without comorbidities would reduce the likelihood of bias, although even that would not rule out bias completely. It is hardly possible to adjust for this analytically, even when information on why certain microbial tests were (or were not) obtained was available.

Because of global aging and the corresponding increase in the number of comorbidities, which will also occur in patients with CAP, a recommendation to include MDR antibiotic coverage based on comorbidities inevitably increases antibiotic use. In an era of increasing antimicrobial resistance and a high prevalence of nosocomial *Clostridium difficile* infection, such recommendations should be based on unbiased results. Because the cause of CAP cannot be reliably determined by clinical data, empirical antibiotic therapy should be guided by local epidemiologic data, the site of admission, and prior microbial culture results, rather than by the presence of comorbidities.

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Response

To the Editor:

We appreciate the interest of Dr Postma and colleagues in our recently published article in *CHEST*.[1] In the Discussion section of our article, we already acknowledged that a potential limitation of the study is not having 100% microbial etiologies. This study comes from our database, with information collected prospectively over 12 years from patients with community-acquired pneumonia (CAP) who had a well-defined diagnostic and treatment protocol from the very beginning; we disagree with the suggestion that "apparently, microbial testing was left to the discretion of the treating physician."

We are not aware of any CAP study having > 75% etiologies. This is impossible for the following reasons: (1) Blood cultures are poorly sensitive, (2) sputum is often unavailable or contaminated, (3) bronchoscopies cannot be performed in many patients, (4) patients are lost in follow-up (for serologies), and (5) polymerase chain reaction techniques are still not available for all microorganisms and all hospitals. Having said that, we believe that our results are very representative of CAP microbiology in patients older than 65 years of age, given the high number of patients included (2,149).

We do not think that our recommendation will increase the number of antibiotics administered. In fact, we believe the opposite will occur because we restricted our recommendations to (and only included) patients older than 65 years of age.

Finally, we doubt sincerely that site of care is the best approach for empirical treatment in CAP. In fact, patients in the ward who should be admitted to the ICU (particularly with Pneumonia Severity Index V[2]) from the beginning can frequently be observed. Instead, we believe that we have to move forward to treat patients empirically according to severity scales.[3] We thank Dr Postma and colleagues again for their interest in our article.

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Sarcoidosis, Fatigue, and Sleep Apnea

To the Editor:

We read with interest the article by Patterson et al[1] in a recent issue of *CHEST* (June 2013) that demonstrated a high proportion of daytime sleepiness using the Epworth Scale in patients with sarcoidosis. The authors concluded that sleepiness could be a contributing factor to fatigue in some of these patients. The authors are careful to point out that daytime sleepiness is not the same as fatigue, as emphasized by Brown[2] in the accompanying editorial. We recently published a double-blind, placebo-controlled, crossover study examining the treatment of sarcoidosis-associated fatigue

with armodafinil.[3] In that study, we screened all patients for OSA and only included in the study those patients with an apnea/hypopnea index < 6/h. All patients then underwent an overnight sleep study followed by a multiple sleep-latency test before and after each treatment arm.

Despite the absence of OSA, we found one-half of the patients with sarcoidosis had a sleep onset latency of < 8 min, indicative of hypersomnolence. Furthermore, despite using two fatigue instruments, there was no difference in the fatigue severity for those patients with or without a shortened sleep-onset latency. In addition, armodafinil significantly improved fatigue in these patients, including those with objective evidence of hypersomnolence.

Fatigue can be due to several factors in patients with sarcoidosis, including not only sleepiness but also depression, medications, and ongoing inflammation from the disease.[4] However, patients with sarcoidosis may still have marked fatigue without either depression or sleep apnea. For these patients, treatment with stimulants[3,5] may be useful.

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Response

To the Editor:

We thank Dr Lower and colleagues for their comments and their interest in our study.[1] In their study, they demonstrated that treatment with the stimulant armodafinil was associated with a reduction in fatigue in patients with sarcoidosis.[2] A subset of their patients had coexisting fatigue and hypersomnolence, although improvements in fatigue were observed in patients with and without hypersomnolence.

In our study of patients referred for polysomnogram testing, we found that excessive daytime sleepiness, or hypersomnolence, was more common in patients with sarcoidosis compared with control subjects.[1] As we did not measure fatigue, we were not positioned to comment on the relationship between fatigue and sleepiness in our cohort. We agree, though, that these are distinct clinical conditions. Even as they may coexist or have overlapping features, distinguishing between fatigue and excessive daytime sleepiness is clinically relevant. While treatment with stimulants may be beneficial in both, patients with fatigue also warrant an evaluation for comorbidities, which may be targeted for other primary interventions.[2-4]

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Airways Disease Presenting as Restrictive Impairment

A Variant in Asthma, a Defining Feature in World Trade Center Lung Disorder

To the Editor:

The recent article by Berger et al[1] in *CHEST* (July 2013) has refocused the interest of readers of *CHEST* to airway disease