Diagnosis and Management of Pneumonia in the Emergency Department

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Pneumonia remains a major cause of death in developed countries [1]. Patients with community-acquired pneumonia (CAP) are most often managed in an outpatient setting. The mortality rate in this patient population is low (<1%) in contrast to patients who require hospitalization, who have a mortality rate of approximately 15%. Because most patients with pneumonia are managed by emergency and primary care physicians, infectious disease specialists tend to see a population of patients with pneumonia that is skewed toward more complicated and severe infections. Emergency physicians may be less inclined than infectious diseases specialists to pursue aggressive diagnostic testing and cultures, except in patients who are seriously ill. Whereas in the past decisions regarding initial antibiotic therapy were deferred to admitting primary care and consulting physicians, quality standards currently reinforce timely initiation of antibiotics in the emergency department (ED). The practicality and ultimate consequences of arbitrary time standards are debated, however. Pneumonia management remains challenging because of several constantly changing factors,

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including an expanding spectrum of pathogens, changing antibiotic resistance patterns, the availability of newer antimicrobial agents, and increasing emphasis on cost effectiveness and outpatient management.

For patients with classic complaints of fever and productive cough, the clinical diagnosis of pneumonia is straightforward, especially when accompanied by pulmonary infiltrate on plain chest radiographs. More challenging, however, is identifying pneumonia in patients who present with atypical complaints (eg, abdominal pain). Once pneumonia is diagnosed, the priorities in the ED are to provide appropriate respiratory support, assess the severity of disease, initiate appropriate empiric antibiotic therapy based on the most likely pathogens, and make decisions regarding hospitalization and the need for isolation. Issues for which emergency physicians and infectious disease specialists may have different perspectives include the use of blood and sputum cultures, indications for hospital admission, appropriate level of care, and the breadth of antimicrobial spectrum for empiric therapy.

Diagnostic testing for pneumonia in the emergency department

Cough is a common presenting complaint; however, only a small fraction of patients who present with cough are diagnosed with pneumonia (4% in one large series) [2]. Patients with respiratory complaints should be screened with pulse oximetry at triage because hypoxia may not be otherwise clinically suspected, and its presence is an important diagnostic clue with therapeutic implications [3]. In most healthy older children and adults, the diagnosis of pneumonia can be reasonably excluded on the basis of history and physical examination, with suspected cases further evaluated by chest radiography. Absence of any abnormalities in vital signs or chest auscultation substantially reduces the likelihood of pneumonia. No single clinical finding, however, is highly reliable in establishing or excluding a diagnosis of pneumonia [4].

Chest radiography

Chest radiography is generally the most important test for establishing the diagnosis of pneumonia. Although it is clear that many chest radiographs are performed unnecessarily on 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 [5]. Routine chest radiography for all patients who present with cough is not necessary but may be reserved for patients without a history of asthma who have other suggestive findings (eg, fever, tachycardia, decreased oxygen saturation, or focal abnormality on lung examination) [6]. Among patients who are suspected of having pneumonia, these clinical findings have been prospectively validated and are better predictors of a radiographic infiltrate than physician judgment [7]. Patients with serious underlying disease or severe sepsis/septic shock in whom hospitalization is

considered should have a chest radiograph performed. CT of the chest seems to be more sensitive than plain radiography for detecting the presence of pulmonary consolidation, although the natural history and clinical significance of CT-positive, plain radiograph-negative pneumonia are not clear [8]. CT may play a role in evaluating for other pulmonary diagnoses that may mimic pneumonia, such as pulmonary emboli, and may further delineate the nature of the pneumonia, such as in the case of necrotizing infection associated with community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA).

Young, healthy adults with a presumptive diagnosis of pneumonia who are treated as outpatients may have chest radiography deferred unless there is a suspicion of immunocompromised status, tuberculosis (TB), or other unusual features of disease. Chest radiography should be performed subsequently if there is a poor initial response to treatment. Routine performance of chest radiography for patients with exacerbation of chronic bronchitis or chronic obstructive pulmonary disease is of low yield and may be limited to patients with other signs of infection or congestive failure [9]. Studies of infants with fever show that routine chest radiography is of low yield in the absence of other symptoms or signs of lower respiratory tract infection (eg, cough, rales, or elevated respiratory rate) [10,11]. One study found that leukocytosis was associated with occult pneumonia in children [12], but it is not clear whether identifying these cases has any clinical significance [13].

Rarely, patients with a clinical picture that strongly suggests pneumonia have a normal chest radiograph, and some are found to have an infiltrate noted within the next 24 to 48 hours. The absence of findings on a chest radiograph should not preclude the use of antimicrobial therapy in appropriate patients with a clinical diagnosis of pneumonia [14]. Whether the state of hydration can affect the radiographic appearance of pneumonia is unclear. Although severe dehydration theoretically could result in a diminished exudative response by decreasing blood volume and hydrostatic pressure, this finding has not been demonstrated in experimental models [15,16].

Laboratory studies

Laboratory studies are of limited use for establishing the diagnosis and specific cause of pneumonia. Although the finding of a white blood cell (WBC) count of more than $15,000/mm^3$ increases the probability of the patient having a pyogenic bacterial origin rather than a viral or atypical origin, this finding depends on the stage of the illness and is neither sensitive nor specific enough to aid decisions regarding therapy in an individual patient. A WBC count may be helpful if it reveals evidence of immunosuppression, such as neutropenia or lymphopenia, which may indicate immunosuppression from AIDS. Serum lactate dehydrogenase may be helpful in evaluating possible *Pneumocystis* pneumonia (PCP) in patients known or suspected to have HIV infection. Several rapid HIV tests are available that may be

helpful in this situation [17]. Although CD4 counts usually cannot be obtained within the time frame of an ED visit, total lymphocyte count of less than 1000/mm^3 predicts a higher likelihood of CD4 count less than 200/mm^3 [18]. When suspicion exists for severe sepsis/septic shock, serum chemistry and coagulation studies may be helpful in evaluating patients for metabolic acidosis, renal and hepatic dysfunction, and disseminated intravascular coagulation. In the absence fluid-unresponsive hypotension, elevated arterial and central venous lactic acid levels also may indicate the need for early and aggressive hemodynamic resuscitation and broader empirical antibiotic therapy.

Patients with a pleural effusion should have a diagnostic thoracentesis performed with fluid sent for cell count, differential, pH (pH <7.2 predicts a need for chest tube), Gram stain, and culture. Although it is preferred to obtain pleural fluid specimen as early as possible, because of time constraints it is not always possible to perform diagnostic thoracentesis in every patient with pleural effusion in the ED. For most patients, thoracentesis can be deferred until after hospital admission. Patients in significant respiratory distress or with evidence of tension and mediastinal shift require emergent diagnostic and therapeutic thoracentesis, however. Assessment of respiratory function with pulse oximetry is important in the evaluation of patients with pneumonia. Because clinical assessment of oxygenation can be inaccurate [3], a pulse oximetry reading should be obtained in any patient suspected of having pneumonia in the ED—and ideally upon ED triage. Arterial blood gas measurement is usually unnecessary.

Identifying a specific etiologic agent

Many textbook discussions of pneumonia include clinical features that may predict a specific cause of pneumonia (eg, "currant jelly" sputum and bulging fissure on chest radiograph for *Klebsiella pneumoniae*, bullous myringitis for *Mycoplasma*). These findings have poor predictive value, however, and their presence or absence should not guide empiric therapy. Fortunately, recommended empiric regimens for CAP have activity against the most likely etiologies, so a specific etiologic diagnosis is usually unnecessary.

Pneumonia is often divided into two types: (1) "typical" pneumonia, which is caused by pyogenic bacteria, such as *Streptococcus pneumonia* or *Haemophilus influenzae,* and is characterized by productive cough with purulent sputum, high fever, and lobar consolidation, and (2) "atypical" pneumonia, which is caused by organisms such as *Mycoplasma pneumoniae* and *Chlamydophila pneumoniae* (formerly known as *Chlamydia pneumoniae*), and is characterized by a nonproductive cough with diffuse interstitial infiltrates. This differentiation is somewhat artificial; a clear differentiation between these two types of pneumonia on clinical grounds alone is impossible, and coinfection can occur. Factors studied prospectively and found not to be more frequent with atypical pneumonias than with pyogenic bacterial etiologies include gradual onset,

viral prodrome, absence of rigors, nonproductive cough, lower degree of fever, absence of pleurisy, absence of consolidation, low leukocyte count, and an ill-defined infiltrate on chest radiograph [19].

Although it is impossible to determine with a high degree of certainty the specific cause of pneumonia without results of microbiologic or serologic tests, certain clinical features suggest a specific microbial cause. For example, a patient with known or suspected HIV and diffuse interstitial infiltrates should be evaluated for possible PCP. A severe, necrotizing pneumonia in the setting a previously healthy person with influenza should suggest CA-MRSA [20]. A patient with a history of intravenous drug use and plain chest radiograph with multiple focal infiltrates consistent with septic emboli should be suspected of having endocarditis, which is usually caused by S aureus (including MRSA). Geographic exposure to a current outbreak may lead to suspicion of infections such as avian influenza or severe acute respiratory syndrome.

Radiographic findings are poorly predictive of a particular infectious cause. For example, Mycoplasma pneumonia may present as a dense infiltrate, or pneumococcal pneumonia may present as a diffuse interstitial infiltrate. Immunocompromised patients are particularly prone to having atypical radiographic appearances. Findings such as apical pulmonary infiltrates, hilar adenopathy, and cavitation suggest TB, however, and should prompt initiation of appropriate isolation measures.

Sputum Gram stain and culture

Sputum Gram stain is often recommended as a means to determine the presence of a bacterial pathogen, allowing more specific antimicrobial therapy, but rarely results in a change in therapy or outcome. The routine use of sputum Gram stain as a basis for empiric therapy in the ED can be problematic for several reasons. Many patients are not able to provide an adequate sputum specimen. Induction of sputum without adequate isolation facilities can put patients and staff at risk if sputum is induced from persons with unrecognized TB. Correlation between identification of pneumococcus on Gram stain and sputum culture results is poor, even when commonly used criteria for an adequate sputum specimen (less than five squamous epithelial cells and >25 WBC/high power field) are applied. Gram stains are even less likely to demonstrate gram-negative pathogens, such as H influenzae, and should not be relied on to rule out a gram-negative cause.

Earlier recommendations regarding sputum analysis arose in the era of narrow spectrum antibiotics and may be less important in the era of broader spectrum agents that are given routinely for empirical treatment. Empirical antimicrobial agents are usually highly clinically effective if chosen based on clinical information without sputum analysis. With a high proportion of S pneumoniae strains resistant to penicillin, most physicians would not choose to treat a patient with penicillin even if a well-done sputum Gram stain

revealed a predominance of gram-positive diplococci. The most recent Infectious Disease Society of America/American Thoracic Society (IDSA/ATS) consensus guidelines suggest that more aggressive diagnostic testing can be reserved for the more seriously ill patients. If sputum Gram stain and culture are considered, they should be reserved for the subset of patients with more serious illness (eg, admitted to the intensive care unit [ICU]), in whom the bacteriologic diagnosis is highly uncertain and for whom it is felt that the outcome may depend on optimal antimicrobial therapy. For example, in the ED, patients with pneumonia and respiratory failure who require intubation should have endotracheal suction specimens obtained.

Blood cultures

The need for routine blood cultures among patients admitted for pneumonia is also controversial [21,22]. Routine blood cultures for patients admitted with pneumonia have shown mixed results in terms of improved diagnostic accuracy or ability to guide therapy [23]. Most studies have revealed that the rates of false-positive culture results are similar to true-positive results, and false-positive culture results increase costs and prolong hospital stays [24,25]. In one prospective study of 760 patients with CAP, a change of antibiotic therapy based on blood cultures may have improved clinical outcome in only three cases (0.4%) [26]. Blood cultures should be obtained in seriously ill patients; if drawn, they should be obtained before the initiation of antibiotics (although antibiotics should not be delayed for this reason). When results are positive, blood cultures reflect the etiologic agent more accurately than sputum cultures but still only uncommonly lead to a rational change in antimicrobial therapy. Bacteremia occurs in approximately 25% to 30% of hospitalized pneumococcal pneumonia cases, but the diagnosis and therapy are usually well established before blood culture results are available.

It seems that blood cultures are useful for only a small fraction of admitted pneumonia patients, but it may be difficult to clearly identify who they are. Increasing reports of severe pneumonia caused by CA-MRSA illustrate the importance of pursuing the cause of pneumonia, at least in some cases [20,27,28]. It is reasonable to target patients with more severe illness for two reasons: (1) the incidence of bacteremia is higher [29] and (2) they have more to lose if empiric therapy is inappropriate.

The Joint Commission and the Centers for Medicare and Medicaid Services have removed routine blood cultures for all admitted patients as a quality measure, and the most recent edition of the IDSA/ATS guidelines for management of pneumonia does not recommend routine blood cultures for all admitted patients [1]. The Centers for Medicare and Medicaid Services still include a quality measure for obtaining blood cultures for patients admitted or transferred to the ICU within 24 hours of hospital arrival [30]. Unfortunately, it is often not possible to predict later transfer to ICU when

a patient is admitted from the ED. Infectious disease consultants who see the minority of patients who deteriorate after initial treatment in the hospital may criticize the lack of blood cultures obtained on admission. Perspective from these occasional cases should not lead to a conclusion that all patients admitted with pneumonia need blood cultures. Blood cultures obtained from patients who do not show signs of serious infection at the time of ED evaluation are of lower yield and often give false-positive results (ie, contaminants). The follow-up of false-positive blood culture results can be costly and labor intensive and may lead to unnecessary use of agents such as vancomycin or linezolid when results are initially reported as gram-positive cocci in clusters.

The big decision: hospital or home?

Probably the single most important decision made in the ED is whether to admit a patient to the hospital or discharge home. Inpatient treatment of pneumonia is approximately 25 times more expensive per patient than outpatient treatment, and most patients are more comfortable in a home environment. There is tremendous variability in physician admission decisions for pneumonia. The more common tendency is overestimation of disease severity, which leads to hospitalization of patients at low risk for death or serious complications [31]. Although no firm guidelines exist regarding hospital admission, several well-recognized risk factors are associated with an increased risk of death or a complicated clinical course [32,33]. It is becoming more common practice for many hospitals and managed care systems to use some type of scoring system to assist with decisions regarding hospitalization for patients who have pneumonia.

One commonly used system is based on the Pneumonia Patient Outcomes Research Team study, a prospectively validated predictive rule for mortality among immunocompetent adults with CAP [34]. This model (also known as the pneumonia severity index [PSI]) suggests a two-step approach to assess risk. Patients in the lowest risk class who are recommended for outpatient management are younger than age 50, do not have significant comorbid conditions (eg, neoplasm, congestive heart failure, cerebrovascular disease, renal disease, liver disease), and do not have the following findings on physical examination: altered mental status, pulse 125 beats/min or more, respiratory rate 30 breaths/min or more, systolic blood pressure less than 90 mm Hg, or temperature less than 35°C or 40°C or more. Patients who do not fit the lowest risk category are classified into categories based on a scoring system that takes into account age, comorbid illness, physical examination findings, and laboratory abnormalities (Tables 1 and 2). Hospitalization is recommended for patients with a score more than 91 (class IV-V), and brief admission or observation may be considered for patients with a score of 71 to 90 (class III).

Table 1
Pneumonia severity index: scoring system for pneumonia mortality prediction

Patient characteristics	Points
Demographic factor	
Age	
Male	Years of age
Female	Years of age − 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 finding	
Altered mental status	20
Respiratory rate >30	20
Systolic blood pressure <90 mm Hg	20
Temperature <35°C or >40°C	15
Pulse >125 beats/min	10
Laboratory or radiographic finding	
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

Adapted from Bartlett JG, Dowell SF, Mandell LA, et al. Practice guidelines for the management of community-acquired pneumonia in adults: Infectious Diseases Society of America. Clin Infect Dis 2000;31(2):347–82; with permission.

Although this method of assessing the likelihood of successful outpatient management is helpful in establishing general guidelines, it can be cumbersome to use, has not been modeled to predict acute life-threatening events, does not take into account dynamic evaluation over time, and has many important exceptions (eg, an otherwise low-risk patient with severe hypoxia would be discharged by strict interpretation of this rule). Additional discharge criteria could include improving and stable vital signs over a several-hour observation period, ability to take oral medications, an ambulatory pulse oximetry more than 90%, home support, and ability to follow-up. Good clinical judgment should supersede a strict interpretation of a scoring system. A study in which physicians were educated and provided the patient's risk score, however, revealed a significantly lower overall admission rate, cost savings, and similar quality-of-life scores compared with patients conventionally managed by their physicians [35]. Another study randomized patients in PSI class II-III to admission or discharge home and found that outcomes such as mortality and hospital readmission were similar, with higher patient satisfaction among outpatients [36]. EDs

Table 2
Risk classes and 30-day mortality rates for the pneumonia severity index

Risk class	Points	Mortality
I		0.1%
II	≤70	0.6%
III	71–90	0.9%
IV	91–130	9.3%
V	>130	27%

Data from Fine MJ, Auble TE, Yealy DM, et al. A prediction rule to identify low-risk patients with community-acquired pneumonia. N Engl J Med 1997;336(4):243–50.

that use higher intensity efforts to implement guidelines, including PSI, have lower admission rates for low-risk patients and higher compliance with antibiotic recommendations [37].

A similar tool that is easier to use is known as the CURB-65 rule [38]. This rule uses only five simple criteria to determine patients at lower risk for adverse events: Confusion, Uremia (blood urea nitrogen ≥20 mg/dL), Respiratory rate 30 breaths/min or more, Blood pressure less than 90 mm Hg systolic or 60 mm Hg or less diastolic, and age 65 or more. The risk of 30-day mortality increases with a greater number of these factors present: 0.7% with no factors, 9.2% with two factors, and 57% with five factors. It is recommended that patients with zero to one feature receive outpatient care, patients with two features be admitted, and ICU level care be considered for patients with three or more factors. No randomized trials of hospital admission strategies have directly compared the PSI to the CURB-65 score. In a comparison of scores in the same population of CAP patients, the PSI gave a slightly higher percentage of patients in the low-risk category, with a similar low mortality rate [39].

The disposition of HIV-infected patients with possible PCP is dictated by the likelihood of progression to severe disease and by the feasibility of close outpatient follow-up. Factors associated with decreased survival in patients who have AIDS and PCP include history of prior PCP, elevated respiratory rate, abnormal chest examination, WBC count more than 10,300/mm^3, elevated lactate dehydrogenase, hypoxemia, hypoalbuminemia, and abnormal chest radiograph [40]. Patients without multiple poor prognostic factors or hypoxia may be discharged from the ED with close outpatient follow-up, ideally within 2 to 3 days.

The decision to hospitalize a patient with pneumonia is not necessarily a commitment to prolonged inpatient care. Twelve- to 24-hour ED or hospital ward observation may allow the early discharge of some patients. Other strategies sometimes used in the ED for patients with borderline indications for hospitalization include an initial parenteral dose of a longer half-life antibiotic, such as ceftriaxone, with a brief (eg, 2- to 6-hour) observation period. There are no evidence-based guidelines to identify which types of patients may be best managed with this strategy. Because these patients receive

parenteral antibiotic therapy that would be equivalent to the antimicrobial component of inpatient care for the first day, it seems appropriate for inter-mediate-risk patients.

Level of care

In some cases it is obvious that a patient requires admission to an ICU, including patients who are intubated or require vasopressors for hemody-namic stabilization. It is more difficult to identify patients who do not require these interventions initially but may be at greater risk for deteriora-tion and require a level of monitoring that may be beyond what is available on the typical hospital ward. Up to 45% of patients with CAP who ulti-mately require ICU admission are initially admitted to a non-ICU setting [41]. Transfer to the ICU for delayed onset of respiratory failure or septic shock is associated with increased mortality [42]. Defining "severe" pneu-monia also has implications for empiric antimicrobial selection. Most stud-ies of "severe" pneumonia have simply defined it as pneumonia in a patient admitted to the ICU. 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 these criteria were retrospectively studied in a cohort of CAP patients, they did not perform better than actual physi-cian decisions [43]. The 2007 IDSA/ATS guidelines include criteria for defin-ing severe CAP that are based on ATS minor criteria and CURB variables (Table 3) [1]. They recommend that either of the two major criteria is an in-dication for ICU admission and that presence of at least three minor criteria would indicate a need for ICU admission. They also acknowledge that pro-spective validation of these criteria is needed.

Physicians may prefer to "err on the side of caution" and admit lower-acuity patients to a higher level of care rather than risk later ICU transfer from the hospital ward. It is important to recognize that this practice comes with a cost that may put other patients at higher risk for morbidity and mor-tality, however. Many ICUs in US hospitals are operating at capacity or near capacity, and seriously ill patients are spending more time boarding in EDs when no beds are available. ED overcrowding has adverse impacts that include ambulance diversions and longer transport times, longer wait-ing times for patients (some of whom have serious illness that cannot be rec-ognized until they are evaluated by a physician), and lower overall quality of care [44]. Transfer of a deteriorating patient to the ICU from the hospital ward is not necessarily a failure. Some of the appropriate reasons for admit-ting patients to the hospital are to observe them and quickly move them to a higher level of care if necessary.

Isolation and infection control

Most patients with CAP do not need respiratory isolation. Patients who are suspected of having a cause of pneumonia that could pose a threat of

Table 3
Criteria for severe community-acquired pneumonia

Minor criteria[a]
  Respiratory rate[b] ≥ 30 breaths/min
  PaO_2/FiO_2 ratio[b] ≤ 250
  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, $<36°C$)
  Hypotension requiring aggressive fluid resuscitation
Major criteria
  Invasive mechanical ventilation
  Septic shock with the need for vasopressors

Abbreviations: BUN, blood urea nitrogen; PaO_2/FiO_2, arterial oxygen pressure/fraction of inspired oxygen.

[a] Other criteria to consider include hypoglycemia (in patients who do not have diabetes), acute alcoholism/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 >30 breaths/min or a PaO_2/FiO_2 ratio <250.

[c] As a result of infection alone.

Adapted from Mandell LA, Wunderink RG, Anzueto A, et al. Infectious Diseases Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. Clin Infect Dis 2007;44(Suppl 2):S27–72; with permission.

transmission to other patients (eg, influenza, varicella, TB, plague) should be isolated. Isolation should be instituted as early as possible in the ED [45]. Patients who have neutropenia are generally placed in reverse isolation. The ED is a high-risk area for transmission of TB [46]. In many public hospitals, most patients who have pulmonary TB initially present through the ED [47]. Patients at high risk for TB, such as homeless persons, substance abusers, immigrants, and medically underserved low-income populations, frequently use the ED for health care [48]. Patients with TB risk factors often are cared for at busy public hospitals with long waiting times and crowded waiting rooms, which increases risk of health care transmission. Most US EDs do not have TB isolation facilities that comply with recommendations of the Centers for Disease Control and Prevention [49].

Patients suspected of possible pulmonary TB because of a history of TB exposure, suggestive symptoms (eg, persistent cough, weight loss, night sweats, hemoptysis), or belonging to a group at high risk for TB (eg, homelessness, intravenous drug use, alcoholism, HIV risk, immigration from high-risk area) should be placed immediately into respiratory isolation until active TB can be ruled out by further evaluation, including chest radiography [50]. Several published prediction models have attempted to assist clinicians with deciding which patients require TB isolation [51–53]. These

models are limited by the small number of patients included with TB, however, and some are complex point-assignment models that are not easily applied in the busy ED setting. A systematic review of nine clinical prediction rules for isolating inpatients with suspected TB found that self-reported TB skin test results and upper lobe chest radiograph abnormalities demonstrated the strongest association with the diagnosis of TB [54].

HIV-infected patients who present with pneumonia ideally should be isolated until TB can be evaluated by sputum smears for acid-fast bacilli, particularly individuals with other risk factors for TB. Chest radiography cannot be relied on to exclude TB in patients who have AIDS because it often demonstrates diffuse infiltrates as opposed to characteristic cavitary lesions. Depending on individual risk assessment, other patients with noncavitary pulmonary infiltrates, such as inner-city homeless persons or intravenous drug users, may need to be isolated for possible TB. EDs that frequently care for patients at risk for TB should adopt triage protocols to rapidly identify these individuals and get masks and expedited chest radiographs before patients, visitors, or staff are unnecessarily exposed.

Empiric antimicrobial treatment of pneumonia in the emergency department

Timing of antimicrobial therapy

As with any seriously ill patient in the ED, initial attention should focus on ensuring adequate oxygenation, ventilation, and perfusion. Patients with underlying asthma or chronic obstructive pulmonary disease who present in respiratory distress may benefit from bronchodilator therapy and corticosteroids. Seriously ill patients who present with severe sepsis or septic shock require fluid resuscitation and vasopressors [55]. In the ED, empiric antimicrobial therapy for pneumonia is started before a definite microbiologic cause is established. For seriously ill patients who require hospital admission, antimicrobial therapy should be initiated as soon as possible once a reasonable suspicion of pneumonia exists because timely administration of antimicrobial agents has been shown to improve outcomes for hospitalized CAP patients [56].

The Centers for Medicare and Medicaid Services and the Joint Commission established administration of antibiotics within 4 hours of ED presentation for adults admitted to the hospital for CAP as a quality measure. This policy was largely based on a 2004 study that showed that administration of antimicrobial agents within 4 hours of hospital arrival was associated with lower mortality and reduced length of stay for Medicare patients over age 65 [57]. For various reasons, many facilities have had difficulty meeting this standard in a high proportion of patients. EDs in many US cities face critical overcrowding issues, and patients with pneumonia who do not appear seriously ill at triage may spend hours in the waiting room before

evaluation. Because the "clock starts ticking" when the patient arrives at triage, the 4-hour time limit may pass by the time a patient is evaluated by a physician. The diagnosis of pneumonia is not always straightforward, and even minimal diagnostic testing in the ED may put a patient beyond the 4-hour window. Much like the problem of retrospective judgment, case reviews based on the final hospital diagnosis of pneumonia are biased toward overestimating therapeutic delays of patients who initially appear to have other conditions, such as congestive heart failure [58,59]. Some facilities have even resorted to a policy of giving antibiotics upon arrival for any patient with respiratory complaints that might be possibly caused by pneumonia. This strategy may improve compliance with the standard but obviously leads to many patients receiving unnecessary antibiotics [60,61].

The most recent IDSA/ATS CAP treatment guidelines state that the first dose should be given in the ED (preferably within 6–8 hours of arrival to the ED) but do not designate a specific time threshold [1]. Giving the first dose in the ED rather than on arrival to the hospital ward is associated with more rapid time to first dose of antibiotic and shorter length of hospital stay [62]. A rush to treatment without a diagnosis of CAP can result in inappropriate antibiotic use, however.

Choice of antimicrobial agents

The antibiotics chosen should provide coverage of the likely causes based on clinical, laboratory, radiologic, and epidemiologic information. Although it is not possible to predict a specific cause of pneumonia with a high degree of accuracy, it is possible to choose empiric therapy that covers the likely pathogens without being unnecessarily broad spectrum. For most older children and adults with CAP, it is appropriate to choose empiric regimens with activity against *S pneumoniae*, *H influenzae*, and atypical organisms such as *C pneumoniae* and *M pneumoniae*. Recommendations for empiric therapy for CAP in adults are summarized in Tables 4 and 5.

CA-MRSA has rapidly emerged as the most common pathogen isolated in community-acquired skin and soft-tissue infections [63] and is increasingly recognized as a cause of CAP. CA-MRSA pneumonia typically presents as a severe, rapidly progressing pneumonia with sepsis, often in children or healthy young adults [20]. Antimicrobial agents with consistent in vitro activity against CA-MRSA isolates include vancomycin, trimethoprim-sulfamethoxazole, daptomycin, tigecycline, and linezolid. Optimal therapy for MRSA is a subject of current debate in light of increasing minimum inhibitory concentrations for vancomycin [64,65]. Post hoc subgroup analysis of clinical trials for health care–associated pneumonia found that linezolid treatment in the subgroups found to have MRSA was associated with improved survival compared with vancomycin [66]. Prospective trials are ongoing to determine whether a difference truly exists. Daptomycin is inactivated by pulmonary surfactant, so it would not be appropriate for

Table 4
Community-acquired pneumonia in adults: inpatient antimicrobial treatment

Clinical setting	Antibiotic regimen	Comments
Community-acquired, nonimmunocompromised	Ceftriaxone, 1 g, every 24 h + azithromycin, 500 mg, every 24 h IV or orally Respiratory fluoroquinolone (levofloxacin, 750 mg, IV every 24 h, or moxifloxacin, 400 mg, IV every 24 h)	Could substitute cefotaxime, ampicillin-sulbactam, or ertapenem for ceftriaxone Treats most common bacterial and atypical pathogens Active versus DRSP
Severe pneumonia (ICU)	Ceftriaxone, 1g IV every 24 h + levofloxacin, 750 mg, IV every 24 h + vancomycin, 1g, IV every 12 h	Can substitute cefotaxime, cefepime, ertapenem, or β-lactam/β-lactamase inhibitor for ceftriaxone Can substitute moxifloxacin for levofloxacin Can substitute linezolid for vancomycin
Severe pneumonia with neutropenia, bronchiectasis, or recent hospitalization (risk for *Pseudomonas*)	Cefepime, 2 g, IV every 12 h + ciprofloxacin, 400 mg, IV every 12 h + vancomycin, 1g, IV every 12 h	Can substitute other antipseudomonal β-lactam, such as piperacillin-tazobactam, imipenem, or meropenem for cefepime Can substitute aminoglycoside plus macrolide for ciprofloxacin
Presumed *Pneumocystis* pneumonia	Trimethoprim-sulfamethoxazole, 160/800 mg IV every 6 h	Add ceftriaxone to TMP/SMX, if severe, until PCP confirmed Alternatives for sulfa allergy include pentamidine + third-generation cephalosporin; clindamycin + primaquine; atovaquone + ceftriaxone

Doses are for 70-kg adult with normal renal and hepatic function.
Abbreviations: DRSP, drug-resistant *S pneumoniae*; IV, intravenously.

empiric therapy of pneumonia. Although it is not necessary to provide empiric coverage of MRSA for all pneumonia cases, it should be strongly considered for patients with severe pneumonia associated with sepsis, especially persons with concurrent influenza, contact with someone infected with MRSA, or radiographic evidence of necrotizing pneumonia.

Patients in nursing homes or other extended care facilities are often brought to the ED when they develop an acute problem, such as dyspnea or fever. Depending on their level of activity, comorbid conditions, history

Table 5
Community-acquired pneumonia in adults: outpatient treatment

Clinical setting	Antibiotic regimen	Comments
Previously healthy, no antimicrobials in last 3 months	Doxycycline, 100 mg orally, twice a day	Preferred for adolescent/ young adult when likelihood of mycoplasma is high; variable activity versus *S pneumoniae*
	Azithromycin	Treats common typical bacterial and atypical pathogens
		Variety of dosing regimens: 500 mg once followed by 250 mg daily for 4 days; 500 mg orally daily for 3 days; 2 g orally extended-release suspension once
		Can substitute clarithromycin
Comorbidities or antimicrobials in last 3 months	Levofloxacin, 750 mg orally, daily for 5 days	Can substitute moxifloxacin or gemifloxacin
		Treats common typical and atypical bacterial pathogens; active versus DRSP Use if recently received β-lactam or macrolide
	Cefpodoxime, 200 mg orally, twice a day + azithromycin, 500 mg orally, daily	Use if recently received fluoroquinolones
		Can substitute cefdinir, cefprozil, or amoxicillin/ clavulanate for cefpodoxime Variable activity against DRSP

Doses are for 70-kg adult with normal renal and hepatic function.
Abbreviation: DRSP, drug-resistant *S pneumoniae*.

of prior antibiotic use, and hospitalization, these patients are at increased risk for infection with resistant organisms such as *Pseudomonas aeruginosa, K pneumoniae* (including strains producing extended spectrum β-lactamases), *Acinetobacter* species, and hospital-associated strains of MRSA. Other risk factors for infection with multidrug-resistant pathogens include (1) hospitalization for 2 or more days in an acute care facility within 90 days of infection, (2) attending a hemodialysis clinic, and (3) receiving intravenous antibiotic therapy, chemotherapy, or wound care within 30 days of infection. Any patient with pneumonia that fulfills any of these historical features, including patients from a nursing home or long-term care facility,

is designated as having health care–associated pneumonia, which is associated with a greater likelihood of resistant pathogens, such as *Pseudomonas* and MRSA. Mortality is also higher than with CAP [67]. It is appropriate to give broader spectrum empiric therapy to patients with health care–associated pneumonia, usually with a combination of antimicrobial agents to increase the chance that at least one antibiotic is active against the causative pathogen. Appropriate combinations include an antipseudomonal β-lactam agent, such as piperacillin/tazobactam, cefepime, imipenem or meropenem, combined with either an aminoglycoside or a fluoroquinolone and vancomycin or linezolid to cover for MRSA [68].

Studies of antiviral agents for influenza have generally focused on uncomplicated cases, but the impact of treatment on patients hospitalized with influenza or bacterial complications of influenza is less clear. It is reasonable to add antiviral treatment for pneumonia patients with positive antigen or culture-positive influenza or begin empiric treatment in patients with compatible clinical findings when influenza is in the community [1]. Neuraminidase inhibitors, such as oseltamivir, are a better choice than amantadine and rimantadine because they are active against influenza A and B and because many strains currently circulating in the United States are resistant to these older agents [69,70].

Patients who have HIV present an extra challenge because of possible risk for opportunistic pathogens. Because of the potential toxicity of sulfamethoxazole/trimethoprim (TMP/SMX), empiric treatment with this agent for well-appearing outpatients with a low probability of PCP is generally not recommended. An empiric trial of a macrolide may be indicated for treatment of mild CAP in a patient at low risk for PCP (eg, recent CD4 count $> 350/\text{mm}^3$). Any deterioration on outpatient oral antibiotics should prompt admission for a more extensive evaluation. Some emergency physicians initiate oral outpatient therapy with TMP/SMX or an alternate drug for patients with a high probability of PCP and favorable clinical parameters, but this should only be done if a patient can be followed closely for continued diagnostic evaluation and observation for toxicity. It is best done in consultation with the patient's continuing care physician.

It is common practice to initiate outpatient therapy in moderately ill patients for whom hospitalization might be considered, with administration of an initial parenteral dose of a long-acting antibiotic such as ceftriaxone (plus an initial dose of macrolide) and extended observation (ie, 12–24 hours) while administering supportive care such as hydration, antipyretics, and bronchodilators before discharge on an oral regimen. Certain patients also might be brought back to the ED for follow-up in 24 hours, either in person or by telephone. An outpatient regimen of an oral respiratory fluoroquinolone is another option that may be advantageous for moderately ill patients who are considered borderline for hospitalization. These agents have more reliable activity against drug-resistant *S pneumoniae* and good oral absorption that provides serum levels comparable to parenteral therapy [71].

Summary

Emergency physicians encounter a spectrum of pneumonia patients that is different than that encountered by infectious disease specialists. Most patients who have pneumonia and present to the ED can be safely discharged home on empiric oral antimicrobial agents with minimal diagnostic testing. Emergency physicians must be skilled at identifying patients who may require more extensive diagnostic testing, have severe sepsis/septic shock, are at risk for opportunistic infections such as PCP, and for whom empiric therapy should be expanded to cover less common organisms, such as CA-MRSA. Empiric antibiotics should be initiated in the ED, but using arbitrary time cutoffs for initiating antibiotics as a quality measure for patients ultimately diagnosed as having pneumonia is problematic. Decisions regarding hospital admission and level of care are central to emergency medicine practice and can be aided with prognostic models. It is also important to initiate infection control measures in the ED when appropriate.

References

[1] Mandell LA, Wunderink RG, Anzueto A, et al. Infectious Diseases Society of America/ American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. Clin Infect Dis 2007;44(Suppl 2):S27–72.

[2] Metley JP, Stafford RS, Singer DE. National trends in the use of antibiotics by primary care physicians for adult patients with cough. Arch Intern Med 1998;158(16):1813–8.

[3] Mower WR, Sachs C, Nicklin EL, et al. Effect of routine emergency department triage pulse oximetry screening on medical management. Chest 1995;108(5):1297–302.

[4] Metlay JP, Kapoor WN, Fine MJ. Does this patient have community-acquired pneumonia? JAMA 1997;278(17):1440–5.

[5] Singal BM, Hedges JR, Radack KL. Decision rules and clinical prediction of pneumonia: evaluation of low yield criteria. Ann Emerg Med 1989;18(1):13–20.

[6] Heckerling PS, Tape TG, Wigton RS, et al. Clinical prediction rule for pulmonary infiltrates. Ann Intern Med 1990;113(9):664–70.

[7] Emerman CL, Dawson N, Speroff T, et al. Comparison of physician judgment and decision aids for ordering chest radiographs for pneumonia in outpatients. Ann Emerg Med 1991; 20(11):1215–9.

[8] Syrjala H, Broas M, Suramo I, et al. High-resolution computed tomography for the diagnosis of community-acquired pneumonia. Clin Infect Dis 1998;27(2):358–63.

[9] Sherman S, Skoney JA, Ravikrishnan KP. Routine chest radiographs in exacerbations of chronic obstructive pulmonary disease: diagnostic value. Arch Intern Med 1989;149(11):2493–6.

[10] Bramson RT, Meyer TL, Silbiger ML, et al. The futility of the chest radiograph in the febrile infant without respiratory symptoms. Pediatrics 1993;92(4):524–6.

[11] Baraff LJ. Management of fever without source in infants and children. Ann Emerg Med 2000;36(6):602–14.

[12] Bachur R, Perry H, Harper MB. Occult pneumonias: empiric chest radiographs in febrile children with leukocytosis. Ann Emerg Med 1999;33(2):166–73.

[13] Green SM, Rothrock SG. Evaluation styles for well-appearing febrile children: are you a "risk-minimizer" or a "test-minimizer"? Ann Emerg Med 1999;33(2):211–4.

[14] Melbye H, Berdal BP, Straume B, et al. Pneumonia: a clinical or radiographic diagnosis? Scand J Infect Dis 1992;24(5):647–55.

[15] Hall FM, Simon M. Occult pneumonia associated with dehydration: myth or reality? Am J Radiology 1987;148(5):853–4.

[16] Caldwell A, Glauser FL, Smith WR, et al. The effects of dehydration on the radiologic and pathologic appearance of experimental canine segmental pneumonia. Am Rev Respir Dis 1975;112(5):651–6.

[17] Centers for Disease COntrol and Prevention. General and laboratory considerations: rapid HIV tests currently available in the United States. Available at: http://www.cdc.gov/hiv/topics/testing/resources/factsheets/rt-lab.htm. Accessed October 1, 2007.

[18] Blatt SP, Lucey CR, Butzin CA, et al. Total lymphocyte count as a predictor of absolute CD4+ count and CD4+ percentage in HIV-infected persons. JAMA 1993;269(5):622–6.

[19] Fang GD, Fine M, Orloff J, et al. New and emerging etiologies for community-acquired pneumonia with implications for therapy: a prospective multicenter study of 359 cases. Medicine 1990;69(5):307–16.

[20] Centers for Disease Control and Prevention. Severe methicillin-resistant *Staphylococcus aureus* community-acquired pneumonia associated with influenza: Louisiana and Georgia, December 2006–January 2007. MMWR Morb Mortal Wkly Rep 2007;56(14):325–9.

[21] Moran GJ, Abrahamian FM. Blood cultures for pneumonia: can we hit the target without a shotgun? Ann Emerg Med 2005;46(5):407–8.

[22] Walls RM, Resnick J. The Joint Commission on Accreditation of Healthcare Organizations and Center for Medicare and Medicaid Services community-acquired pneumonia initiative: what went wrong? Ann Emerg Med 2005;46(5):409–11.

[23] Kennedy M, Bates DW, Wright SB, et al. Do emergency department blood cultures change practice in patients with pneumonia? Ann Emerg Med 2005;46(5):393–400.

[24] Chalasani NP, Valdecanas MA, Gopal AK, et al. Clinical utility of blood cultures in adult patients with community-acquired pneumonia without defined underlying risks. Chest 1995;108(4):932–6.

[25] Corbo J, Friedman B, Bijur P, et al. Limited usefulness of initial blood cultures in community acquired pneumonia. Emerg Med J 2004;21(4):446–8.

[26] Campbell SG, Marrie TJ, Anstey R, et al. The contribution of blood cultures to the clinical management of adult patients admitted to the hospital with community-acquired pneumonia: a prospective observational study. Chest 2003;123(4):1142–50.

[27] Frazee BW, Salz TO, Lambert L, et al. Fatal community-associated methicillin-resistant *Staphylococcus aureus* pneumonia in an immunocompetent young adult. Ann Emerg Med 2005;46(5):401–4.

[28] Hageman JC, Uyeki TM, Francis JS, et al. Severe community-acquired pneumonia due to *Staphylococcus aureus*, 2003–04 influenza season. Emerg Infect Dis 2006;12(6):894–9.

[29] Mandell LA, Marrie TJ, Grossman RF, et al. Canadian guidelines for the initial management of community-acquired pneumonia: an evidence-based update by the Canadian Infectious Diseases Society and the Canadian Thoracic Society. The Canadian Community-Acquired Pneumonia Working Group. Clin Infect Dis 2000;31(2):383–421.

[30] Agency for Healthcare Research and Quality. National quality measures clearinghouse: pneumonia. Available at: http://www.qualitymeasures.ahrq.gov/summary/summary.aspx?doc_id=9499. Accessed October 1, 2007.

[31] McMahon LF Jr, Wolfe RA, Tedeschi PJ. Variation in hospital admission among small areas: a comparison of Maine and Michigan. Med Care 1989;27(6):623–31.

[32] Fine MJ, Smith DN, Singer DE. Hospitalization decision in patients with community-acquired pneumonia: a prospective cohort study. Am J Med 1990;89(6):713–21.

[33] Black ER, Mushlin AI, Griner PF, et al. Predicting the need for hospitalization of ambulatory patients with pneumonia. J Gen Intern Med 1991;6(5):394–400.

[34] Fine MJ, Auble TE, Yealy DM, et al. A prediction rule to identify low-risk patients with community-acquired pneumonia. N Engl J Med 1997;336(4):243–50.

[35] Marrie TJ, Lau CY, Wheeler SL, et al. A controlled trial of a critical pathway for treatment of community-acquired pneumonia. JAMA 2000;283(6):749–55.

[36] Carratala J, Fernandez-Sabe N, Ortega L, et al. Outpatient care compared with hospitalization for community-acquired pneumonia: a randomized trial in low-risk patients. Ann Intern Med 2005;142(3):165–72.

[37] Yealy DM, Auble TE, Stone RA, et al. Effect of increasing the intensity of implementing pneumonia guidelines: a randomized, controlled trial. Ann Intern Med 2005;143(12):881–94.

[38] Lim WS, van der Eerden MM, Liang R, et al. Defining community-acquired pneumonia severity on presentation to hospital: an international derivation and validation study. Thorax 2003;58(5):377–82.

[39] Aujesky D, Auble TE, Yealy DM, et al. Prospective comparison of three validated prediction rules for prognosis in community-acquired pneumonia. Am J Med 2005;118(4):384–92.

[40] Masur H. Prevention and treatment of Pneumocystis pneumonia. N Engl J Med 1992; 327(26):1853–60.

[41] Ewig S, de Roux A, Bauer T, et al. Validation of predictive rules and indices of severity for community-acquired pneumonia. Thorax 2004;59(5):421–7.

[42] Leroy O, Santre C, Beuscart C, et al. A five-year study of severe community-acquired pneumonia with emphasis on prognosis in patients admitted to an intensive care unit. Intensive Care Med 1995;21(1):24–31.

[43] Angus DC, Marrie TJ, Obrosky DS, et al. Severe community-acquired pneumonia: use of intensive care services and evaluation of American and British Thoracic Society Diagnostic criteria. Am J Respir Crit Care Med 2002;166(5):717–23.

[44] Committee on the Future of Emergency Care in the United States Health System. Hospital-based emergency care: at the breaking point. Washington, DC: National Academies Press; 2006.

[45] Rothman RE, Irvin CB, Moran GJ, et al. Respiratory hygiene in the emergency department. Ann Emerg Med 2006;48(5):570–82.

[46] Sokolove PE, Mackey D, Wiles J, et al. Exposure of emergency department personnel to tuberculosis: PPD testing during an epidemic in the community. Ann Emerg Med 1994;24(3): 418–21.

[47] Moran GJ, McCabe F, Morgan MT, et al. Delayed recognition and infection control for tuberculosis patients in the emergency department. Ann Emerg Med 1995;26(3):290–5.

[48] Baker DW, Stevens CD, Brook RH. Regular source of ambulatory care and medical care utilization by patients presenting to a public hospital emergency department. JAMA 1994; 271(24):1909–12.

[49] Moran GJ, Fuchs MA, Jarvis WR, et al. Tuberculosis infection-control practices in United States emergency departments. Ann Emerg Med 1995;26(3):283–9.

[50] Centers for Disease Control and Prevention. Guidelines for preventing the transmission of Mycobacterium tuberculosis in health-care facilities, 1994. MMWR Recomm Rep 1994; 43(RR-13):1–132.

[51] El-Solh A, Mylotte J, Sherif S, et al. Validity of a decision tree for predicting active pulmonary tuberculosis. Am J Respir Crit Care Med 1997;155(5):1711–6.

[52] Tattevin P, Casalino E, Fleury L, et al. The validity of medical history, classic symptoms, and chest radiographs in predicting pulmonary tuberculosis. Chest 1999;115(5):1248–53.

[53] Wisnivesky JP, Henschke C, Balentine J, et al. Prospective validation of a prediction model for isolating inpatients with suspected pulmonary tuberculosis. Arch Intern Med 2005; 165(4):453–7.

[54] Wisnivesky JP, Serebrisky D, Moor C, et al. Validity of clinical prediction rules for isolating inpatients with suspected tuberculosis: a systematic review. J Gen Intern Med 2005;20(10): 947–52.

[55] Nguyen HB, Rivers EP, Abrahamian FM, et al. Severe sepsis and septic shock: review of the literature and emergency department management guidelines. Ann Emerg Med 2006;48(1): 28–54.

[56] Meehan TP, Fine MJ, Krumholz HM, et al. Quality of care, process, and outcomes in elderly patients with pneumonia. JAMA 1997;278(23):2080–4.

[57] Houck PM, Bratzler DW, Nsa W, et al. Timing of antibiotic administration and outcomes for Medicare patients hospitalized with community-acquired pneumonia. Arch Intern Med 2004;164(6):637–44.

[58] Pines JM, Morton MF, Datner EM, et al. Systematic delays in antibiotic administration in the emergency department for adult patients admitted with pneumonia. Acad Emerg Med 2006;13(9):939–45.

[59] Fee C, Weber EJ. Identification of 90% of patients ultimately diagnosed with community-acquired pneumonia within four hours of emergency department arrival may not be feasible. Ann Emerg Med 2007;49(5):553–9.

[60] Pines JM, Hollander JE, Lee H, et al. Emergency department operational changes in response to pay-for-performance and antibiotic timing in pneumonia. Acad Emerg Med 2007;14(6):545–8.

[61] Kelen GD, Rothman RE. Community pneumonia practice standard mandates: can't see the forest for the trees. Acad Emerg Med 2006;13(9):986–8.

[62] Battleman DS, Callahan M, Thaler HT. Rapid antibiotic delivery and appropriate antibiotic selection reduce length of hospital stay of patients with community-acquired pneumonia. Arch Intern Med 2002;162(6):682–8.

[63] Moran GJ, Krishnadasan A, Gorwitz RJ, et al, for The EMERGEncy ID NET Study Group. Methicillin-resistant *S. aureus* infections among patients in the emergency department. N Engl J Med 2006;355(7):666–74.

[64] Mohr JF, Murray BE. Point: vancomycin is not obsolete for the treatment of infection caused by methicillin-resistant *Staphylococcus aureus*. Clin Infect Dis 2007;44(12):1536–42.

[65] Deresinski S. Counterpoint: vancomycin and *Staphylococcus aureus*. An antibiotic enters obsolescence. Clin Infect Dis 2007;44(12):1543–8.

[66] Wunderink RG, Rello J, Cammarate SK, et al. Linezolid vs. vancomycin: analysis of two double-blind studies of patients with methicillin-resistant *Staphylococcus aureus* nosocomial pneumonia. Chest 2003;124(5):1789–97.

[67] Kollef MH, Shorr A, Tabak YP, et al. Epidemiology and outcomes of health-care-associated pneumonia: results from a large US database of culture-positive pneumonia. Chest 2005;128(6):3854–62.

[68] American Thoracic Society. Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. Am J Respir Crit Care Med 2005;171(4):388–416.

[69] Gubareva LV, Kaiser L, Hayden FG. Influenza virus neuraminidase inhibitors. Lancet 2000;355(9206):827–35.

[70] Centers for Disease Control and Prevention. High levels of adamantane resistance among influenza A (H3N2) viruses and interim guidelines for use of antiviral agents: United States, 2005–06 influenza season. MMWR Morb Mortal Wkly Rep 2006;55(02):44–6.

[71] Moran GJ. Approaches to treatment of community-acquired pneumonia in the emergency department and the appropriate role of fluoroquinolones. J Emerg Med 2006;30(4):377–87.