Assessing Airflow Obstruction: When Everything Is Not So Obvious

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According to the current international guidelines on lung function testing, airflow obstruction is defined as a disproportionate reduction of flow during a forced expiratory maneuver with respect to the maximal volume that can be expired with a single maneuver [1, 2]. Taking the forced expiratory volume in 1 s (FEV₁) and vital capacity (VC) as surrogates of flow and volume, respectively, airflow obstruction is therefore detected when the FEV₁/VC ratio falls below the normal range [1, 2]. If in general, detection of the pulmonary obstructive defect is relatively easy on this basis, it might not be so in some circumstances.

In a recent issue of the *Respiration*, Stănescu and Veriter [3] described lung function patterns in a small group of relatively young male subjects with no evident history of respiratory or cardiovascular diseases. Both FEV₁ and VC were decreased to similar extents relative to predicted values so that their ratio was normal, but total lung capacity (TLC) was surprisingly normal. According to the current international guidelines, the pattern would be consistent with lung restriction on the basis of the low FEV₁ and VC with a normal FEV₁/VC ratio, whereas the normal TLC would exclude the restriction.

More than three decades ago, Olive and Hyatt [4] examined the bronchoconstrictor response to inhaled allergens in asthma and documented a decrease in both FEV₁ and forced VC of similar extent in a substantial number of subjects. TLC did not change during the bronchial challenge, so that the increase in residual volume (RV) was entirely accounted for by the decrease in VC. Total respiratory resistance increased as a result of airway narrowing. As a tentative explanation, they suggested that the pattern could have been the result of patchy closure of peripheral airways occurring early on forced expiration. That airway closure and/or extreme airflow limitation may occur in humans without changes in TLC has since been repeatedly documented under a variety of different conditions [5–10].

The study by Stănescu and Veriter [3] does unfortunately not bring further or solid evidence that a low FEV₁ and VC with a normal FEV₁/VC ratio is definitely the result of airflow obstruction besides the fact that most of these subjects had a history of smoking and a lower CO diffusing capacity than an age-matched control group. Neither does it shed light on the underlying physiologic mechanisms. Yet, this study has the merit to remind us

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that this pattern does really exist in clinical practice and cannot be ignored for the simple reason that it may be present in a minority of patients. It also tells that if in general VC is a quite good index of flow in practice, it still remains but a mere surrogate of TLC. Thus, if 'VC is of

little use in discriminating between restrictive, obstructive, and mixed ventilatory defects', as clearly stated in the 1993 ERS document [2], then measurement of absolute lung volumes must still play a cardinal part of our daily clinical practice in the lung function laboratory.

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