Response

To the Editor:

We thank Dr Hochhegger and colleagues for their comments on our recent article in CHEST. The research conducted aimed to highlight that COPD is a heterogeneous condition and requires a multidimensional approach to characterization. CT scanning is now emerging as an important noninvasive tool in the multidimensional approach to phenotyping COPD, and our study demonstrated that spirometry and physiologic assessments alone could not differentiate between the radiologic phenotypes that exist within COPD. In particular, the study found that there was a great overlap in the detection of bronchiectasis, bronchial wall thickening, and emphysema in subjects with COPD. In their correspondence, Dr Hochhegger and colleagues correctly point out that there is a superior role of emphysema description using quantitative CT scan analysis; however, there is a recognized difficulty in quantification of airway wall dimensions required for bronchial wall thickening and bronchiectasis in COPD. In our study, we have defined the presence of bronchiectasis, bronchial wall thickening, and emphysema using established international thoracic radiologic guidelines, and more importantly, we have previously demonstrated that at our institution, the interobserver correlation of diagnosis of emphysema, bronchial wall thickening, and bronchiectasis is good while using tools widely available in clinical practice. This is particularly important because COPD is defined by chronic airflow limitation, which can include parenchymal destruction or airway wall thickening.

We agree with Dr Hochhegger and colleagues that the multidimensional phenotyping of COPD is important and that quantitative CT scanning analysis should be part of this for emphysema, especially in early disease. Better methods are required to interpret the airway wall geometry and airway densitometry with careful quality control of quantitative CT scanning in terms of standardized algorithms to capture the images, corrections for scanner variability, and standardized software for analysis. However, as an aid in the advancement of the field, we concur wholeheartedly that CT scanning is an important tool in phenotyping COPD.

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Overlooking Cardiovascular Risk in Patients With COPD

To the Editor:

In their excellent overview of the systemic effects of COPD in a recent issue of CHEST (January 2011), Nussbaumer-Ochsner and Rabe state that patients with COPD should be carefully evaluated for more general disorders associated with chronic systemic inflammation, such as cardiovascular disease (CVD). We present evidence that cardiovascular (CV) risk assessment is overlooked in this group.

We conducted a retrospective analysis of 117 patients with acute exacerbations of COPD consecutively admitted to two English district general hospitals over a period of 2 months in 2008. Evidence of preexisting CVD (a history of stroke, transient cerebral ischemia, myocardial infarction, angina, or peripheral vascular disease) was noted. For those with no previous CV events, we estimated the 10- and 20-year event risk in each case using a model from the Framingham data set based on gender, age, smoking status, BP, presence or absence of diabetes, and lipid profile. We also recorded prescriptions of statins and antiplatelet agents, drug classes both recommended for individuals at high risk of CV events.

Thirty-two of the 117 patients had preexisting CVD; 22 of this group were prescribed an antiplatelet agent and 17 a statin. A further 58 patients (50% of the cohort) had an estimated 10-year CV risk >10%; 32 of these patients had a risk of >20%. Less than 60% of this primary prevention group was taking antiplatelet drugs, and less than one-third of them had been prescribed a statin (Table 1).

Low prescription rates of aspirin for secondary CVD prevention may be explained by concerns of increased bleeding risk from concurrent coumarin anticoagulation; warfarin was prescribed in eight of the 10 patients with established CVD who were not taking antiplatelet agents (no patient in our cohort was prescribed warfarin in addition to an antiplatelet agent). For primary cardiovascular prevention, levels of antiplatelet use perhaps reflect an unresolved conflict between current guidance supporting their use and increasing evidence of net harm from bleeding events.

The evidence for the benefit of statins for both secondary and primary prevention is more robust, however, particularly where the risk of cardiovascular events exceeds 2% per year. Statins were underused in this cohort, particularly in the highest cardiovascular risk category, even in those patients with measured total cholesterol >5 mmol/L.

References

Low rates of statin and aspirin prescribing in patients with both COPD and increased cardiovascular risk imply that CVD is considered too infrequently in this group. Because COPD itself may well be an additional risk factor for CVD, it follows that statin therapy, along with other interventions to modify cardiovascular risk, is especially important in this complex group.1

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Chest Ultrasonography as a Replacement for Chest Radiography in the ED

To the Editor:

The study by Zanobetti et al1 in a recent issue of CHEST (May 2011) proposes chest ultrasonography as a replacement for chest radiography as the initial imaging modality in the ED. Although the superior sensitivity of ultrasonography over chest radiography for assessment of effusion is acknowledged and in keeping with previous studies,2 there are a number of caveats to using ultrasonography in favor of chest radiography as a first-line imaging test.

First, the training required for detecting the sonographic features of pneumothorax, localized atelectasis, and pulmonary fibrosis is extensive, and probably requires at least level 2 Royal College of Radiology training in chest ultrasonography in the United Kingdom (if not level 3, which would be equivalent to a radiologist).3 In addition, acquisition and interpretation of sonographic images is notoriously operator dependent, unlike interpretation of chest radiographs or CT images. This also presents issues with how a critical mass of operators who are adequately trained can be generated for the ED environment.

Second, acquisition of ultrasound equipment has cost implications for financially rationed health-care systems. This usually requires the demonstration of cost utility, which may be demonstrable for effusions4 but not the other conditions encountered in the study, on the basis of its performance against chest radiography.

Third, in the detection of pneumothorax, the chest radiograph (unlike ultrasonography) provides useful ancillary information as to the anatomic extent and location of most pneumothoraces (unless too small to be visible, in which case CT scan is needed). Many guidelines use the chest radiograph appearance in pneumothorax to guide further management.5

Finally, the chest radiograph may also give useful ancillary information not available from the ultrasonograph (eg, a more central lung neoplasm, mediastinal adenopathy, or a dilated right interlobar pulmonary artery suggesting pulmonary hypertension). In summary, chest ultrasonography has many applications, especially in the assessment of pleural effusion, but it should not replace the chest radiograph as a first-line imaging test in the assessment of acutely dyspneic patients presenting to the ED.

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