To the Editor:

We appreciate the comments by Dr Vorselaars and colleagues on our article in CHEST and congratulate them on completing their chart review. Their review adds to an emerging hypothesis-generating data pool on the 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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Response

To the Editor:

We read with great interest the article by Ding and colleagues 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 the 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 being incorporated in most Pulmonary, Critical Care, and Emergency Medicine fellowship programs in the United States and familiarity with PUS and pneumothorax signs (lung sliding, lung point, and comet tail) are essential before using PUS to make decisions.

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Response

To the Editor:

We thank Dr Alrajab and colleagues for their comments on our recent article in CHEST.1 Their suggestions and opinions on the key points relating to the diagnosis of pneumothorax (PNX) by ultrasonography will also benefit our research. Our responses to their comments are as follows.

First, the extent to which a Cochrane review can draw conclusions about the effects of an intervention depends on whether the data and results from the included studies are valid. For our analysis, the most valid studies would have been prospective, double-blind studies with a large sample, which would have had the same gold standard for comparison. However, there were only a few studies which complied with these criteria. All of the 20 studies we included in our analysis complied with at least seven of 10 quality of diagnostic accuracy studies tool items (shown in e-Table 1 of our article2), and the study published in 1995 complied with nine of 10 quality of diagnostic accuracy studies tool items. The possible sources of heterogeneity across the studies were explored using metaregression analysis, which implied that heterogeneity resulted from factors other than the way in which a study was designed.

Second, in our experience and in the opinion of other experts,2 the accuracy of pleural ultrasonography (PUS) in diagnosing PNX depends on the operator’s skill. The lung point is a specific sign that allows PNX to be confirmed and the PNX volume to be determined. Furthermore, not all of the PNX diagnosed by PUS requires an invasive procedure. Only a large PNX needs emergency treatment. In these circumstances, a well-trained operator can evaluate PNX precisely with PUS. For mild to moderate PNX, clinicians can wait for the results of CT scans or chest radiographs (CXR)s before performing an invasive procedure.

Third, we indicated that PUS complements CXR rather than replaces it. Both PUS and CXR have advantages and limitations in detecting PNX. There is no conflict between these two modalities. The choice should be made depending on the specific conditions, including available equipment, clinician ability, and patient circumstances. PUS and CXR each have a role in detecting PNX.

Sufficient training and certification of the operator are essential before performing PUS. Despite the limitations of PUS, from our analysis we believe it is a promising alternative for the diagnosis of PNX, especially when performed by clinicians in critically ill patients.3

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Increased Adverse Events After Percutaneous Coronary Intervention in Patients With COPD

To the Editor:

In reference to the recent article by Enriquez et al (September 2011), we commend the authors for highlighting the problem of significantly lower rates of β-blocker and statin therapy given to patients with COPD. This finding suggests that patients with COPD may be undertreated for concomitant cardiovascular disease. In a similar vein, we previously reported on statin therapy in subjects undergoing evaluation for lung transplantation in whom pulmonary hemodynamic data were available and found that only 67% of subjects with angiographically proven severe coronary disease (CAD) were receiving statin therapy prior to evaluation.2 Applying a more liberal definition of CAD to include any degree of angiographically observed coronary disease, only 42% of the patients used statin therapy. Patients using β-blockers found to have severe vs any CAD on angiography was very low at 11% and 5%, respectively.

It has been demonstrated that COPD is associated with excess cardiovascular disease even after controlling for potential confounders, such as tobacco exposure.6,7 These studies, however, are not able to account for treatment differences, which could significantly affect the observed associations. The study by Enriquez et al has implications applicable both clinically and for future research design. Clinically, physicians should work to challenge the misperception that β-blockers are contraindicated in COPD. Data clearly show that cardioselective β-blockers are well tolerated in COPD and should not be withheld for this reason. The disparity of statin prescription is somewhat perplexing and perhaps explained by the observation of favorable lipid profiles, which may be associated with COPD.7 In terms of future research design, it is clearly imperative that studies of CAD in COPD take

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