Introduction

Hospital-acquired pneumonia (HAP) is a respiratory infection which occurs 48 hours after hospital admission. It is an important cause of hospital morbidity, observed most frequently in medical wards and in elderly patients with severe underlying illnesses. HAP is the second most frequent cause of nosocomial infection, accounting for 15–20% of such infections. It occurs in 0.5–1.5% of inpatients and is usually caused by bacteria.

HAP accounts for more than 50% of all antibiotics prescribed. It is currently the most common infection contributing to high mortality and morbidity. HAP is the leading cause of death among hospital-acquired infections, with the crude mortality rate for HAP perhaps as high as 20–71%, but many of the patients with HAP die of their underlying disease rather than pneumonia. An increase in mortality attributable to HAP is related to the presence of multidrug-resistant (MDR) pathogens. The clinical characteristics of pneumonia in the elderly differ substantially from those in younger patients, and the severity of the disease is strongly associated with increased age and age-related comorbid disorders.

Key Words: aged, aging, cross infection, pneumonia
amend the antibiotic choice in the elderly. Aspiration pneumonia is also an important clinical feature in elderly patients suffering from cerebrovascular disorder-associated dysphagia.

Risk Factors and Prognosis

The parameters associated with an increased risk of pneumonia include alcoholism, swallowing disorders, male gender, age greater than 60 years, neoplasms, chronic obstructive pulmonary disease (COPD), diabetes, heart failure, immunosuppression, intubation, and prolonged ventilation. Low serum albumin is also one of the predictors for pneumonia developing in the elderly, and malnutrition is more often found in elderly patients with pneumonia.

There are different risk factors for HAP with varied pathogens. Risk factors for infection with *Pseudomonas aeruginosa* include age greater than 65 years, B-lactam therapy within the previous 3 months, alcoholism, cirrhosis, chronic cardiovascular or pulmonary disease, diabetes mellitus, corticosteroid use, multiple comorbidities, functional or anatomic asplenia, cerebrospinal fluid leak, and immunosuppressive disorder or treatment. Risk factors for enteric Gram-negative rods include nursing home residence, cardiopulmonary disease, multiple comorbidities, and recent antibiotic therapy. Infection by *Pseudomonas aeruginosa* is associated with a number of risk factors that are not associated with age but with structural lung diseases, recent antibiotic treatment, broad spectrum antibiotic use for more than 7 days in the last month, malnutrition, and prednisone > 10 mg/day. Risk factors for infection with *Staphylococcus aureus* include end-stage renal disease, injection drug abuse, prior influenza, and prior antibiotic use (particularly fluoroquinolones), diabetes mellitus, head trauma, and intubation.

Conditions known to increase mortality include prolonged mechanical ventilatory support, an underlying fatal condition, age greater than 60 years, bilateral radiographic infiltrates, prior antibiotic therapy, prior pneumonia with superinfection, chronic lung disease, low body mass index, Gram-negative bacilli, polymicrobial flora, and microbes that have acquired antibiotic resistance. Increased mortality rates were also associated with bacteremia, especially with *P. aeruginosa* or *Acinetobacter* species, medical rather than surgical disease, and therapy with ineffective antibiotic treatment.

Etiology

HAP may be caused by a wide spectrum of bacterial pathogens, and is more frequently polymicrobial in ventilator-associated pneumonia. HAP is rarely caused by viral or fungal pathogens in immunocompetent patients. It has been established that colonization dramatically increases in patients with acidosis, alcoholism, azotemia, coma, diabetes mellitus, hypotension, leukocytosis, leukopenia, pulmonary disease, endotracheal or nasogastric intubation, and in patients given antimicrobial agents. With gastric pH > 4, microorganisms are able to increase to high concentrations in the stomach. This can happen in patients with advanced age, achlorhydria, ileus, upper gastrointestinal disease, enteral feeding, antacids, and histamine2 (H2) antagonists. Impaired intestinal motility is also attributed to gastric colonization in patients. In addition, certain conditions such as malnutrition, severe illness or post-operative state can increase the adherence of Gram-negative bacteria. There are a number of factors that enhance colonization of the oropharynx and/or stomach by microbes, including administration of antimicrobial agents, presence of underlying chronic lung disease, prolonged mechanical ventilatory support, contact with contaminated or colonized hands, extreme old age, malnutrition, severe underlying conditions, and immunosuppression. The high incidence of Gram-negative bacillary pneumonia in hospitalized patients appears to be the result of factors that promote colonization of the pharynx by Gram-negative bacilli and the subsequent entry of these microorganisms into the lower respiratory tract.

Early-onset HAP occurs within the first 4 days of hospitalization and usually has a better prognosis. Early-onset HAP without risk factors for MDR is often caused by typical antimicrobial-susceptible organisms such as *S. pneumoniae, Haemophilus influenzae*, methicillin-sensitive *S. aureus*, and antibiotic-sensitive enteric Gram-negative bacilli (*Escherichia coli*, *Klebsiella pneumoniae*, *Enterobacter* species, *Proteus* species, and *Serratia marcescens*). Several studies have demonstrated that pneumonia caused by *Mycoplasma pneumoniae* is less common in elderly patients, and viruses, mainly influenza and the respiratory syncytial virus, also play a role in the occurrence of pneumonia in the elderly.

Silent aspiration during sleep seems to happen more frequently in elderly people with pneumonia, regardless of neurologic disorders. Aspiration of nasopharyngeal...
or gastric contents is a frequent occurrence among elderly patients, being related mainly to neurologic disorders that cause dysphagia and decreased cough reflex. Persons with abnormal swallowing, such as those who have depressed consciousness, respiratory tract instrumentation, gastrointestinal tract instrumentation, or who have just undergone surgery, especially thoracic and/or abdominal surgery, are particularly prone to aspiration. An acute cerebrovascular disorder seems to be the most common cause of swallowing difficulties. Up to 80% of these patients present with dysphagia, and 40–50% develop aspiration pneumonia. It has been reported that the oral hygiene in elderly patients, being related mainly to neurologic disorders that cause dysphagia and decreased cough reflex, is similar to that in ventilated patients. Some microbes, such as MRSA and K. pneumoniae, are more common in non-ventilated than ventilated patients, whereas certain resistant Gram-negative bacilli are more common in patients with ventilator-associated pneumonia (P. aeruginosa, Stenotrophomonas maltophilia, and Acinetobacter species). In one study of 52 patients aged 70 years who failed to respond to 72 hours of antibiotics, MRSA (33%), Gram-negative enterics (24%) and Pseudomonas species (14%) were the most frequent pathogens isolated by bronchoscopy. It was noted that 72% had at least two comorbidities, whereas 23% had three or more. The frequency of specific MDR pathogens causing HAP may vary by hospital, patient population, exposure to antibiotics, type of intensive care unit patient and changes over time, addressing the need for timely, local surveillance data.

Environmental microorganisms such as Legionella species and Aspergillus species should be considered in HAP in patients not receiving ventilation. Legionella pneumophila is increased in immunocompromised hosts, such as organ transplant recipients, human immunodeficiency virus disease, diabetes mellitus, underlying lung disease, and end-stage renal disease. HAP due to Legionella species is more common in hospitals where the pathogen is present in the hospital water supply. Detection is based on the widespread use of Legionella urinary antigen. Disease due to serogroups other than serogroup 1 may be underdiagnosed. Controlling the risk of Legionella in hospital water supplies decreases the risk of Legionella pneumonia.

**Evaluation and Diagnosis**

It is difficult to diagnose HAP. Traditional criteria, such as fever, cough, sputum production and pleuritic chest pain, are not sufficiently accurate to confirm pneumonia. The clinical presentation of pneumonia in older patients is often without the typical acute symptoms observed in young adults. This clinical paucity causes a delay of diagnosis by ≥72 hours, contributing to further mortality. However, altered mental status (delirium) may be a common manifestation of pneumonia in older patients. Both clinical features and physical findings may be lacking or misrepresented in elderly patients. All patients should be screened by pulse

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oximetry, which may imply the presence of pneumonia in patients without obvious signs of pneumonia. In addition, elderly patients with pneumonia are more likely to have an unclear pulmonary infiltrate on chest radiography. For patients with suspected pneumonia but who have negative chest radiography findings, it may be reasonable to treat their condition presumptively with antibiotics and repeat the imaging in 24–48 hours. Chest tomographies may be more sensitive. The presence of a new or progressive radiographic infiltrate with at least two of three clinical features, fever greater than 38°C, leukocytosis or leukopenia, and purulent secretions, represent the most accurate clinical criteria for beginning empirical antibiotic therapy. Direct staining is a simple procedure, providing valuable information within 1 hour. A reliable Gram stain can be used to guide initial antibiotic therapy and may increase the diagnostic value of the clinical pulmonary infection score (CPIS). Reevaluation of the decision to use antibiotics according to the results of semiquantitative lower respiratory tract cultures and serial clinical evaluations is necessary by day 3 or sooner. A CPIS of 6 or less for 3 days is an objective criterion to select patients at low risk for early discontinuation of empirical therapy for HAP.

The application of noninvasive diagnostic tests did not allow us to determine the importance of microorganisms that are normal oropharyngeal flora or which colonize hospitalized patients. The etiologic diagnosis was only established in one-third of the cases of HAP in noncritical patients because of the inability to perform invasive procedures in most of the patients.

If bronchoscopic sampling is not immediately available, non-bronchoscopic sampling can reliably get lower respiratory tract secretions for quantitative cultures, which can be used to guide antibiotic therapy. Invasive sampling methods involve bronchoscopic techniques to obtain quantitative cultures of protected specimen brush and bronchoalveolar lavage. The reported sensitivities and specificities of these methods range from 70–100% and 60–100%, respectively, depending on the tests or diagnostic criteria with which they are compared. A major factor causing false-negative quantitative cultures is the recent initiation of, or change in, antimicrobial therapy, especially in the prior 24 hours, but up to 72 hours. The threshold of quantitative cultures of protected specimen brush is 10^3 colony-forming unit/mL or more. The threshold of bronchoalveolar lavage is 10^4 or 10^5 colony-forming unit/mL, and the advantage of looking for intracellular organisms is the ability to obtain data of high predictive value in a rapid time frame. The diagnostic threshold may be lowered if the patient has had changes in antibiotic therapy recently or if the probability of infection is high. Soluble triggering receptor expressed on myeloid cells coupled with the classic clinical criteria and results of microbiologic cultures may be a valuable tool with which to enhance the specificity and maintain the sensitivity of HAP diagnosis. Delays in the initiation of appropriate antibiotic therapy can increase the mortality and, therefore, therapy should not be postponed for the purpose of performing diagnostic studies in patients who are clinically unstable. Clinically unstable patients with signs of infection should receive therapy, in spite of initial bronchoscopic findings. The use of lung tissue for diagnosis of pneumonia is not recommended in most ventilated patients; it can, however, be useful in some immunocompromised patients.

Treatment

It is widely presumed that treatment outcomes for HAP will be affected by the susceptibilities of the infecting organisms. Treatment should be administered as soon as possible after the diagnosis is considered probable, especially in cases of severe HAP. Early proper antibiotic therapy improves the outcome of HAP with a lower attributable mortality. There is a survival benefit to increasing the percentage of patients who receive antibiotics within the first 4–8 hours. Early appropriate broad-spectrum antimicrobial treatment should be prescribed with sufficient doses to optimize antibiotic efficacy. Therapies which the patients have received within the previous 2 weeks should also be considered, avoiding use of the same antimicrobial class, when possible. Microbiologic results may come too late to influence outcome, and broad-spectrum empirical antimicrobial therapy is often used before microbiologic diagnosis is confirmed. A lower respiratory tract culture needs to be collected before antimicrobial treatment, but collection of cultures should not defer the initiation of therapy in critically ill patients. Either “quantitative” or “semiquantitative” culture data can be used for the treatment of patients with HAP. Quantitative cultures increase specificity of the diagnosis of HAP without negative effects, and the specific quantitative technique should be selected on the basis of
local expertise and experience. Specimens obtained after initiation of antimicrobial treatment are unreliable and must be interpreted carefully.

Negative lower respiratory tract cultures can be used to discontinue antimicrobial therapy in a patient who has had cultures performed in the absence of an antimicrobial change in the past 72 hours. A clinical protocol utilizing CPIS ≤ 6 would regard elderly patients at low risk of HAP, suitable for early discontinuation of empirical antimicrobial treatment for HAP on the third day. Clinical improvement usually takes 48–72 hours and, thus, treatment should not be changed during this time unless there is a rapid clinical deterioration. De-escalation of antimicrobials should be considered once data becomes available regarding the results of lower respiratory tract cultures and the patient’s clinical response. Use of a shorter duration of antimicrobial therapy (7–8 days) is suggested for patients with uncomplicated HAP who have received initially negative microorganisms63,64. Elderly patients with more chronic comorbidities may need a longer duration of therapy until complete resolution of pneumonia-related symptoms is achieved65.

Early-onset HAP without risk factors for MDR pathogens is usually caused by antimicrobial-susceptible microbes such as *S. pneumoniae*, *H. influenzae*, methicillin-sensitive *S. aureus*, and antibiotic-sensitive enteric Gram-negative bacilli (*E. coli, K. pneumoniae, Enterobacter species, Proteus species, and S. marcescens*). Therapeutic choices of early-onset HAP without risk factors for MDR pathogens include ceftriaxone or levofloxacin, moxifloxacin, ciprofloxacin, ampicillin/sulbactam, or ertapenem. Presently, many strains of *S. pneumoniae* are penicillin resistant66. Despite low and moderate resistance to penicillins and cephalosporins in vitro, clinical results in patients with pneumococcal pneumonia and bacteremia treated with these antibiotics have been satisfactory66. The MDR pneumococci are currently sensitive to vancomycin or linezolid, and most remain sensitive to broad-spectrum quinolones. The overuse of fluoroquinolones has also resulted in the emergence of fluoroquinolone-resistant pneumococci67. The advantage of combination therapy is most striking in the more severely ill patients68. *Klebsiella* species are intrinsically resistant to aminopenicillins and can obtain resistance to cephalosporins and aztreonam by the production of extended-spectrum β-lactamases69. Plasmid mediated resistance, such as extended-spectrum β-lactamase production, is a more common mechanism for β-lactam resistance in nosocomial isolates, and is increasingly recognized in isolates of *K. pneumoniae* and *E. coli, and Enterobacter species*70. Use of a fourth-generation cephalosporin such as
Acinetobacter species have a particularly high mortality and should be used at adequate doses. Extended-spectrum β-lactamases have been reported. Some MDR pathogens of P. aeruginosa have β-lactamase and carbapenem resistance. Treatment should include an antipseudomonal cephalosporin (cefepime or cefepime for this infection is disputed). A reliable option is a carbapenem, which is generally active against these pathogens. Piperacillin–tazobactam against microbes with extended-spectrum β-lactamase is unpredictable and should be used at adequate doses.

A clinical distinction between pneumonia and aspiration pneumonitis may be important for therapeutic management in the elderly. Neither signs and symptoms nor laboratory tests can identify aspiration pneumonia. However, the history of witnessed or suspected aspiration is the only parameter that restricts antimicrobial use in these patients. A reliable selection comprises clindamycin, metronidazole or β-lactamase inhibitor.

MDR organisms are more commonly isolated from patients with severe chronic underlying disorders and patients with late-onset HAP. Having pathogens resistant to the empirical treatment has been independently associated with mortality. An initially appropriate antibiotic regimen could allow reduction of the period of treatment from the traditional 14–21 days to durations as short as 7 days, provided that the etiologic microbe is not P. aeruginosa and that the patient has a good clinical response with resolution of clinical characteristics of infection. Elderly patients with multiple comorbid disorders often recover more slowly. Short-duration treatment may be suboptimal for patients with initial treatment not active against the identified microbes, lung tissue necrosis, bacteremia, S. aureus, and P. aeruginosa.

Patients superinfected with P. aeruginosa or Acinetobacter species have a particularly high mortality, around 90% in some series. P. aeruginosa and Acinetobacter are common causes of late-onset pneumonia, especially in the ventilated patient. P. aeruginosa has intrinsic resistance to many antimicrobial agents. Infection caused by P. aeruginosa is associated with a significantly higher incidence of therapeutic failure than those caused by other pathogens. Treatment should include an antipseudomonal cephalosporin (cefepime, ceftazidime), an antipseudomonal carbapenem (imipenem or meropenem), β-lactam/β-lactamase inhibitor (piperacillin–tazobactam) plus an antipseudomonal fluoroquinolone (ciprofloxacin or levofloxacin), or aminoglycoside (amikacin, gentamicin or tobramycin). Resistant strains of P. aeruginosa with imipenem-type enzymes and other carbapenemases have been reported. Some MDR pathogens of P. aeruginosa are susceptible only to polymyxin B. Some anecdotal experience has led to a recommendation of aerosolized antibiotics as an adjunct to systemic treatment in patients with highly resistant P. aeruginosa pneumonia. Combination therapy has been continued for less than the full course of therapy, with cessation of the aminoglycoside after 5–7 days if the patient is improving.

Acinetobacter species are generally less virulent than P. aeruginosa, but have, nonetheless, become resistant to common used antimicrobial agents. Carbapenem resistance has been found in 40% of 3,601 isolates of Acinetobacter baumannii. The most active agents in vitro are the polymyxins, i.e., polymyxin B and polymyxin E (colistin). The nephrotoxicity rates of polymyxin range up to 36%, and neurotoxicity is currently uncommon. An alternative to carbapenem-resistant A. baumannii is combination therapy with other antibiotics, such as rifampin or aminoglycosides, sulbactam, ampicillin–sulbactam, and the polymyxins. Adjunctive treatment with an aerosolized aminoglycoside or polymyxin for carbapenem-resistant A. baumannii should be considered, particularly in patients who are not improving with systemic therapy.

Stenotrophomonas maltophilia, which shares with Burkholderia cepacia a tendency to colonize the respiratory tract rather than cause an invasive disorder, is uniformly resistant to carbapenems because of a ubiquitous metallo-β-lactamase. S. maltophilia and B. cepacia are most likely to be susceptible to trimethoprim–sulfamethoxazole, ticarcillin–clavulanate or a fluoroquinolone. B. cepacia is also commonly susceptible to ceftazidime and carbapenems.

S. aureus is ubiquitous in critical care areas. More than 50% of the intensive care unit infections are caused by MRSA. Vancomycin has been the accepted standard for treatment of MRSA. Dosing vancomycin in patients with fluctuating renal function is difficult and needs frequent monitoring of levels. There is an increased risk of nephrotoxicity in patients receiving vancomycin together with other nephrotoxic agents, especially aminoglycosides. Combination therapy with other antimicrobials, such as rifampin and aminoglycosides, has been tried but no prospective clinical data have validated the value of this regimen. Linezolid may also be preferred if patients have renal insufficiency or are taking other nephrotoxic agents. Linezolid resistance is rare in S. aureus presently. However, no firm conclusion can be drawn in considering the use...
of linezolid or a glycopeptide as optimal therapy for patients with HAP caused by MRSA. 

*Legionella pneumophila* occurs sporadically but may be endemic in hospitals with contaminated water systems or cooling units. The incubation period of *Legionella* infection commonly is 2–10 days; infections that occur more than 10 days after admission are considered to be nosocomial, and infections that develop between 4 and 10 days are considered as possibly nosocomial. The rates of *L. pneumophila* vary remarkably between hospitals. It occurs more frequently with serogroup 1 when the water supply is colonized. Mono-therapy with one of the newer macrolides (especially azithromycin) and the quinolones are effective for *Legionella* infection. Other agents include tetracycline, doxycycline, and trimethoprim–sulfamethoxazole. Combination therapy with rifampin is only recommended in patients for whom standard therapy appears to be failing. Patients from long-term care facilities, patients with a nosocomial infection or patients who have received transplants should be treated with a fluoroquinolone to provide better coverage of other Gram-negative bacilli. The total course of treatment for *Legionella* pneumonia is 7–10 days for azithromycin, 5 days for levofloxacin (750 mg once daily), and 10–14 days for other medications. Patients who are severely ill or immunocompromised should receive treatment for a total duration of 21 days.

### General Prophylaxis

Patients at high risk of HAP include those with abdominal aortic aneurysm repair, thoracic surgery or emergency surgery, general anesthesia, age more than 60 years, totally dependent functional status, weight loss of more than 10%, use of steroids for chronic diseases, a recent history of alcohol use, COPD or smoking during the preceding year, impaired sensorium, a history of cerebrovascular disorder with residual neurologic deficit, low (<8 mg/dL) or high (>22 mg/dL) blood urea nitrogen level, as well as having received more than four units of blood before operations. An important issue that deserves more recognition regarding the management of HAP in the elderly is prevention. Effective plans include strict infection control, alcohol-based hand disinfection, microbiologic surveillance with timely availability of data on local MDR pathogens, monitoring and early removal of invasive devices, and programs to reduce or alter antimicrobial-prescribing practices.

It is important to preoperatively educate patients at high risk of developing pneumonia on the postoperative use of incentive spirometry. Taking deep breaths and becoming mobile as soon as possible in the postoperative period should also be stressed. Chest physiotherapy may be of value to patients suffering from or at risk of HAP, and it might be appropriate to regard it as a therapeutic option.

Aspiration due to dysphagia is a condition that must be considered, especially in patients with a cerebrovascular accident, confusion, alcoholism, and in bedridden patients. A supine position facilitates aspiration, which may be reduced by semirecumbent positioning. Therefore, patients should be managed in a semirecumbent position, particularly during feeding. The use of oral, endotracheal and orogastric tubes, rather than nasotracheal and nasogastric tubes, can decrease the frequency of nosocomial sinusitis and possibly pneumonia. Parenteral nutrition is associated with a higher risk of intravascular device-associated infections and loss of intestinal villous architecture. Enteral nutrition is favored over parenteral nutrition to reduce the risk of complications. The rate and volume of enteral feeding should be adjusted to avoid gastric distension and to lower the risk of aspiration. A pooled analysis showed no benefit from post-pyloric versus gastric tube feeding in critically ill patients.

Intubation increases the risk of HAP and hence should be avoided whenever possible. To prevent HAP, daily interruption or reduction of sedative/paralytic agents and maintenance of endotracheal cuff pressure at greater than 20 cmH₂O in patients with intubation is recommended. Repeat insertion of the endotracheal tube and emergency intubation are risk factors for pneumonia. Using noninvasive ventilation and avoiding reintubation after initial extubation may reduce the risk of HAP. Noninvasive positive pressure ventilation is an alternative for patients with acute exacerbations of COPD or acute hypoxemic respiratory failure, and for immunosuppressed patients with pulmonary infiltrates and respiratory failure. A reduced duration of intubation can be gained by the use of weaning protocols to reduce the duration of mechanical ventilatory support and to reduce the risk of HAP.

An inclination to reduce pneumonia with sucrafate and to avoid blood transfusion is necessary. There is a slightly higher rate of clinically significant
gastric bleeding when sucralfate is used for stress bleeding prophylaxis. If necessary, stress bleeding prevention with either H₂ antagonists or sucralfate is acceptable. Exposure to blood products is a risk factor for postoperative infection and postoperative pneumonia, and the length of time of blood storage is another factor-modulating risk. Immunosuppressive effects of non-leukocyte-depleted red blood cell units increase the risk of infection. Leukocyte-depleted red blood cell transfusions can reduce HAP in selected patients. We recommend that to prevent HAP, red cell transfusions should be avoided if possible and, if used, should be carried out with fresh red cells.

Prophylaxis for pneumonia in the elderly, with influenza and pneumococcal vaccination, nutritional measures and oral hygiene, is important. Studies in elderly persons have apparently demonstrated that the polysaccharide vaccine prevents an invasive pneumococcal disorder and assures protection for 5–6 years. The pneumococcal vaccine is associated with a decreased antibody response in the elderly. Recent findings have shown that revaccination in elderly people with the polysaccharide vaccine is safe and may give a further immune response. The pneumococcal polysaccharide vaccine is suggested for persons >65 years of age and particularly for those with selected high-risk concomitant disorders (chronic cardiovascular disease, chronic pulmonary diseases, diabetes mellitus, alcoholism, chronic liver disease, or cerebrospinal fluid leaks, functional or anatomic asplenia, HIV infection, leukemia, lymphoma, Hodgkin disease, multiple myeloma, generalized malignancy, chronic renal failure, nephrotic syndrome, or other conditions related to immunosuppression, such as solid organ transplantation or human stem-cell transplantation, and persons receiving immunosuppressive chemotherapy). Vaccination may be carried out either at hospital discharge or during outpatient treatment. Nutrition is often overlooked in the elderly with HAP. A low albumin level is a risk factor for developing pneumonia, and a low body mass index is associated with a higher mortality rate. A sufficient diet should be recommended, and the nutritional status of patients should be routinely checked.

Conclusion

Pneumonia in the elderly is a very serious condition that requires swift management. The antibiotic selection should be made based on the most recent guidelines. It is important to recognize the variability of bacteriology from one hospital to another, specific sites within the hospital and from one time period to another, and to use this information to guide the selection of an appropriate antibiotic treatment regimen. Avoiding the overuse of antibiotics by tailoring therapy to the results of lower respiratory tract cultures and shortening the duration of therapy to the minimal effective period is required. The application of preventative strategies aimed at adjustable risk factors should be seriously considered.

References


