Identifying Smokers at Risk for Developing Airway Obstruction

COPD currently ranks 12th among the conditions that contribute to the global burden of disease and is predicted to rank 5th by the year 2020.1 This increase will affect the quality of life in a large segment of the population and the increased costs will challenge the health-care system. Tobacco smoking is the major risk factor for COPD because a cigarette smoke-induced inflammatory process is the key to the pathogenesis of all of the important components of this syndrome, including chronic cough and sputum production,2 peripheral airways obstruction,3 and emphysematous destruction of the lung surface.4 However, as only 15 to 20% of heavy smokers develop airways obstruction, there is merit in identifying the smokers who are at the greatest risk in order to focus interventions on those that are most likely to benefit.

Until the mid 1960s, the prevailing concept was that the major site of airways resistance in the normal lungs was in the small airways. This changed with Weibel's quantitative studies of lung anatomy which established that the cross-sectional area of the peripheral airways was much greater than previously recognized.5 Shortly thereafter, direct measurements of airways resistance in animal and human lungs showed that the peripheral airways resistance was very low in the normal adult lungs and very high in patients with COPD.6,7 This introduced the concept that the peripheral conducting airways were the lungs' "silent zone" where disease could remain at a subclinical level for many years before airways obstruction became apparent. Unfortunately, the tests designed to detect small airways disease have not been found to be particularly helpful in managing patients with COPD. Furthermore, most of the population studies designed to detect subclinical disease have been cross-sectional in nature, with very few providing the much-needed longitudinal data required to identify the subset of heavy smokers who are at risk of developing significant airways obstruction.

In this issue of CHEST (see page 416), Stanescu and colleagues readdress this problem in a group of heavy smokers they have followed over a 13-year period. Their results show there was no excess risk for developing COPD during the follow-up period if the FEV1/FVC was normal on entry. Even as the FEV1/FVC became abnormal, it did not predict difficulty unless the abnormality was associated with an elevated slope of the nitrogen washout curve. Those who had both a reduction in the FEV1/FVC and an abnormal nitrogen washout were much more likely to have developed COPD by the end of the study. These results support the hypothesis that smokers who are going to develop COPD can be detected before there is a significant decline in function. The question as to whether intervention at this stage would have positive value remains to be determined by longitudinal studies that compare an intervention to a control group. However, their study shows that it may be possible to detect the minority of a smoking population that is going to develop COPD using these prospective measurements of lung function. This finding might be of interest to those who seek to prevent a decline in lung function by intervention as well as those who search for the genetic and environmental reasons why only a fraction of heavy smokers develop airways obstruction.

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REFERENCES
5 Weibel ER. Morphometry of the human lung. NY Acad Press, 1963

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This issue of CHEST contains a nonrandomized study report from Mer and collaborators (see page 426) on the clinical improvement of patients...