both diagnostic and therapeutic purposes. If the fluid has a pH of <7, a glucose level of <2. 2 mmol/L, and a lactate dehydrogenase concentration of >1000 U/L or if bacteria are seen or cultured, then it should be completely drained; a chest tube is often required and video-assisted thoracoscopy may be needed for late treatment or difficult cases.

Follow-Up Fever and leukocytosis usually resolve within 2–4 days in otherwise healthy patients with CAP, but physical findings may persist longer. Chest radiographic abnormalities are slowest to resolve (4–12 weeks), with the speed of clearance depending on the patient's age and underlying lung disease. Patients may be discharged from the hospital once their clinical conditions, including comorbidities, are stable. The site of residence after discharge (nursing home, home with family, home alone) is an important discharge timing consideration, particularly for elderly patients. For a hospitalized patient, a follow-up radiograph ~4–6 weeks later is recommended. If relapse or recurrence is documented, particularly in the same lung segment, the possibility of an underlying neoplasm must be considered.

PROGNOSIS

The prognosis of CAP depends on the patient's age, comorbidities, and site of treatment (inpatient or outpatient). Young patients without comorbidity do well and usually recover fully after ~2 weeks. Older patients and those with comorbid conditions can take several weeks longer to recover fully. The overall mortality rate for the outpatient group is <1%. For patients requiring hospitalization, the overall mortality rate is estimated at 10%, with ~50% of deaths directly attributable to pneumonia.

PREVENTION

The main preventive measure is vaccination (Chap. 148). Recommendations of the Advisory Committee on Immunization Practices should be followed for influenza and pneumococcal vaccines.

A pneumococcal polysaccharide vaccine (PPV23) and a protein conjugate pneumococcal vaccine (PCV13) are available in the United States. The former product contains capsular material from 23 pneumococcal serotypes; in the latter, capsular polysaccharide from 13 of the most frequent pneumococcal pathogens affecting children is linked to an immunogenic protein. PCV13 produces T cell–dependent antigens that result in long-term immunologic memory. Administration of this vaccine to children has led to an overall decrease in the prevalence of antimicrobial-resistant pneumococci and in the incidence of invasive pneumococcal disease among both children and adults. However, vaccination can be followed by the replacement of vaccine serotypes with nonvaccine serotypes, as was seen with serotypes 19A and 35B after introduction of the original 7-valent conjugate vaccine. PCV13 now is also recommended for the elderly and for younger immunocompromised patients. Because of an increased risk of pneumococcal infection, even among patients without obstructive lung disease, smokers should be strongly encouraged to stop smoking.

Two forms of influenza vaccine are available: intramuscular inactivated vaccine and intranasal live-attenuated cold-adapted vaccine. The latter is contraindicated in immunocompromised patients. In the event of an influenza outbreak, unprotected patients at risk from complications should be vaccinated immediately and given chemoprophylaxis with either oseltamivir or zanamivir for 2 weeks—i.e., until vaccine-induced antibody levels are sufficiently high.

HEALTH CARE–ASSOCIATED PNEUMONIA

HCAP represents a transition between classic CAP and typical HAP. The definition of HCAP is still in some flux because of a lack of consistent large-scale studies. Several early studies were limited to patients with culture-positive pneumonia. In these studies, the incidence of MDR pathogens in HCAP was as high as or higher than in HAP/VAP. MRSA in particular was more common in HCAP than in traditional HAP/VAP. Conversely, prospective studies in nontertiary-care centers have found a low incidence of MDR pathogens in HCAP.

The patients at greatest risk for HCAP are not well defined. Patients from nursing homes are not always at elevated risk for infection with MDR pathogens. Careful evaluation of nursing home residents with pneumonia suggests that their risk of MDR infection is low if they have not recently received antibiotics and are independent in most activities of daily living. Recent hospitalization (i.e., in the preceding 90 days) is also a major risk factor for infection with MDR pathogens. Conversely, nursing home patients are at increased risk of infection with influenza virus and other atypical pneumonia pathogens. Undue concern about MDR pathogens occasionally results in failure to cover atypical pathogens when treating nursing home patients. In addition, patients receiving home infusion therapy or undergoing chronic dialysis are probably at particular risk for MRSA pneumonia but may not be at greater risk for infection with *Pseudomonas* or *Acinetobacter* than are other patients who develop CAP.

In general, the management of HCAP due to MDR pathogens is similar to that of MDR HAP/VAP. This topic will therefore be covered in subsequent sections on HAP and VAP. The prognosis of HCAP is intermediate between that of CAP and VAP and is closer to that of HAP.

VENTILATOR-ASSOCIATED PNEUMONIA

Most hospital-acquired pneumonia research has focused on VAP. However, the information and principles based on this research can be applied to non-ICU HAP and HCAP as well. The greatest difference between VAP and HCAP/HAP studies is the dependence on expectorated sputum for a microbiologic diagnosis of VAP (as for that of CAP), which is further complicated by frequent colonization by pathogens in patients with HAP or HCAP. Therefore, most of the literature has focused on HCAP or HAP resulting in intubation, where, once again, access to the lower respiratory tract facilitates an etiologic diagnosis.

Etiology Potential etiologic agents of VAP include both MDR and non-MDR bacterial pathogens (Table 153-6). The non-MDR group is nearly identical to the pathogens found in severe CAP (Table 153-2); it is not surprising that such pathogens predominate if VAP develops in the first 5–7 days of the hospital stay. However, if patients have other risk factors for HCAP, MDR pathogens are a consideration, even early in the hospital course. The relative frequency of individual MDR pathogens can vary significantly from hospital to hospital and even between different critical care units within the same institution. Most hospitals have problems with *P. aeruginosa* and MRSA, but other MDR pathogens are often institution-specific. Less commonly, fungal and viral pathogens cause VAP, usually affecting severely immunocompromised patients. Rarely, community-associated viruses cause mini-epidemics, usually when introduced by ill health care workers.

TABLE 153-6 MICROBIOLOGIC CAUSES OF VENTILATOR-ASSOCIATED PNEUMONIA

Non-MDR Pathogens	MDR Pathogens
Streptococcus pneumoniae	*Pseudomonas aeruginosa*
Other *Streptococcus* spp.	MRSA
Haemophilus influenzae	*Acinetobacter* spp.
MSSA	Antibiotic-resistant
Antibiotic-sensitive	Enterobacteriaceae
Enterobacteriaceae	*Enterobacter* spp.
Escherichia coli	ESBL-positive strains
Klebsiella pneumoniae	*Klebsiella* spp.
Proteus spp.	*Legionella pneumophila*
Enterobacter spp.	*Burkholderia cepacia*
Serratia marcescens	*Aspergillus* spp.

Abbreviations: ESBL, extended-spectrum β-lactamase; MDR, multidrug-resistant; MRSA, methicillin-resistant *Staphylococcus aureus;* MSSA, methicillin-sensitive *S. aureus.*