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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 article1), 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) 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 that 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 al1 (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.3,4 These studies, however, are not able to account for treatment differences, which could significantly affect the observed associations. The study by Enriquez et al1 has implications applicable both clinically and for future research design. Clinically, physicians should work to challenge the misconception that β-blockers are contraindicated in COPD. Data clearly show that cardioselective β-blockers are well tolerated in COPD5,6 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
into consideration the possible confounding effect of potentially inadequate cardioprotective regimens given to these patients.

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Regarding the excess cardiovascular disease and worse outcomes among patients with COPD, we agree with Reed and Scharf that this is a complex issue with multiple potential mediators related to demographic and clinical characteristics, chronic hypoxic or systemic inflammatory processes, and treatment-related variables. For such reasons, we attempted to elucidate the relative contribution of such variables to outcomes within our study through the creation of stratified multivariate models, in which we found that demographic, angiographic, and treatment-related variables all appear to contribute to mortality, although demographic variables appear to contribute more to other outcomes evaluated. We concur that future studies of COPD and its association with adverse outcomes should also consider rigorous adjustment for potential confounding variables and should aim to identify factors contributing to the significant cardiovascular disease morbidity and mortality among these patients.

Questions posed by Reed and Scharf regarding physicians’ reasons for withholding guideline-recommended coronary artery disease therapies, such as statins, are very relevant and certainly worthy of further study. We agree that the lower lipid levels among patients with COPD, compared with those without COPD, could be one explanation for the decreased use of statins. Additionally, providers may be less inclined to escalate cardiac therapies because of the perception of patient frailty or because of a perception that atypical symptoms may be due to respiratory rather than cardiac causes. Regardless of the reasons for differences in treatment, we believe the results of our study support a greater awareness that patients with COPD represent a high-risk group, requiring focused attention after coronary revascularization for the prevention and management of adverse events.

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