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To the Editor:

We have read with interest the letter of Drs. Sajkov and McEvoy commenting on our article published in CHEST.1 Indeed, we were well aware of their excellent recently published study,2 and we have mentioned their results in the discussion. In fact the method of Sajkov et al2 was rather different from ours: they excluded patients with an associated lung disease, whereas our series comprised unselected OSAS patients; and they used Doppler ultrasound, whereas we used conventional right heart catheterization. Our results were also at variance with those of Sajkov et al: in our study, PH was observed in a minority of patients (37/220 vs 11/27 in the series of Sajkov et al) and was generally associated with marked abnormalities of daytime arterial blood gas values (mean PaO2=64±9 mm Hg), whereas the average PaO2 of the PH patients of Sajkov et al was of 72.2±7.6 mm Hg.

Furthermore, we do not agree with the way Drs. Sajkov and McEvoy interpret our own results: they emphasize the fact that 35% (13/37) of our PH patients were not significantly hypoxemic and add that we have “argued that these pulmonary hypertensive, nonhypoxemic patients must have developed their PH secondary to repetitive sleep apnea desaturations.” In fact only one patient exhibiting PH had a normal daytime PaO2 (>80 mm Hg) and this patient probably had some degree of left ventricular dysfunction. In the remaining 12 patients, hypoxemia was mild but not absent since PaO2 was generally in the range 65 to 75 mm Hg. Indeed, the mean asleep PaO2 was certainly lower in these patients due to the repetition of apneas and hypopneas. We have written that “the combination of marked nocturnal with mild to moderate daytime hypoxemia could explain the development of PH” but our data do not support the hypothesis that isolated nocturnal hypoxemia, that is in the absence of daytime hypoxemia, could lead to permanent PH in a significant number of OSAS patients. There could be some individual exceptions, particularly those patients who are high responders to hypoxia. Perhaps these patients were represented among the PH group (n=11) of Sajkov et al.

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REFERENCES

Osteoporosis in Patients Undergoing Lung Transplantation

To the Editor:

Recently, attention has been called to a particularly high prevalence of bone loss and debilitating osteoporotic fractures in organ transplant recipients,1 which may seriously compromise their quality of life. In a recent issue of CHEST, Aris et al reported in a cross-sectional observation low age-adjusted bone mineral density (BMD, Z scores) in the lumbar spine and proximal femur of lung transplant recipients and also, albeit to a lesser degree, of patients awaiting lung transplantation.2 These highly interesting observations are compatible with an accelerated bone loss likely triggered by diminished mobility, poor nutritional status, and corticosteroid therapy. However, by using age-adjusted BMD (Z scores) and by defining a “fracture threshold” as BMD below −2 Z scores, both the actual prevalence of osteoporosis and the risk of osteoporotic skeletal fractures may have been underestimated. Indeed, this risk continuously increases as absolute BMD values decline below peak bone mass.3 According to World Health Organization criteria of osteoporosis, ie, BMD 2.5 SDs below sex-matched peak bone mass (T scores), we found that as many as 35% of 20 adult candidates for lung transplantation, most with COPD, had osteoporosis.4 By contrast, in the study by Aris et al, BMD expressed as Z scores appeared relatively preserved in COPD patients, particularly as compared to patients with cystic fibrosis. This difference, however, could result from an earlier beginning of the respiratory disease and the potential involvement of the digestive tract in patients with cystic fibrosis who will therefore not achieve adequate peak bone mass. Besides, taking into account age-adjusted BMD (Z scores) in older COPD patients may be misleading, because both disease-related and age-related bone losses contribute to decrease in absolute bone mass in such patients, and thereby to an increase in fracture risk. Under these conditions, it appears that evaluation of absolute BMD (T scores) and prevention of bone loss should be considered early in the treatment of all patients with severe respiratory failure. However, pooling BMD measurements performed between 3 months and 3 years after surgery, as done in the study by Aris et al, could have blunted the importance of an early posttransplantation bone loss. Indeed, in a prospective investigation of bone mass changes in 12 lung transplant recipients receiving calcium and vitamin D supplements, we observed a significant bone loss at the spine, but not the femur, occurring within 6 months after surgery.5 Noteworthy, two patients suffered osteoporotic spinal fractures during this period. By contrast, bone loss appeared to be partly corrected 1 year after transplantation, possibly suggesting that long-term successful lung transplantation may be beneficial with regards to bone mass also. This also indicates that efficient prevention of bone loss occurring early in lung transplant recipients is urgently needed.

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To the Editor:

We appreciate Dr. Ferrari’s comments and concur strongly with his conclusion that bone loss is widespread in patients both

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