Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Please include a cover letter with a complete list of authors (including full first and last names and highest degree), corresponding author’s address, phone number, fax number, and email address (if applicable). An electronic version of the communication should be included on a 3.5-inch diskette. Specific permission to publish should be cited in the cover letter or appended as a postscript. CHEST reserves the right to edit letters for length and clarity.

Criteria for Bronchodilator Response

To the Editor:

I read with interest the recent article by Newton et al (April 2002).1 In their “Material and Methods” section, it was not clear to me whether the authors determined the significance of the FVC response to bronchodilator according to the guidelines of the Intermountain Thoracic Society2 (which requires that the forced expiratory time [FET100%] not increase by >10%), or those of the American Thoracic Society3 (which do not set a specific FET100% criterion). In any case, there is no mention of what happened to the FET100% in responders and nonresponders, or among the former, in the flow responders vs the volume responders. It may be that a postbronchodilator increase in FET100% and/or FVC may have to be considered evidence of a significant response.

In the “Discussion” section, I found myself wishing that the authors had compared their observations on flow-vs-volume response with those by Paré et al.4 It would have been interesting to check the FEV1/FVC ratio (to separate flow responders from volume responders) in their patient population. Although, Paré et al4 studied a small group (n = 15) of asthmatics, their degree of hyperinflation appears quite similar to the patients of Newton et al.5 Paré et al4 suggested that volume response (ΔFEV1/ΔFVC < 1) correlated with the presence of a marker of small airway dysfunction (density dependence of flow), and they suggested a postbronchodilator recruitment of peripheral airways as a possible explanation. The hypothetical explanation of Newton et al5 appears similar, and would explain their frequent observation of directly measured postbronchodilator lung volume increase.

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To the Editor:

Our article in CHEST (April 2002)1 examined the effect of a bronchodilator on static and dynamic volumes. The largest effect was seen in residual volume. Dr. de la Hoz specifically focuses on one specific aspect of volume improvement, namely, that in FVC. Regarding the criteria for bronchodilator response in FVC, we used the American Thoracic Society guidelines2 and thus did not consider forced expiratory time (FET100%). However, it is reasonable to assume that in patients with no significant improvements in flow, but with increases in FVC, FET100% should have increased as well.

We used functional criteria to exclude asthmatic patients from the analysis. Furthermore, only a minority of the 15 asthmatic patients of Paré et al3 had the degree of hyperinflation seen in our COPD patients. Thus, the two study populations were quite different. Nevertheless, the mechanisms underlying the volume effects could be similar. Although the results of the study by Paré et al3 are consistent with a volume response due to peripheral airway recruitment, our data do not support this relationship. To the extent that maximal forced expiratory flows at the midexpiratory phase (FEF25–75) and at 75% of vital capacity (FEF75) reflect the contribution of the peripheral airways, we found no significant correlations between these flow parameters and the improvements in our volume indices (Table 3 of Newton et al1). ΔFEV1/FVC also did not correlate with ΔFEF25–75 and ΔFEF75 (unpublished data). Alternatively, FEF25–75 and FEF75 may not reflect the effects on peripheral airways in COPD patients, and the mechanism proposed by Paré et al3 does pertain. Further studies are required to elucidate this issue.

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