



CORRESPONDENCE

Obstructive ventilatory defect with normal forced expiratory volume in one second/vital capacity ratio

To the Editors:

PELLEGRINO *et al.* [1] now define obstructive ventilatory defect not only on the basis of a low forced expiratory volume in one second (FEV₁)/vital capacity (VC) ratio but also as a particular pattern with a normal FEV₁/VC ratio. Although they write that “the definition of an obstructive pulmonary defect given in the present document is consistent with the 1991 American Thoracic Society [2] statement of interpretation,” a definition of an obstructive defect characterised by a normal FEV₁/VC ratio cannot be found in this latter document.

Incidentally, this pattern was described several years ago and called “small airways obstructive syndrome”. It is characterised by a normal total lung capacity (TLC) and FEV₁/VC ratio, but high residual volume (RV) and low VC and FEV₁ due to premature airways closure [3]. It was emphasised that a decrease in FEV₁, provided FEV₁/VC is normal, calls for measurement of lung volumes. In the absence of such measurements, this pattern would be ignored and called a restrictive or a “nonspecific defect” [4].

Subsequently, lung function was compared in healthy subjects and those with small airways obstructive syndrome [5, 6]. The pattern is characterised by a parallel displacement to the left of the flow–volume curve with respect to that of the healthy subjects, resulting in significant decreases of maximal flows.

None of these cited papers can be found in the reference section of the article by PELLEGRINO *et al.* [1]. Curiously enough, recently, two authors of this report felt it appropriate to write an editorial on these data, emphasising their interest [7].

Some authors have reported physiological abnormalities compatible with this pattern or mimicking some of its particularities [8, 9]. However, its individuality as an obstructive defect has not been previously recognised. For example, in patients with acute induced asthma, OLIVE and HYATT [10] reported a parallel displacement to the left of the flow–volume curve with respect to the control curve, resulting in a decrease of maximal flows and airway conductance and an increase in RV. There was a slight increase in TLC. No comments were made on the FEV₁/VC ratio. On calculating this ratio, it appeared that about half of the subjects did not change their FEV₁/VC following induced bronchoconstriction.

PELLEGRINO *et al.* [1] write that this pattern is “observed” or “caused by failure of the patient to inhale or exhale completely or when the flow is so slow that the subject cannot exhale long enough to empty the lungs to RV [...] Measurement of slow VC

(inspiratory or expiratory) may then give a more correct estimate of the FEV₁/VC ratio.”

Failure to inhale completely would result not only in a reduced VC but also, necessarily, in a reduced TLC, and therefore the defect should be called restrictive not obstructive.

Failure to exhale completely, or when the flow is so slow that the subject cannot exhale long enough to empty the lungs, would reduce vital capacity and increase residual volume but would not influence forced expiratory volume in one second, therefore not complying with the definition of this pattern, see figure 1b in [1], also see [7]. Indeed, forced expiratory volume in one second, which reflects maximal flow at high lung volumes and mid-vital capacity, is not decreased as a result of a slow expiration or incomplete emptying of the lung. Therefore, this physiological abnormality cannot be called obstructive. Only premature small airways closure may explain this pattern.

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From the authors:

We would like to thank D. Stanescu for giving us the opportunity to clarify the definition of airflow obstruction recently given in the interpretative strategies document from the American Thoracic Society/European Respiratory Society (ERS) Task Force [1]. The document states that in most cases a ratio of forced expiratory volume in one second (FEV₁) to vital capacity (VC) below the lower limits of normality is the parameter that best and most frequently embodies the concept of airflow obstruction with few exceptions. However, in some cases, a low FEV₁ with a normal FEV₁/VC may also be consistent with an obstructive pattern if total lung capacity is normal. This pattern has been observed after exposing the airways to a constrictor agent [2, 3] or in natural respiratory diseases [4], reproduced in healthy subjects [5, 6], and has already been interpreted as consistent with airway narrowing in the 1993 ERS guidelines on lung function testing [7]. The fact that this may be caused by a patchy collapse of the small airways on early expiration or expiratory flow limitation that, in turn, would cause an increase in residual volume was suggested by OLIVE and HYATT [2] in 1972 and then by other investigators in the years that followed [3, 5, 6]. However, this hypothesis still needs confirmation, especially after the demonstration that even the large airways may close with induced bronchoconstriction [8], thus suggesting that the small airways are not the only ones that may contribute towards the generation of air trapping and an increase in residual volume.

As previously mentioned in an editorial [9], the recent papers by STANESCU and co-workers [10, 11] merely confirm once again that this pattern exists and must be correctly identified in our daily clinical practice. Yet, neither of the papers bring any evidence that small airways closure is the underlying mechanism, as claimed by the author, thus leaving the field open to further evidence or alternative hypotheses [12]. If we accept that closure or extreme flow limitation are the probable mechanisms for the decrease in forced expiratory volume in one second with normal forced expiratory volume in one second/vital capacity ratio and total lung capacity, then we should also accept that they are the extreme forms of airway narrowing. In this sense, this pattern should be interpreted as obstructive in nature until disproved.

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Inhaler technique blind spot

To the Editors:

The paper by LAFOREST *et al.* [1] in a recent issue of the *European Respiratory Journal* is an important reminder of the poor overall management of patients with asthma. A large number of

French patients were studied, but one can assume that similar results would have been found in any country in Europe. The message that this paper clearly sends to all concerned with the management of asthma is that patients under the supervision of specialists do better than those under the care of general