on a CT slice. Using 10 mm thick CT slices, the diagnostic yield from fiberoptic bronchoscopic procedures (FOB) was 60 percent for SPNs with the positive bronchus sign, and 30 percent for SPNs without this sign. Some SPNs were also scanned using 1.5 mm thick slices (thin-section CT), and the diagnostic yield of SPN without the positive bronchus sign dropped from 30 to 14 percent. This suggests that thin-section CT is more effective than standard CT in identifying whether lesions may be accessible by bronchoscopy. A 14 percent diagnostic yield by FOB for SPNs without the positive bronchus sign is far less than the expected 40-60 percent FOB yield reported by Cortese and McDougall and Radke et al for peripheral lung lesions. If a larger series of thin-section CT through SPN confirms the low yield of FOB for SPN without the positive bronchus sign, percutaneous needle aspiration biopsy would be the diagnostic procedure of choice for these SPNs. Using modified aspirating needles, 95 percent of malignant lesions and 85 percent of benign lesions can be diagnosed. Modified aspirating needles such as 20-22 gauge Turner or Greene needles (Cook, Bloomington, IN) combine the safety of a "skinny" needle with the ability to obtain a cytologic specimen for diagnosis of malignant SPN and a tissue core for diagnosis of benign SPN.

Thin-section CT has proved itself clinically useful in identification of benign SPNs, assessment of bronchietasis, and evaluation of focal airway disease. As Naïdich et al conclude, thin-section CT may also determine the best diagnostic approach for the SPN.

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REFERENCES

Clinical Value of Assessment of Acute Reversibility of Airways Obstruction in Patients with COPD

The study by Berger and Smith in this issue (see page 541) is a model for studies done in a purely clinical setting. They conclude that "in the clinical setting, changes in FEV, will identify the overwhelming majority of patients with significant, acute response to bronchodilators, and that drawing conclusions from a one-time no-response test may be erroneous in many cases." They further point out that, despite controversy, there is a common perception "that establishing marked reversibility . . . a) supports a diagnosis of asthma; b) justifies aggressive bronchodilator therapy; c) may predict a positive response to corticosteroids . . . ; d) implies a better prognosis than 'fixed' obstruction."

I assess reversibility of airways obstruction in patients with COPD routinely in my clinical practice—most of us do. The question (it is not a problem—a problem is solvable) is, what is the clinical value of spirometric assessment of acute reversibility of airways obstruction in patients with COPD? To be useful in day-to-day clinical practice, tests should, in my opinion, meet at least one of these basic criteria: 1) they should have a definite "normal" value, or range of "normality;" 2) they should strongly influence therapeutic decisions; 3) they should give a reasonable indication of prognosis. Does clinical assessment of acute reversibility of airways obstruction in COPD meet any or all of these criteria? The only honest answer is a resounding maybe.

First, what constitutes "significant" reversibility of airways obstruction in COPD? Other than arbitrarily chosen values, only two recent studies have addressed the question, and there is no consensus among clinicians or research physiologists as to what constitutes a "significant response." What, indeed, is the definition of asthma vs COPD? Who among us, who actually take care of patients on a day-to-day basis, has not seen patients with classic COPD, due to their long history of cigarette smoking, who show a 20 percent, 30 percent, or even more pronounced response to inhaled bronchodilators from time to time?

Second, although one widely quoted study related...
significant airway responsiveness to reliable predictions of response to orally administered theophylline in patients with COPD, most clinicians have not found it useful in every-day practice, dealing with individual patients. Additionally, as Berger and Smith point out, one test is not enough, "and . . . drawing conclusions from a one-time no-response test may be erroneous in many cases." However, with the constraints placed upon us by patients themselves, third party carriers, and governmental agencies, most patients get just one pre- and post-bronchodilator test. The fact is that whether or not the patient demonstrates acute reversibility of airways obstruction on that one test, most of us treat the symptomatic COPD patient with inhaled $\beta_2$-agonists and/or oral theophylline anyway—if not for bronchodilation, for the purported effects on mucociliary clearance and/or diaphragmatic function.

Third, although at least one study$^9$ reported longer patient survival in patients with COPD who showed significant responsiveness to an inhaled bronchodilator, other studies$^{6-7}$ have correlated acute reversibility of airways obstruction in COPD with a more rapid annual decline in FEV, and shorter patient survival.

It's enough to make the average blue-collar pulmonologist, whether in private practice or in an academic setting, throw up his or her hands and say, "The heck with it."

Many, many clinical questions, important in the day-to-day diagnosis and care of patients with obstructive lung diseases, hang in the balance. More clinical research is needed, but we're embroiled in the classic conflict between the tacticians, fighting the battle, and the strategists, far removed from the battle, with their grandiose plans and ideas. On the one hand we have the National Heart, Lung, and Blood Institute$^8$ tell us that pulmonary research at the cellular and molecular level is "Where it's at," and that the good ol' boys and girls who study clinical pulmonary physiology are dinosaurs doomed to rapid extinction. On the other hand, these vexing clinical questions, which if satisfactorily answered, could be of enormous benefit to those of us in clinical practice, our patients, and to third party carriers and governmental agencies, go unanswered.

Perhaps all of us (the tacticians and the strategists) should give Berger and Smith a standing ovation for a fine "huff n' puff" study performed in a clinical setting (while acknowledging the coequal importance of studies at the cellular and molecular level), and remember the words from the song by the little chap from Hibbing, Minnesota: "The answer, my friend, is blowin' in the wind . . . ."

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