Community-acquired pneumonia (CAP) remains a common and often serious infection. Recent studies have suggested that the impact of CAP, particularly in the elderly population, extends beyond the time of the initial infection—in some cases, extending up to 1 year after the initial infection—and is associated with significant mortality [1, 2].

Two major issues associated with the treatment of any patient with CAP are diagnosis and treatment. When presented with a patient who seemingly has CAP, the physician must determine 2 things: is this pneumonia, and what is the pathogen responsible?

The approach to treatment flows from the answers to these questions. The problem however, is that there is currently no "gold standard" for the diagnosis of pneumonia, and the physician usually does not know (with any degree of certainty) what the etiologic agent is at the time that the therapeutic decision is made. For these reasons, initial treatment is usually empirical in nature rather than directed against a specific pathogen.

In an attempt to address this problem and to optimize the care of patients with CAP, a number of societies around the world have developed guidelines for the treatment of CAP. Beyond the immediate and obvious benefit of providing physicians with an approach to the initial treatment of these patients, some of the guidelines have helped to highlight the gaps and deficiencies in our knowledge about this disease, thereby helping to direct future studies. The guidelines have also codified our treatment of patients with CAP and have resulted in a reduction in mortality, length of hospital stay, and cost [3].

What is essential to the development of any CAP guideline is a knowledge of the pathogens that must be treated, as well as their antimicrobial susceptibility patterns. The article by Charles et al. [4] in this issue of *Clinical Infectious Diseases* is an attempt to gather Australian data on the etiology, usefulness of severity assessment tools, and treatment outcomes for CAP. The authors are to be congratulated on their carefully performed study and on the interesting article that they have produced. This is the sort of study—particularly from the point of view of determining etiology—that should ideally be performed in other countries, so that local data can be established. There are, however, a number of questions that come to mind in reading their article.

An obvious question is why patients with cases classified as Pneumonia Severity Index (PSI) class I–III or in CURB-65 group 1 were admitted to the hospital in the first place? These patients constitute 46%—almost one-half—of the study population. In North America, both the Infectious Diseases Society of America–American Thoracic Society (IDSA-ATS) guidelines and the Canadian Infectious Diseases Society–Canadian Thoracic Society (CIDS-CTS) guidelines recommend treatment of such patients on an outpatient basis [5, 6]. Also, the PSI is not a true measurement of severity and was designed and validated primarily as a means of identifying low-risk patients who do not require hospital care for CAP [7].

In the discussion section, the authors state that the recent guidelines from the IDSA-ATS “have promoted the use of respiratory fluoroquinolones” (p. 1519). However, a careful reading of the guidelines indicates that this is, in fact, not the case. The fluoroquinolones are important drugs and certainly have a role to play in the management of CAP, but their role is a fairly selective one. Depending on such variables as comorbidity or recent use of certain antibiotics, the 46% of patients with PSI class I–III in the Australian trial could have been treated either with a macrolide alone or with a β-lactam (e.g.,...
amoxicillin) plus a macrolide, had the
IDSA-ATS guidelines been followed.

This leads to the next obvious question. Given that 250 episodes (or 27% of all episodes in the study) were classified as only PSI class I or II, why was mono-
therapy with a macrolide or possibly dox-
cycline never used? Although data are provided on the susceptibility of pneum-
occci to penicillin, we are not given any information regarding susceptibilities to
macrolides or doxycycline.

In North America, ~80% of patients with CAP are treated as outpatients, and
20% are treated as inpatients. The majority of inpatients are handled in a hospital ward, and ~10% of those who are admitted to the hospital are treated in the intensive care unit. For ward patients, the IDSA-ATS guidelines recommend therapy involving either a respiratory fluoroquinolone or a β-lactam plus a macrolide. The β-lactam can be a nonpseudomonal third-
generation cephalosporin (e.g., cefotaxime or ceftriaxone), ampicillin, or the carba-
penem ertapenem. Preference is not given to either regimen, and in fact, my col-
leagues and I have stated that “the major discriminating factor between the 2 regi-
mens is the patient’s prior antibiotic ex-
posure within the past three months” [5, p. S48]. The 3-month period is based on
data that showed that antibiotic use in the previous 3 months could have a significant impact on the likelihood of infection with a strain of Streptococcus pneumoniae that is resistant to the previously used drug, particularly if the drug was a macrolide with a long half-life or a fluoroquinolone [8]. The findings of an association be-
tween prior clarithromycin use and an increased likelihood of erythromycin resis-
tance and between prior azithromycin use and an increased risk of resistance to mac-
rolides, penicillins, and trimethoprim–sul-
famethoxazole in subsequent infecting strains of S. pneumoniae certainly are not
ringing endorsements for the widespread use of macrolides. A reasonable argument
can be made to administer fluoroquinolo-
ones if only to relieve some of the anti-
biotic selection pressure generated by un-
restricted macrolide use.

If a physician did select a fluoroquin-
olone, the evidence strongly supports such a choice. The use of monotherapy rather
than combination therapy carries with it
obvious logistical and financial benefits,
and the respiratory fluoroquinolones pro-
vide coverage for virtually all of the path-
ogens of concern, including atypical pathogens, such as Mycoplasma or Chla-
mydophila pneumoniae or Legionella spe-
cies; S. pneumoniae, including any peni-
cillin-resistant strains; Haemophilus influenzae; and the Enterobacteriaceae. If
Pseudomonas aeruginosa or community-
acquired methicillin-resistant Staphylococ-
cus aureus are of concern, specific recom-
mandations are provided. Even for
persons who are severely ill with CAP and
who are admitted to an intensive care unit,
a choice of a β-lactam plus either azith-
romycin or a fluoroquinolone is offered if
Pseudomonas infection is not a con-
sideration.

The authors themselves note that their
data may not be generalized to other set-
ings, because many of the patients were
from 3 large urban tertiary care centers. This may well be the case, because another
Australian study of hospitalized patients
with CAP who were admitted to the Royal
Darwin Hospital in Tropical Northern Australia found that the most frequently encountered pathogens were S. pneumoniae and Burkholderia pseudomallei and that “atypical pneumonia organisms were un-
common” [9, p. 15].

Although there is not a comparable North America–based study of the etiol-
ology of CAP to use for comparisons with
the data presented by Charles et al. [4],
some important differences between their
article and the North American experience
come to mind. Neither community-acqui-
quired methicillin-resistant S. aureus nor
P. aeruginosa appeared to be significant pathogens in the Australian article,
whereas both these organisms present
problems in the United States. An in-
creased number of centers are encounter-
ing community-acquired methicillin-re-
sistant S. aureus as a cause of severe CAP,
and in my experience and in a recent study
[10], P. aeruginosa was identified as a path-
ogen in patients with CAP admitted to
both hospital wards and the intensive care
unit.

I wholeheartedly agree with the authors that physicians should ideally avoid reli-
ance on the findings of international stud-
ies when developing treatment guidelines and
should rely instead on data from one’s
own country. Unfortunately, this may not
always be possible and for countries as
large and diverse as Canada and the
United States; such data may simply not
exist. The IDSA-ATS and the CIDS-CTS
guidelines, however, have always exhorted physicians to use the guidelines simply as
a template and to consider local etiological and epidemiological data whenever pos-
sible. The article by Charles et al. [4] is
important because of the information it
provides for use by Australian physicians
and because it helps highlight the potential differences between countries and the
importance of local data.

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