Pulmonary hypertension and obstructive sleep apnea


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We thank Simonson et al1 for raising awareness about the issue of pulmonary hemodynamics and sleep-disordered breathing in patients with systemic sclerosis.2 Regarding the link between sleep-disordered breathing and pulmonary hypertension, Sajkov et al3 showed that pulmonary hypertension associated with obstructive sleep apnea is generally mild to moderate, reversible with positive airway pressure therapy, and often associated with marked hypoxic vasoreactivity (ie, major elevations in pulmonary artery pressure with hypoxic exposure). In the case of fibrotic lung disease, including that which can occur in scleroderma, pulmonary artery pressures may be elevated by hypoxic pulmonary vasoconstriction as well as destruction of lung parenchyma. Pulmonary artery dilation does not necessarily correspond with elevated pulmonary artery pressures, but dilation has been associated with sleep-disordered breathing.1,4 The association between sleep-disordered breathing and pulmonary artery dilation was further noted in our study in individuals with systemic sclerosis.2

In the case of parenchymal lung disease, specifically chronic obstructive pulmonary disease (COPD), a prominent paper in New England Journal of Medicine suggested that pulmonary artery dilation may be predictive of COPD exacerbation.4 We wonder whether sleep-disordered breathing may be important causally in this association, particularly in light of recent evidence showing reduced risk of rehospitalizations by giving positive airway pressure to patients with frequent exacerbations of COPD.5 We also note that the definitions of pulmonary hypertension have recently changed (ie, mean pulmonary artery pressure of 20 mm Hg rather than 25 mm Hg), suggesting that older studies may have underestimated the true burden of pulmonary hypertension. Thus, we are highly supportive of further efforts to characterize the links between pulmonary hypertension, pulmonary artery dilation, and sleep-disordered breathing, particularly in patients with parenchymal lung disease. Further study could assess important mechanisms as well as the impact of positive airway pressure on cardiopulmonary physiology as well as other health outcomes.

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REFERENCES

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