy leukoplakia, oral candidiasis, and diffuse tinea infections strongly suggests immunosuppression.

SARS-CoV-2 is a novel beta-coronavirus that emerged in 2019 causing a pandemic (see Chapter 120). Patients with COVID-19, which refers to the disease, have a variable presentation with pulmonary symptoms predominating. COVID-19 causes a range of severity from asymptomatic or minimally symptomatic respiratory infection to severe pneumonia with hypoxemia in up to 20% of patients. The route of transmission is primarily respiratory droplets and is easily spread between close contacts. Risk factors for severe disease include obesity, advanced age, hypertension, and other comorbid conditions.[9]

Patients in nursing homes and extended-care facilities are at increased risk for infection with resistant organisms such as *P. aeruginosa, K. pneumoniae* (including strains producing extended-spectrum β-lactamases), *Acinetobacter* spp., and hospital-associated strains of MRSA. In 2019, the American Thoracic Society/Infectious Disease Society of America (ATS/IDSA) issued revised guidelines for the management of patients who may be at risk for multidrug resistance based on recent exposure to the health care system.[10] These guidelines for the treatment of CAP abandoned the use of a prior categorization of health care–associated pneumonia (HCAP) which indicated risk of drug resistant pathogens and suggested broad spectrum antibiotics. The guidelines now recommend using local data on resistance patterns and individual patient risk factors to decide when to cover for pathogens such as MRSA or *Pseudomonas*. Critical illness at time of presentation to the ED, living in a nursing home, and immunosuppression are risk factors for drug-resistant pathogens.

Differential Diagnoses

Differentiation between upper and lower respiratory tract infections may be difficult. A chest radiograph helps differentiate between upper respiratory tract infection or bronchitis and pneumonia. Many noninfectious conditions may result in inflammatory lung processes, including exposure to mineral dusts (e.g., silicosis), chemical fumes (e.g., chlorine and ammonia), toxic drugs (e.g., bleomycin), radiation, thermal injury, or oxygen toxicity. Immunologic disease (e.g., sarcoidosis, anti-glomerular basement membrane disease, and collagen vascular disease) or hypersensitivity to environmental agents (e.g., farmer's lung disease) may also result in pneumonia. Tumors may be confused radiographically with pneumonia or may appear initially as a postobstructive infection or adenopathy with peripheral infiltrates. The lymphangitic spread of lung malignancy may resemble that of interstitial pneumonia.

It is important to distinguish between the acute aspiration of gastric contents or other liquids and bacterial pneumonia that may develop later as a complication of aspiration. Aspiration of liquids into the lung disrupts surfactant and causes an inflammatory response that may lead to hypoxia and respiratory failure. The aspiration of acidic gastric contents is particularly damaging to the lungs and is common in patients who are unconscious from intoxication or anesthesia or who have neurologic deficits. Patients may initially have coughing or shortness of breath or may appear well initially and then develop respiratory dysfunction during the next several hours.

Acute aspiration of acidic fluid into the lungs causes a chemical pneumonitis. This may produce fever, leukocytosis, purulent sputum, and radiographic infiltrates that mimic those of bacterial pneumonia. Although some patients go on to develop bacterial pneumonia, prophylactic administration of antibiotics is not recommended, and anaerobic coverage should be avoided. Antibiotics should be initiated if the patient develops signs of bacterial pneumonia, including new fever, expanding infiltrate appearing more than 36 hours after aspiration, or unexplained deterioration.[11]

Diagnostic Testing

Although many chest radiographs are obtained unnecessarily for patients with upper respiratory tract infections or bronchitis, it is difficult to identify a set of specific criteria to direct test ordering that is better than the clinical judgment of an experienced physician. A routine chest radiograph for all patients with cough is not necessary. Computed tomography (CT) of the chest is more sensitive than plain radiography for detecting the presence of pulmonary consolidation, although the natural history of CT-positive, plain radiograph–negative pneumonia is not clear. CT of the chest should be considered in patients such as older adults or those with significant comorbidities, for whom identification of a subtle infiltrate would change management. Young healthy adults with a presumptive diagnosis of pneumonia who will be treated as outpatients may have a chest radiograph deferred unless there is a suspicion of immunocompromise or other unusual features of disease. A chest radiograph should be obtained subsequently if there is a poor initial response to treatment. Routine performance of chest radiography for patients with exacerbation of chronic bronchitis, asthma, or COPD is of low yield and may be limited to patients with other signs of infection or congestive heart failure.

Although the causative agent cannot be determined solely by the results of chest radiography, certain radiographic patterns may suggest the possibility of specific pathogens. In pyogenic bacterial pneumonias, radiographs usually show an area of segmental or subsegmental infiltration and air bronchograms (Fig. 62.1). Lobar consolidation is present in a few cases of bacterial pneumonia, often caused by pneumococci or *Klebsiella.* A dense lobar infiltrate with a bulging fissure appearance on a chest radiograph is often described with pneumonia caused by *Klebsiella,* but this finding is nonspecific, and most cases manifest as a more subtle bronchopneumonia. Pneumonia resulting from the spread of infection along the intralobular airway results in fluffy or patchy infiltrates in the involved areas of the lung. A wide variety of bacteria and agents such as *Chlamydophila*, *Mycoplasma*, and *Legionella* spp., viruses, and fungi may cause this pattern.

An interstitial pattern on a chest radiograph (Fig. 62.2) typically is caused by *Mycoplasma* spp., viruses, or *P. jiroveci*. The classic radiographic findings in PCP are bilateral interstitial infiltrates that begin in the perihilar region (Fig. 62.3). Radiographic manifestations of PCP can vary considerably, including normal appearance and lobar infiltrates, pleural effusions, hilar adenopathy, parenchymal nodules, and cavitary disease. Tiny nodules disseminated throughout both lungs represent a miliary pattern typical of granulomatous pneumonias, such as TB or fungal disease. The location of infiltrates may also suggest the cause. Aspiration pneumonia occurs in dependent areas of the lung, usually the superior segments of the lower lobes or posterior segments of the upper lobes. Infiltrates from pneumonias produced by hematogenous spread (e.g., *S. aureus*) tend to be multiple and peripheral. Apical infiltrates suggest TB.

The presence of additional radiographic features in association with infiltrates may suggest a specific cause. An infiltrate associated with hilar or mediastinal adenopathy suggests the presence of TB or fungal disease or may indicate pneumonia associated with a neoplasm. Bacteria most likely associated with cavitation (Fig. 62.4) are anaerobes, aerobic gram-negative bacilli, and *S. aureus.* Cavitation also may be

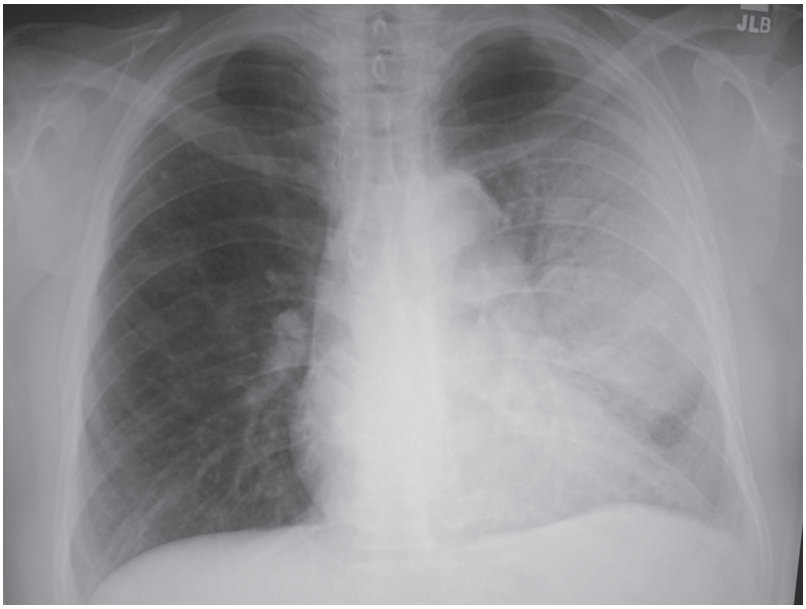

Fig. 62.1 Posteroanterior Chest Radiograph Reveals Left Upper Lobe Pneumonia. A variety of organisms can produce this pattern, usually *Streptococcus pneumoniae*, *Haemophilus influenzae*, or gram-negative bacilli, but also *Chlamydophila pneumoniae, Mycoplasma,* or *Legionella* spp.

present in fungal disease or TB and with noninfectious processes (e.g., malignancy and pulmonary vascular disease). Pneumatoceles or spontaneous pneumothorax may be seen in HIV patients with PCP. Pleural effusions occur with a wide variety of organisms, including many types of pyogenic bacterial pneumonias, *Chlamydophila* and *Legionella* spp., and TB. Anaerobic infections associated with an effusion are especially prone to the development of empyema. The diagnosis and aspiration of pleural effusions can be aided by bedside ultrasonography in the ED.

Lung ultrasonography is an emerging modality in the differentiation of respiratory complaints in the ED. The use of lung ultrasound has been shown to effectively differentiate pneumonia, congestive heart failure, and COPD/asthma in the acutely dyspneic patient.[12] Ultrasonography has been shown to be highly sensitive and specific for the diagnosis of pneumonia when compared to CT.[13] Normally air-filled alveoli in pneumonia are surrounded by fluid and on ultrasound appear as B lines which are a form of reverberation artifact. (Fig. 62.5)

Radiographic findings are nonspecific for predicting a specific infectious organism in pneumonia. *Mycoplasma* pneumonia may manifest as a dense infiltrate, or pneumococcal pneumonia may manifest as a diffuse interstitial infiltrate. Immunocompromised patients are particularly prone to having atypical radiographic appearances. Patients with a clinical picture strongly suggestive of pneumonia may have a normal initial chest radiograph and develop a radiographic infiltrate in the next 2 days. A chest radiograph is not required for the diagnosis of pneumonia. The absence of findings on a chest radiograph should not preclude the use of antimicrobial therapy in patients with clinical signs and symptoms of pneumonia. Immunocompromised patients, older adults, and patients with significant comorbidities may be treated with empirical antibiotics in the setting of signs and symptoms indicating pneumonia, even with a negative chest radiograph.

Laboratory studies also are nonspecific for identifying the cause of pneumonia. Although the finding of a white blood cell (WBC) count greater than 15,000/mm^3 increases the probability of the patient having a pyogenic bacterial cause rather than a viral or atypical cause, the predictive value of this finding depends on the stage of the illness and

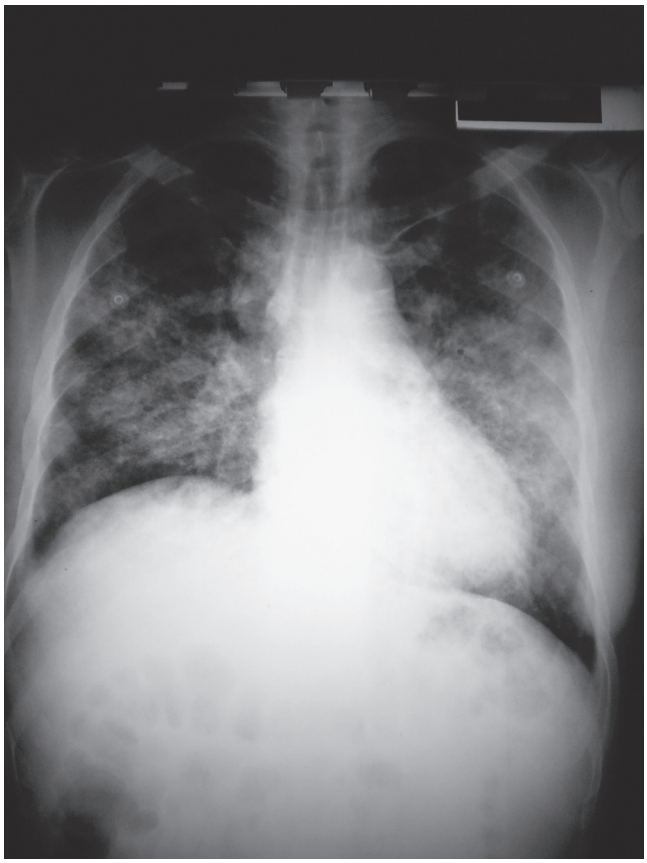

Fig. 62.2 Posteroanterior Chest Radiograph Reveals Patchy Interstitial Infiltrates. Viruses and *Mycoplasma* are the most likely causes in an otherwise healthy patient, but many bacterial organisms may also produce this pattern.

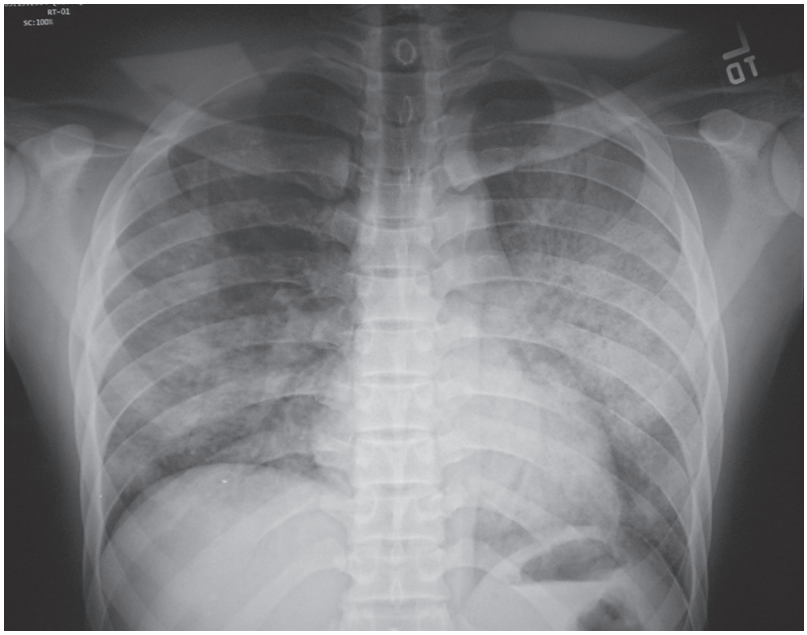

Fig. 62.3 Posteroanterior chest radiograph of a human immunodeficiency virus (HIV)–infected patient reveals interstitial disease mixed with patchy alveolar infiltrates. *Pneumocystis jiroveci* is the most common cause, but bacterial pathogens and tuberculosis also are considered.

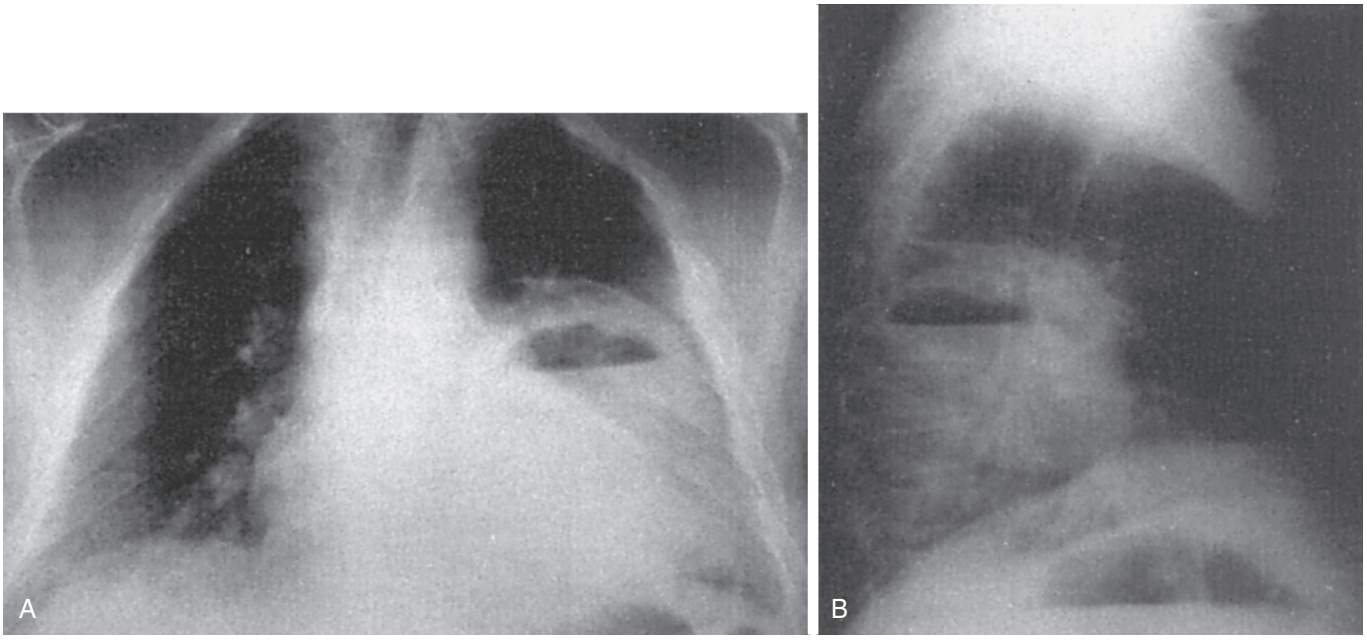

Fig. 62.4 Posteroanterior (A) and lateral (B) chest radiographs reveal a lung abscess in the left lower lobe, with a distinct air-fluid level.

likely prevalence of various causes. This is neither sensitive nor specific enough to aid decisions regarding therapy in an individual patient. A WBC count may be helpful if it yields evidence of immunosuppression, such as neutropenia, or if it reveals lymphopenia that may indicate immunosuppression from HIV. Basic metabolic panels may help identify patients with renal or hepatic dysfunction or metabolic acidosis associated with sepsis. These findings predict a complicated course and influence decisions regarding disposition, choice of antimicrobial agents, and dosages. The serum lactate dehydrogenase level is significantly elevated in patients with PCP compared with patients with non-PCP pneumonia. Inflammatory markers such as the erythrocyte sedimentation rate and C-reactive protein levels are not helpful in clinical decision making regarding pneumonia. The procalcitonin level has been suggested to assess the likelihood of a bacterial cause, response to antimicrobial therapy, and prognosis. A recent study of hospitalized patients with CAP failed to find a procalcitonin threshold that discriminated between viral and bacterial pathogens.[14] Empiric antibiotic therapy should be initiated in patients with suspected pneumonia regardless of procalcitonin level.[11]

Assessment of respiratory function with pulse oximetry is important in the evaluation of patients with pneumonia because the clinical assessment of oxygenation can be inaccurate. Pulse oximetry should be

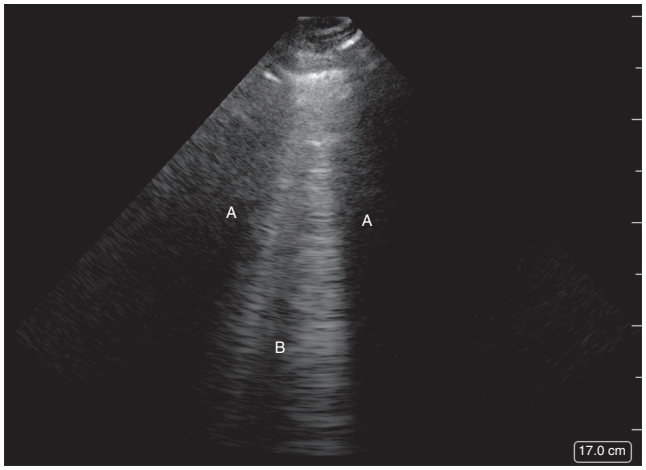

Fig. 62.5 Ultrasound image of the lung showing (A) hypoechoic rib shadows and (B) "comet tailing" or B-lines caused by reverberation artifact in lung tissue. B-lines when present anteriorly to posteriorly from the pleural line to the bottom of the image represent pneumonia or consolidation. (Image courtesy of Jackie Shibata and Alan Chiem.)

performed in any patient suspected to have pneumonia, and pneumonia should be considered in patients with low oxygen saturation.

Sputum gram staining rarely results in a change in therapy or outcome. Correlation between the identification of pneumococcus on Gram staining and sputum culture results is poor, even when commonly used criteria for an adequate sputum specimen are applied (<5 squamous epithelial cells and >25 WBCs/high-power field). Gram staining is even less likely to show gram-negative pathogens, such as *H. influenzae,* and should not be relied on to rule out a gram-negative cause. Empirical antimicrobial agents are usually highly clinically effective if chosen based on clinical information without sputum analysis. ATS/IDSA guidelines for the management of CAP support limiting sputum Gram staining and culture to patients with severe disease or risk factors for unusual pathogens.[10] Confirmation of the diagnosis of PCP requires sputum induction and staining and, in some cases, further invasive procedures, such as bronchoscopy with bronchoalveolar lavage or biopsy.

Routine blood cultures are of essentially no value in immunocompetent adults with pneumonia, in whom there is a very low prevalence of bacteremia, and management is rarely changed based on the results. Follow-up of false-positive blood cultures is costly and labor-intensive and may lead to the unnecessary use of antibiotics, such as vancomycin or linezolid, when contaminant growth is initially reported as gram-positive cocci. Blood samples for culturing should be obtained from immunocompromised patients, those with severe sepsis or requiring ICU admission, or those with risk factors for endovascular infection (e.g., prosthetic valves, IV drug use, cavitary infiltrates).[11] When culture specimens are drawn, they should ideally be obtained before the initiation of antibiotics, although antibiotics should not be delayed more than 60 minutes for this reason.

Patients with pneumonia and a large pleural effusion should undergo diagnostic thoracentesis. Fluid should be sent for cell count, differential, pH (pH <7.2 predicts the need for a thoracostomy tube), Gram staining, and culture. For most patients, thoracentesis can be safely deferred until after hospital admission. Patients in significant respiratory distress, or with evidence of tension and mediastinal shift, should undergo emergent diagnostic and therapeutic thoracentesis in the ED.

Serologic tests are widely available for the diagnosis of many organisms, including *C. pneumoniae, Legionella* spp., and fungi. The use of serologic tests to determine the cause of pneumonia may be helpful retrospectively, but these have traditionally required acute and convalescent serum titers and are of little use in the ED. Urine antigen tests for *S. pneumoniae* and *Legionella* are available and may be obtained within the time frame of an ED evaluation.

Rapid diagnostic testing using PCR such as the BioFire have increasingly become available for rapid identification of a wide variety of respiratory bacterial and viral pathogens. The use of a rapid diagnostic respiratory panel has been associated with a potential positive impact on hospital antibiotic stewardship.[15] The role and cost-effectiveness of rapid diagnostic respiratory pathogen testing in the ED is unclear. Rapid PCR testing may be especially useful in outbreak settings such as the SARS-CoV-2 pandemic to assist in isolating or cohorting patients with highly transmissible pathogens.

MANAGEMENT

The possibility of communicable disease should prompt consideration for early isolation. Patients with a history of TB exposure or suggestive symptoms (e.g., persistent cough, weight loss, night sweats, hemoptysis) or who belong to a group at high risk for TB (e.g., undomiciled, history of IV drug use or alcoholism, HIV risk, immigrant from high-risk area) should be given a mask and placed in respiratory isolation before evaluation, including chest radiography.[16] Because pulmonary TB cannot be distinguished reliably from other pulmonary infections at presentation in patients with advanced HIV, TB should be considered in all HIV-infected patients with respiratory complaints, and respiratory isolation should be initiated. The chest radiograph cannot exclude TB in this population because it may not have a typical appearance of TB. EDs that frequently care for patients at risk for TB should consider triage protocols to identify these individuals rapidly before patients, visitors, or staff are unnecessarily exposed. Suspected infection with organisms transmitted by respiratory droplet during seasonal transmission (e.g., influenza or SARS-CoV-2) should prompt infection control precautions, such as a mask being placed on the patient and proper personal protective equipment (PPE) worn by the provider.

Antimicrobials should be administered in the ED for patients who are being admitted to the hospital with suspected pneumonia. The timely administration of antimicrobials is associated with improved outcomes for hospitalized pneumonia patients, although confounding factors limit a full understanding of this relationship. Any presumed benefit of early antibiotic administration should be weighed against the risk of inappropriate use for patients in which the diagnosis is unclear. The antibiotics selected should cover the likely causes based on clinical, laboratory, radiologic, and epidemiologic information. The regimen should also be as selective as possible to avoid drug toxicity, emergence of resistance to broad-spectrum agents, and excessive cost.

The prevalence of drug-resistant *S. pneumoniae* (DRSP) has been increasing. In the United States more than 30% of isolates of *S. pneumoniae* are resistant to one or more antibiotic.[17] DRSP that is resistant to penicillin is usually resistant to other β-lactams, macrolides, tetracyclines, and trimethoprim-sulfamethoxazole (TMP-SMX). Extended-spectrum (or respiratory) fluoroquinolones, such as levofloxacin, are active against DRSP and other typical and atypical bacterial pathogens. Because the oral bioavailability of fluoroquinolones is high, oral therapy provides serum and tissue levels essentially equivalent to parenteral therapy. It is not clear, however, the extent to which in vitro resistance is related to adverse clinical outcome. Most cephalosporins achieve adequate levels in serum and tissues to treat *S. pneumoniae* respiratory tract infections successfully.

TABLE 62.1 Community-Acquired Pneumonia in Adolescents and Adults: Outpatient Treatment

Clinical Setting	Antibiotic Regimen[a]	Comments
Previously healthy, no antimicrobials in last 3 months	Doxycycline 100 mg PO bid for 7 days. or Centered between doxycyline and amoxicillin	Preferred for adolescent or young adult when likelihood of *Mycoplasma* is high; variable activity vs. *Streptococcus pneumoniae*
	Amoxicillin 1000 mg PO tid for 7 days.	Macrolide monotherapy no longer recommended unless local pneumococcal resistance is <25%.
Comorbidities or antimicrobials in last 3 months	Amoxicillin/clavulanate 875 mg/125 mg tid or Cefpodoxime 200 mg PO bid or Cefuroxime 500 mg PO bid for 7 days + azithromycin 500 mg PO on first day and then 250 mg daily for 4 days.	Especially preferable if fluoroquinolones recently received
	Levofloxacin 750 mg PO daily for 5 days	Can substitute moxifloxacin 400 mg daily for 7–14 days; active against DRSP; consider fluoroquinolone if recently received β-lactam.

DRSP, Drug-resistant Streptococcus pneumoniae; *PO,* orally.
[a]Renally dose antibiotics.

CA-MRSA remains an uncommon cause of CAP, but empirical coverage of MRSA should be strongly considered for patients with severe pneumonia associated with sepsis, especially in children or healthy young adults with likely influenza, contact with someone infected with MRSA, or radiographic evidence of necrotizing pneumonia. Antimicrobials with consistent in vitro activity against CA-MRSA isolates include vancomycin, TMP-SMX, tigecycline, linezolid, and ceftaroline. Although vancomycin is used most often for documented MRSA infections, vancomycin may be losing efficacy in light of increasing minimum inhibitory concentrations.[18]

Appropriate agents for the outpatient treatment of adults with CAP include amoxicillin, doxycycline, and fluoroquinolones with enhanced activity against *S. pneumoniae* (Table 62.1). Macrolide monotherapy is no longer routinely recommended for CAP unless local pneumococcal resistance is less than 25%.[11] Few places in the United States currently meet that criterion. In patients properly identified as being at low risk for complications with careful outpatient follow-up, use of amoxicillin 1 g PO three times daily or doxycycline 100 mg PO twice daily for 1 week is recommended. For patients at higher risk of DRSP because of recent antibiotic use or comorbidities such as chronic heart, lung, liver, or renal disease, treatment choices include monotherapy with a respiratory fluoroquinolone or combination therapy with amoxicillin/clavulanate and a macrolide, or a cephalosporin such as cefpodoxime and a macrolide. Patients who have received a fluoroquinolone in the past few months, or are at high risk of a fluoroquinolone adverse effect (e.g., QTc prolongation, tendon rupture, *Clostridioides difficile,* aortic dissection, aortic aneurysm, myasthenia gravis exacerbation), should be treated with a combination macrolide and beta lactam agent (see Table 62.1).

For patients whose illness is severe enough to require hospital admission and parenteral antibiotics, current guidelines use major and minor criteria to distinguish between non-severe and severe pneumonia (Box 62.1). For patients with a non-severe pneumonia needing admission to the hospital, a combination of a β-lactam agent such as ceftriaxone (or ceftaroline, ampicillin-sulbactam, or ertapenem) plus a macrolide such as azithromycin is the regimen recommended in ATS/IDSA guidelines (Table 62.2). Alternatively, an extended-spectrum fluoroquinolone (e.g., levofloxacin, moxifloxacin) can be given as monotherapy, but this regimen may be more likely to promote antimicrobial resistance and predispose the patient to adverse effects. These regimens treat the most common bacterial pathogens, such as *S. pneumoniae*, and *H. influenzae*, and atypical pathogens, such as *Mycoplasma, Chlamydophila,* and *Legionella* spp. β-Lactam monotherapy has been found to be noninferior to β-lactam–macrolide combination therapy or fluoroquinolone monotherapy in nonsevere CAP; it is a reasonable choice for patients without a specific reason to suspect atypical organisms.

Fluoroquinolones have some activity against TB and are important second-line agents. They should be avoided as a primary treatment for CAP in patients for whom TB is a possible diagnosis to avoid partial treatment and selecting for resistant strains. While anaerobic coverage should not be provided routinely for suspected aspiration pneumonia, if lung abscess or empyema is suspected, clindamycin or metronidazole

BOX 62.1 Criteria for Severe Community-Acquired Pneumonia

Minor Criteria[a]

- Respiratory rate ≥30 breaths/min
- Pao_2/Fio_2 ratio ≤250[b]
- Multilobar infiltrates
- Confusion, disorientation
- Uremia (BUN level ≥20 mg/dL)
- Leukopenia[c] (WBC count <4000 cells/mm^3)
- Thrombocytopenia (platelet count <100,000 cells/mm^3)
- Hypothermia (core temperature <36°C [96.8°F])
- Hypotension requiring aggressive fluid resuscitation

Major Criteria

- Invasive mechanical ventilation
- Septic shock with the need for vasopressors

[a]Other criteria to consider include hypoglycemia (in patients who do not have diabetes), acute alcoholism or alcoholic withdrawal, hyponatremia, unexplained metabolic acidosis or elevated lactate level, cirrhosis, and asplenia.
[b]A need for noninvasive ventilation can substitute for a respiratory rate higher than 30 breaths/min or a Pao_2/Fio_2 ratio below 250.
[c]As a result of infection alone.
BUN, Blood urea nitrogen; *Pao_2/Fio_2,* arterial oxygen pressure/fraction of inspired oxygen; *WBC,* white blood cell.
Adapted from Mandell LA, Wunderink RG, Anzueto A, et al; Infectious Diseases Society of America; American Thoracic Society. Infectious Diseases Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. *Clin Infect Dis.* 2007;44:S27–S72.

TABLE 62.2 Community-Acquired Pneumonia in Older Children and Adults: Inpatient Antimicrobial Treatment[a]

Clinical Setting	Antibiotic Regimen	Comments
Community-acquired, immunocompetent patient	Ceftriaxone 1 g q24h ± azithromycin 500 mg q24h IV or PO	Can substitute ceftaroline, ampicillin-sulbactam, or ertapenem for ceftriaxone.
	Respiratory fluoroquinolone (levofloxacin 750 mg IV q24h, or moxifloxacin 400 mg IV q24h)	Treats most common bacterial and atypical pathogens; active against DRSP
Severe pneumonia (ICU)	Ceftriaxone 1 g IV q24h + levofloxacin 750 mg IV q24h + vancomycin 15 mg/kg IV q12h	Can substitute cefepime, ceftaroline, ertapenem, or β-lactam or β-lactamase inhibitor for ceftriaxone; can substitute moxifloxacin for levofloxacin; can substitute linezolid for vancomycin
Increased risk of resistant pathogens with IV antibiotics within the last 90 days or severe pneumonia with neutropenia, bronchiectasis (risk for MRSA, *Pseudomonas*)	Cefepime 2g IV q12h + levofloxacin 750 mg IV q24h + vancomycin 15 mg/kg IV q12h	Can substitute other antipseudomonal β-lactams, such as piperacillin-tazobactam, aztreonam, imipenem, meropenem, or doripenem, for cefepime; can substitute aminoglycoside plus macrolide for ciprofloxacin
Presumed PCP	TMP-SMX component 5 mg/kg	Add ceftriaxone to TMP-SMX if severe, until PCP confirmed; alternatives for sulfa allergy include clindamycin + primaquine

DRSP, Drug-resistant Streptococcus pneumoniae; *ICU*, intensive care unit; *IV*, intravenously; *PCP*, Pneumocystis pneumonia; *PO*, orally; *TMP-SMX*, trimethoprim-sulfamethoxazole.
[a]Renally dose antibiotics.

should be added, or the regimen can include an antibiotic with anaerobic activity, such as ertapenem, ampicillin-sulbactam, piperacillin-tazobactam, tigecycline, or moxifloxacin (see Table 62.2).

Severely ill and compromised patients are at relatively greater risk of infection with *S. pneumoniae,* aerobic gram-negative bacilli, *S. aureus* (including MRSA) and, in some areas, *Legionella* spp. For pneumonia patients admitted to an ICU, adequate activity against DRSP may be more important. Outcomes with severe pneumonia may be better with combination therapy.[11] A third-generation cephalosporin or β-lactam or β-lactamase inhibitor can be combined with a macrolide or fluoroquinolone, and addition of vancomycin or linezolid should be considered for MRSA activity.

Patients with prior use of IV antibiotics, neutropenia, or underlying bronchiectasis are at increased risk of infection with *P. aeruginosa.* Empirical therapy for high-risk patients or critically ill patients should include two agents with extended gram-negative activity, including *P. aeruginosa.* Empirical regimens include cefepime, imipenem, meropenem, doripenem, or piperacillin-tazobactam, plus ciprofloxacin (high dose) or an aztreonam and macrolide. For life-threatening pneumonia in populations at risk for MRSA, vancomycin or linezolid should be added to empiric coverage.

Because hospital-acquired pneumonia is associated with higher mortality and a greater likelihood of unusual pathogens, the use of broader spectrum empirical therapy is often appropriate, usually with a combination of antimicrobials to increase the chance that at least one antibiotic will be active against the causative pathogen. Patients admitted to the hospital for pneumonia should be tested for influenza in the appropriate season and treated with oseltamivir if positive. The use of corticosteroids to treat influenza pneumonia is associated with increased mortality, and only recommended for patients in refractory septic shock.[19]

For patients with HIV, it is important to treat *P. jiroveci* and bacterial pathogens such as *S. pneumoniae.* TMP-SMX is the treatment of choice; the usual regimen is 15 to 20 mg/kg of TMP to be continued for 21 days, in addition to a regimen to cover CAP organisms.[20] For patients allergic to sulfa, options include clindamycin 600 mg IV q6h, plus primaquine, 30 mg (base) PO daily. The addition of corticosteroids (prednisone 40 mg PO bid) reduces mortality and clinical deterioration in patients with hypoxemia. Traditionally, this has been defined as a PaO_2 less than 70 mm Hg or alveolar-arterial gradient greater than 35 mm Hg. In practice, pulse oximetry with an SaO_2 ≤92% on room air or desaturation with exercise should warrant the initiation of corticosteroids. *Mycoplasma, Legionella,* and *Chlamydophila* spp. are uncommon causes of severe pneumonia in HIV patients, so empirical therapy with erythromycin or doxycycline is not routinely recommended.

DISPOSITION

There is tremendous variability among emergency clinicians in deciding whom to admit for pneumonia. It is a common tendency to overestimate disease severity, leading to hospitalization of patients at low risk for death or serious complications. The decision to hospitalize a patient with pneumonia does not necessarily mean that a prolonged inpatient stay is required. Observation for 12 to 24 hours in the ED observation unit or hospital may allow the early discharge of certain moderate-risk patients and help support patients with complex social issues. Inpatient treatment of pneumonia is 15 to 20 times more expensive per patient than outpatient treatment, and most patients are more comfortable in a home environment.

Although no firm guidelines exist regarding hospital admission, scoring systems may assist with hospitalization decisions. One commonly used system is the Pneumonia Severity Index (PSI), a prospectively validated predictive rule for mortality among immunocompetent adults with CAP. This model suggests a two-step approach to assess risk. Patients are assessed as low risk for outpatient management if age is less than 50 years, have normal vital signs and lack of comorbid conditions. High-risk patients are stratified by a scoring system to determine if they need admission (Table 62.3). Hospitalization is recommended for patients with a score greater than 91, and brief admission or observation may be considered for patients with a score of 71 to 90. The PSI may underestimate disease severity in younger patients and oversimplify how clinicians interpret continuous variables such as blood pressure.[11] Clinical judgment should supersede a strict

TABLE 62.3 Scoring System for Pneumonia Mortality Prediction

Patient Characteristics	Points
Demographic Factor	
Age	
• Male	Age (yr)
• Female	Age (yr)—10
Nursing home resident	10
Comorbid Illness	
Neoplastic disease	30
Liver disease	20
Congestive heart failure	10
Cerebrovascular disease	10
Renal disease	10
Physical Examination Findings	
Altered mental status	20
Respiratory rate >30 breaths/min	20
Systolic blood pressure <90 mm Hg	20
Temperature <35°C (95°F) or >40°C (104°F)	15
Pulse >125 beats/min	10
Laboratory or Radiographic Findings	
Arterial pH <7.35	30
Blood urea nitrogen >30 mg/dL	20
Sodium <130 mEq/L	20
Glucose >250 mg/dL	10
Hematocrit <30%	10
Arterial Po_2 <60 mm Hg	10
Pleural effusion	10

TABLE 62.4 SMART-COP Scoring System for Intensive Care Unit Admission

Hypotension	2 points
Multilobar chest radiograph	1 point
Hypoalbuminemia	1 point
Tachypnea	1 point
Tachycardia	1 point
Confusion	1 point
Hypoxia	2 points
Low arterial pH	2 points

interpretation of this scoring system. When emergency clinicians are provided with the patient's risk score, use of the decision rule results in a significantly lower overall admission rate, cost savings, and similar quality of life scores compared with those for patients conventionally managed by their physicians. The ability to take oral medications, access to follow-up, a stable living environment with adequate supportive services, and ambulatory pulse oximetry greater than 90% are important for successful discharge home from the ED.

A simpler tool is the CURB-65 rule. This mnemonic uses five simple criteria to determine patients at lower risk for adverse events—***c***onfusion, ***u***remia (blood urea nitrogen >20 mg/dL), ***r***espiratory rate greater than 30 breaths/min, ***b***lood pressure less than 90 systolic or less than 60 mm Hg diastolic, and age ***65*** years or older. The risk of 30-day mortality increases with more of these factors present: 0.7% with zero factors, 9.2% with two factors, and 57% with five factors. Patients with zero or one feature can receive outpatient care, those with two should be admitted, and ICU care should be considered for those with three or more factors. No randomized trials of hospital admission strategies have directly compared the PSI with the CURB-65 score. CURB-65 is the preferred clinical decision instrument given its simplicity and ability to risk stratify rapidly with readily available clinical information in the ED.

The decision to admit a patient to the ICU is straightforward when patients are intubated or require vasopressors. It is more difficult to identify patients who do not require these interventions initially but may be at greater risk for deterioration and require a level of monitoring beyond that available on the typical hospital ward. Objective criteria using the PSI (class V) and CURB-65 have been proposed but have not been prospectively validated for the ICU admission decision. When similar criteria were retrospectively studied in a cohort of CAP patients, they did not perform better than actual emergency clinician decisions. ATS/IDSA guidelines include criteria for defining severe CAP (see Box 62.1), but these have not been validated.[11] An ICU risk stratification score is abbreviated as SMART COP; intensive respiratory or vasodepressor support is predicted by clinical features such as hypoxia and hypotension (Table 62.4). A SMART-COP score above 3 points has identified 92% of patients who received intensive respiratory or vasodepressor support, including 84% of patients who did not need immediate admission to the ICU.[2] The decision to admit a patient to the hospital largely reflects the potential for acute deterioration, and some rate of floor to ICU transfer is inevitable. The Sequential Organ Assessment Score (SOFA) was found to be superior in identifying a severe state of disease compared to other CURB-65, PSI, and SMART-COP in patients admitted to the ICU with pneumonia.[21] The SOFA score is widely used in predicting mortality in patients admitted to the ICU. The SOFA score does not help differentiate lower-risk patients identified for discharge by the CURB-65 score.

Most patients with CAP do not need respiratory isolation. Patients who could pose a threat of transmission to other patients (e.g., influenza, varicella, TB, SARS-CoV-2) should be isolated. Neutropenic patients generally are placed in respiratory isolation. HIV-infected patients with pneumonia should be isolated until their TB status can be evaluated via sputum acid-fast bacilli smears; this is particularly true for patients with other risk factors for TB. Isolation should be strongly considered for others at high risk for TB.

The references for this chapter can be found online at ExpertConsult.com.

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CHAPTER 62: QUESTIONS AND ANSWERS

1. Which of the following statements is true regarding the treatment of community-acquired pneumonia (CAP)?

a. Amoxicillin monotherapy is not an option as it does not effectively cover atypical pneumonia.

b. Macrolide resistance almost always prohibits azithromycin monotherapy for CAP.

c. The most common atypical pneumonia in CAP is *Chlamydophila.*

d. Clinical history and prodrome often lead to the identification of the causative organism.

Answer: b. The 2020 American Thoracic Society/Infectious Disease Society of America (ATS/IDSA) guidelines no longer recommend macrolide monotherapy as *Streptococcus pneumoniae* resistance is greater than 30% in most settings in the United States. Amoxicillin is now recommended as a first-line agent in the treatment of CAP in healthy patients without comorbidities.

2. A 92-year-old male nursing home resident presents to the emergency department (ED) with a history of hypertension and nonproductive cough. He has a SaO_2 at triage of 82% but is speaking full sentences and with supplemental oxygen his saturation increases to 95%. Chest x-ray reveals diffuse infiltrates. Which of the following viral causes of pneumonia is this patient likely to have?

a. COVID-19

b. Respiratory syncytial virus (RSV)

c. Parainfluenza virus

d. Metapneumovirus

Answer: a. The novel coronavirus causing a worldwide pandemic is more likely to cause severe disease in patients with comorbidities and those living in nursing homes. SARS-CoV-2 often causes profound hypoxia that corrects with supplemental oxygen and causes diffuse infiltrates.

3. Which of the following causative organisms would be an indication for respiratory isolation?

a. *Histoplasma capsulatum*

b. *Herpes simplex virus* (HSV)

c. *Mycobacterium tuberculosis*

d. *Pneumocystis jiroveci*

Answer: c. Any patient with suspected tuberculosis (TB) infection should be placed in respiratory isolation and all staff should take appropriate personal protective precautions. None of the other causative agents require isolation.

4. A 60-year-old man with a past medical history of diabetes controlled with insulin and diagnosis of influenza 7 days prior presents with cough, weakness, and purulent sputum production. Vital signs are temperature 38.3°C (101°F) oral, heart rate, 130 beats/min, blood pressure, 80/50 mm Hg, respiratory rate, 30 breaths/min, and oxygen saturation, 92%. The chest radiograph reveals consolidation in the left lower lung (LLL). What is the most appropriate antibiotic therapy?

a. Ceftriaxone plus levofloxacin plus vancomycin

b. Ceftriaxone with a macrolide

c. Fluoroquinolone only

d. Trimethoprim-sulfamethoxazole (TMP-SMX)

e. Vancomycin only

Answer: a. In patients requiring hospitalization for community-acquired pneumonia (CAP), coverage for CAP would typically be with a combination of a macrolide with a β-lactam. Because this patient has signs of septic shock, consideration should be given to the addition of an agent for methicillin-resistant *Staphylococcus aureus* (MRSA; vancomycin) in addition to CAP coverage.

5. Which of the following is true regarding the use of procalcitonin in emergency department (ED) patients with pneumonia?

a. Procalcitonin is not useful in determining if a pneumonia is bacterial or viral.
b. Procalcitonin predicts mortality in ED patients.
c. It is helpful is predicting who can be discharged from the ED.
d. Procalcitonin is useful in guiding antimicrobial therapy.

Answer: a. While sometimes requested by hospitalist colleagues, the ED use of procalcitonin has not been shown to be useful in determining mortality, viral versus bacterial etiology, mortality, or prognosis.