β₂ adrenergic plus inhaled corticosteroids (ICS), although there are no studies to support (or rule out) this clinical practice.

The limited literature to date suggests a small improvement in some clinical parameters with the use of ICS in some patients with bronchiectasis but, as Dr Stirling comments this is effectively accompanied by an increase in the number of local side effects because the relative resistance of neutrophilic inflammation to corticosteroids makes it necessary to use high doses. With the limited evidence available, however, it is not possible to confirm any significant increase in serious adverse effects such as pneumonia or exacerbations in patients with bronchiectasis. In any case, we completely agree with Dr Stirling that primary therapy in bronchiectasis should be addressed to treating chronic bronchial infection, although it is true that, even after intensive antibiotic treatment, many patients continue to present symptomatic airway obstruction and need antiinflammatory and bronchodilator therapy and, in such cases, combined inhaled therapy may be effective. Therefore, the study, which can only be considered a pilot study, as noted in the “Discussion” section in the article, aimed to increase the scientific evidence in the literature for a common therapeutic practice in patients with bronchiectasis, while assessing, to my knowledge for the first time in the literature, whether long-action β₂ adrenergic therapy can reduce the high ICS doses customarily used and, thus, reduce their adverse effects. I agree with Dr Stirling that in patients with bronchiectasis in which bronchial infection is a cornerstone, treatment with high doses of ICS could be harmful, and individualized treatment with close monitoring for adverse effects should be applied.

Really, however, this study¹ sought not only to assess the efficacy of this treatment but also to make the scientific community aware of the current imbalance between the growing importance of bronchiectasis and the limited scientific evidence available. I hope that similar studies increase the scientific and commercial interest in this disease and trigger the multicenter clinical trials that Dr Stirling have called for in order to respond to the basic questions we face every day regarding patients with bronchiectasis. Until this happens, bronchiectasis, although no longer neglected as an epidemiologic disease, will remain on the dark side of science.

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Increased Number of Pulmonary Embolisms in Sarcoidosis Patients
To the Editor:

We read with great interest the article by Swigris et al in a recent issue of CHEST (November 2011), in which the authors found an increased risk of pulmonary embolisms (PEs) among US decedents with sarcoidosis. They found PE in 2.54% of patients’ death certificates, which compares with 1.13% of the background population. A recent observation in our own clinic of two patients with sarcoidosis experiencing extensive PEs triggered our interest in a possible underlying association.

We have conducted a retrospective chart review on treatment outcomes in sarcoidosis. We studied 177 consecutive patients who required immunosuppressive therapy, other than prednisone, between May 2004 and February 2011. Reviewing comorbidities revealed a disproportionally high number of PE (6.2%) in this sarcoidosis cohort (11 of 177 patients).

This PE rate among the patients in our cohort is even higher than the 2.5% mentioned by Swigris et al.¹ However, the percentage of PE decedents with sarcoidosis and the percentage of PE in a consecutive cohort of patients with sarcoidosis cannot be compared accurately. The cohort comprised 107 male patients and 70 female patients (60.5% and 39.5%, respectively). There was no significant difference in the number of PEs between male and female or white and nonwhite patients, as also described by Swigris et al.¹ Included patients had various disease presentations, including pulmonary sarcoidosis, uveitis, and neurosarcoidosis.

Besides the study by Swigris et al¹ and our cohort, one other study found a twofold-higher rate of PE in sarcoidosis patients in a 35-year record linkage study.² The cause for the association between sarcoidosis and PE remains speculative. There might be a role for medication (eg, corticosteroid use) or coexistence of an antiphospholipid syndrome.³ Immunosuppressive therapy may have played a role in our cohort, because this was a selected population with severe sarcoidosis and previous or current use of steroids was higher than in the general sarcoidosis population. However, two patients had PE before they started immunosuppressive therapy. We have insufficient information about antiphospholipid syndrome in our cohort. Among the 11 patients who had PE, there was only one current smoker and three former smokers (<10 pack-years). Smoking is not a well-established risk factor for PE and, therefore, not a likely explanation for the high rate of PE in this cohort.

In conclusion, this is another report suggesting a possible association between PE and sarcoidosis. This confirms the need for further prospective studies into the cause and relative risk of this finding.

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To the Editor:

We appreciate the comments by Dr Vorselaars and colleagues on our article in CHEST1 and congratulate them on completing their chart review. Their review adds to an emerging hypothesis—association between sarcoidosis and thromboembolic phenomenon. We look forward to seeing results from future studies aimed at deciphering this apparent relationship.

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Bedside Ultrasonography for Evaluation of Pneumothorax

To the Editor:

We read with great interest the article by Ding and colleagues2 in a recent issue of CHEST (October 2011). According to their random-effect meta-analysis, pleural ultrasonography (PUS) was more sensitive than, and of similar specificity to, chest radiograph (CXR). They also indicated that PUS is an attractive alternative to CXR in EDs and ICUs and that PUS is a “rule out” test. Although we strongly agree that bedside PUS is very helpful and a readily available tool with very good sensitivity and specificity to detect pneumothorax, we have some concerns.

First, understanding the goal of the study, which was to estimate the accuracy of PUS and CXR from previous studies, we believe that the analysis included a very diverse sample of studies, including retrospective studies with one arm, old studies (some before 1995), some with a low number of subjects (especially on the PUS arm), some with blinding issues, and, in general, studies that included diverse patient populations. In fact, the F statistics indicated very wide heterogeneity. Attempts were made by the authors to explain some of that heterogeneity by doing subgroup analysis and metaregression. It may be better to include recent ICU or ED studies that have a comparison arm with CXR or the gold standard (chest CT scan) and that meet quality criteria (Quality of Diagnostic Accuracy Studies and Delphi criteria) to obtain more accurate estimates.

Second, PUS is not a very accurate method to estimate the pneumothorax volume. If used alone, PUS can lead to overtreatment with a possibly invasive procedure that can potentially cause more harm than benefit.

Third, we agree that PUS can be an alternative to CXR, but only in a small section of pneumothorax patient populations. In addition to the weak ability of PUS to estimate a clinically significant pneumothorax, it is not accurate in patients with large peripheral bullous emphysema. It also has some other limitations mentioned by the authors, such as the presence of pleural adhesions, pleural calcifications, and subcutaneous emphysema.

We believe that the findings of this analysis do not show that PUS should be used alone and universally on all patients. CXR is still needed in patients who are relatively stable with pneumothorax seen on PUS before deciding on chest tube thoracotomy. Furthermore, the training with bedside PUS in the ICU that is native to CXR in EDs and ICUs and that PUS is a “rule out” test.

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