at the dilator tube-tracheostomy tube transition zone. The percutaneous tracheostomy kit manufactured by Smiths Industries (SIMS Inc, Keene, NH) includes a beveled-tipped, low-profile, flexible tracheostomy tube that snugly fits the introducer dilator essentially eliminating insertion resistance. I can only speculate that elimination of dilator-guided, tracheostomy tube placement resistance results in significantly less incidence of the tracheal cartilaginous ring fracture described in the above-mentioned article. I have absolutely no affiliation or relationship with any company involved with the manufacture of tracheostomy products. In light of the autopsy findings described, I believe that the improvement outlined in the commercially available percutaneous dilatational tracheostomy kit described is a significant step forward.

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To the Editor:

We agree with Dr. Sandifer that the introduction of a standard, rigid tracheotomy tube may be difficult. Often, there is a small ridge of the cannula that prevents its smooth introduction beyond the cartilaginous rings. Many manufacturers try to improve their percutaneous tracheotomy kit to minimize insertion resistance, and among them is the manufacturer of the kit that we have used (Cook, Medical Company; Son, The Netherlands). We believe that until now this kit has the advantage that its results and complications have been well studied.

The fact that smooth introduction of the cannula results in less tracheal ring fractures is a speculation only, as Dr. Sandifer states. We think that the clinical implications of a fractured tracheal ring should not be overestimated. Incision of one or more tracheal rings is a normal part of conventional open tracheotomy. In a study of the late complications of percutaneous tracheotomy, we show that the incidence of tracheal stenosis after percutaneous dilatational tracheostomy is relatively low.1 However, an obliquely inserted cannula may give indentation of fractured tracheal rings into the lumen and result in tracheal stenosis.

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Pulmonary Hemodynamics and Hypoxemia in Sleep Apnea

To the Editor:

We read with interest the report by Chaouat et al3 in which pulmonary hemodynamic findings in a large unselected population of obstructive sleep apnea syndrome (OSAS) patients were described. The population included a significant group of patients who also suffered from chronic lung disease (mainly obstructive). The investigators found that pulmonary hypertension (PH) in their study was associated strongly with an impairment of both lung function and daytime arterial blood gases and not indexes of sleep apnea. They concluded that PH in OSAS is caused predominantly by obstructive airways disease and that the gas exchange disturbance is consequent on the airways disease.

In an earlier study from our laboratory, we measured pulmonary hemodynamics using Doppler ultrasound in 27 OSAS patients selected for the absence of lung disease.2 We found mild PH in approximately 40% of our sample which was not related to lung function impairment. PH was associated with daytime hypoxemia but this was mild (PaO2>69 mm Hg in all but one of the PH patients) and insufficient to lead to awake PH through the mechanism of hypoxic pulmonary vasoconstriction. Since the mild hypoxemia could not be explained on the basis of lung function impairment or the degree of obesity, we speculated that it might be caused by ventilation-perfusion ratio inequality associated with pulmonary vascular remodeling. The latter, we argued, could have resulted from repetitive apnea-induced hypoxic vasoconstriction in sleep. The new finding from our study was that PH could occur in the absence of significant lung disease and in the absence of daytime hypoxemia of sufficient magnitude to cause hypoxic pulmonary vasoconstriction.

Chaouat et al3 argue that their data “do not support the hypothesis that PH can develop in OSAS patients in the absence of daytime hypoxemia,” ie, that their data are at variance with ours. The editorial that accompanied their article3 supports this assertion. We do not agree with these conclusions and believe that the data from Chaouat et al in fact are consistent with our hypothesis. We did not find it surprising that in their unselected group of OSAS patients, in whom 20% had significant obstructive lung disease, strong associations were found among airflow obstruction, hypoxemia, and PH. It has, of course, been recognized for many years that COPD is one of the major causes of PH. Thus, in populations of OSAS patients who may also have COPD, the dominant cause of PH may well be lung disease. The aspect of their results, however, that interested us, and one that is consistent with our findings, is that 35% of their PH patients were not significantly hypoxemic (using their definition of hypoxemia viz PaO2≤65 mm Hg). They argued that these PH, nonhypoxemic patients must have developed their PH secondary to repetitive sleep apnea desaturations. This argument is virtually identical to the one we developed in our article to explain the PH in our patients.

We believe the most interesting question arising from these studies is whether repetitive hypoxemia in sleep is capable of inducing structural remodeling in the pulmonary circulation, thereby increasing ventilation-perfusion mismatch and worsening gas exchange and by these mechanisms accelerating the natural history of OSAS. These questions, we believe, are best addressed by studying subjects with “pure” OSAS and thereby avoiding the confounding influences of hypoxic lung disease on the pulmonary circulation.

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