Gender and COPD in Patients With Chronic Respiratory Insufficiency Requiring Domiciliary Oxygen Therapy

To the Editor:

We read with interest the article in CHEST (October 2005) by de Torres et al in which the authors compared 53 FEV₁-matched men and women, and found that women were younger, had better oxygenation, fewer comorbidities, poorer quality of life, and higher degree of dyspnea than men. However, since only 8% of patients had stage IV COPD, a question remains regarding gender differences in patients with the most severe disease. Therefore, readers may be interested in a similar analysis that we conducted among patients with very severe COPD.

We studied 189 COPD patients (154 men and 35 women) with chronic respiratory insufficiency meeting the indication criteria for long-term oxygen therapy (LTOT). At the time of LTOT initiation, no age differences were seen between men and women (mean [± SD] age, 67 ± 8 vs 69 ± 9 years, respectively). Whereas FEV₁ did not differ between men and women, women had significantly lower FVC and TLC, and higher FEV₁/FVC compared to men (Table 1). No gender differences were seen in arterial blood gases or nutritional status. The proportion of men and women with comorbid conditions was similar: arterial hypertension, 38% vs 50%, respectively; diabetes, 19% vs 13%, respectively; myocardial infarction, 18% vs 7%, respectively; and stroke, 6% vs 3%, respectively.

Our results differ from those by de Torres et al in several aspects. First, unlike significant age differences in moderate COPD, men and women requiring LTOT were of similar age, implying that gender differences might exist in the rate of the annual decline in lung function. Indeed, a recent study points to such possibility. Second, women with moderate COPD had lower comorbidity scores than men, whereas at the time of LTOT initiation no gender differences were seen in comorbid conditions. However, women requiring LTOT were 10 years older compared to those with moderate COPD. Most importantly, our findings of lower FVC and total lung capacity in women at the time of LTOT initiation raise the possibility that gender differences might exist in the pathogenesis of COPD-related lung impairment. Further studies are needed to investigate this in more detail.

To the Editor:

We are pleased that Tkacova et al found our work (October 2005) interesting. As we pointed out, our findings are only applicable to the population included in our study sample (ie, women with mild-to-moderate COPD from the outpatient clinic at University Hospital clinic). The findings in a population of women and men requiring oxygen, as reported by Tkacova and coworkers, represent an even more selected group in whom the final expression of the disease may be due to many other phenotypic and mechanistic issues that cannot be extrapolated to patients without significant hypoxemia. Indeed, the data presented in that letter refer to a population of COPD patients with an uneven number of patients in both gender groups (154 men and 35 women) with similar degrees of airway obstruction (mean [± SD] FEV₁, 39 ± 19% predicted) and, by definition, a similar degree of hypoxemia at the time of the initiation of long-term oxygen therapy (LTOT). By forcing both factors to be similar, it is not surprising that men and women had the same age, similar degrees of comorbidity, equal PaO₂-Paco₂ values, and the same body mass indexes. When we analyzed our database and evaluated only those patients in Global Initiative for Chronic Obstructive Lung Disease stages III and IV (28 men and 28 women) with exactly the same mean FEV₁ (40 ± 7% predicted), we still observed differences in the studied prognostic parameters (ie, men vs women), as follows: mean age, 64 ± 7 vs 59 ± 11 years (p < 0.05); mean modified Medical Research Council score of ≥ 2, 11% vs 64%, respectively (p < 0.05); mean body mass index, 27 ± 3 vs 23 ± 3%, respectively (p < 0.05); mean PaO₂, 64 ± 10 vs 72 ± 11 mm Hg, respectively (p < 0.05); mean PaCO₂, 46 ± 6 vs 40 ± 5 mm Hg, respectively (p < 0.05); mean 6-min walk distance, 99 ± 20 vs 84 ± 21% predicted, respectively (p < 0.005); and mean Charlson scale, 4 (range, 3 to 8) vs 2 (range, 1 to 3) [p < 0.05]. We did not find differences in functional residual capacity percent predicted and all domains of the St. George Respiratory Questionnaire.

The differences between our findings and those of Tkacova et al could be explained in part by the more natural heterogeneity of the population included in their study.
Obesity As a Risk Factor for Developing Postoperative Atrial Fibrillation

To the Editor:

We read with great interest the recent article by Amar et al (November 2005)¹ on reduction in the incidence of postoperative atrial fibrillation with the preoperative use of statin independent of levels of early markers of inflammation. Obesity may influence the plasma levels of such markers, which are nowadays considered as an indicator of subclinical inflammation.² ³ Importantly, however, levels of the analyzed markers of inflammation are influenced by many variables, and they are not related to a single one.

The risk of postoperative atrial fibrillation has been significantly associated with rising body mass index in a large cohort of postoperative patients without a history of atrial fibrillation, and that association was independent of a broad range of clinical, surgical, and demographic factors known to influence the risk of atrial fibrillation. Some factors that have been linked to an increasing incidence of atrial fibrillation such as left atrial enlargement, enhanced neurohormonal activation, and left ventricular diastolic dysfunction are correlated with increasing body mass index or obesity.⁴ ⁵–⁷ Indeed, those cardiac structural abnormalities, which are characterized by the presence of heart failure with preserved left ventricular systolic function, are also commonly observed in patients with diastolic heart failure.⁸ Preliminary work by Fukuta et al⁹ has shown that patients with diastolic heart failure might benefit from the use of statins, and the reported survival benefit might be explained by the effects of statins on cardiac hypertrophy,¹⁰ endothelial function and vascular tone,¹¹ and systemic inflammation.¹² Thus, it would be of interest to have the data on body mass index and body weight for patients with and without atrial fibrillation to further explain the results of the present study.

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To the Editor:

The authors raise some important issues. Mean (± SD) body mass index did not differ in our patients who either did or did not develop atrial fibrillation (AF) [28 ± 5 vs 27 ± 4, respectively; p = 0.3]. We have previously found¹³ no difference in weight

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