reached. This was justified by the inability of the test to confirm the diagnosis in a subset of sleep apnea patients when the interpretation is based on rigid criteria defining SaO₂ decline.² In these circumstances, the sensitivity of home oximetry in pioneer studies such as the one of Williams et al³ may be lower than the one reported in the review by Netzer et al (65% instead of 78%). Following the publication of our results² and the recognition of their importance in clinical practice,⁴ the interpretation algorithm of subsequent studies evaluating the diagnosis value of oximetry was based on SaO₂ variability analysis.³ In complement to the interpretation of the results of the literature detailed by Netzer et al,¹ it should be mentioned that the poor specificity that we and others⁵ observed with this screening method dramatically depends on the definitions used to define breathing abnormalities and especially hypopneas. In fact, most of the articles cited in this review considered a minimal SaO₂ fall as an obligatory event associated with flow reduction to consider the presence of an hypopnea. However, according to the recommendations of up-to-date guidelines,⁶ such SaO₂ changes are no longer required to score such breathing abnormality. At the end of the spectrum, nonapneic, nonhypopneic events (respiratory effort-related arousals) can be observed in the absence of significant nocturnal desaturation. It is clear that if our data were reanalyzed with these new criteria, the specificity would be dramatically enhanced with a minor alteration of the sensitivity of our method. In this context, we believe that the analysis of SaO₂ variability alone is more than ever of first importance in the interpretation of oximetry tracings. Prospective studies that will take into account for these new definitions of sleep-related breathing disorders need to be conducted to evaluate the accuracy of overnight oximetry in the diagnosis of the disease.

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To the Editor:

We appreciate the critical comments of Frédéric Séries and acknowledge the importance of his publication. We were aware of this study but, by oversight, did not include it in our bibliography.

As Dr. Séries mentions, and as we have outlined in our review, the specificity of overnight pulse oximetry in the diagnosis of sleep-disordered breathing depends on the type of breathing abnormalities. This is demonstrated with our data of oximetry samples from hypopneic and obstructive apneas. Calculations of breathing events based on the interpretation of pulse oximetry may underestimate or overestimate the number of hypopneas and central apneas. New calculation models for automatic readings should therefore include different forms of oxygen desaturation per time interval and not only count desaturations with a specified decline of 3% or 4% arterial oxygen saturation. Indeed, newer models for automated pulse oximetry readings are based on arterial oxygen saturation and pulse-rate variabilities. We agree with Dr. Séries that these methods will enhance the sensitivity and specificity of pulse oximetry in the diagnosis of sleep-disordered breathing.

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Changes in Oxygen Saturation in Patients Undergoing Fiberoptic Bronchoscopy

To the Editor:

We read with great interest the article by Jones and O’Driscoll that appeared in CHEST (June 2001).¹ They reported that supplemental oxygen treatment during fiberoptic bronchoscopy (FOB) was required in 151 of the patients (14.4%), and nearly half of the patients who need supplemental oxygen therapy had FEV₁ levels <1 L in a large-scale study. We have recently investigated the predicting factors on the changes in saturation during FOB in a prospective study. In our unit, based in a 500-bed teaching hospital in Turkey, supplemental oxygen is not administered routinely to all patients during FOB. Forty-four patients (33 male and 11 female patients; mean ± SD age, 51 ± 17 years) who did not require oxygen treatment at least 2 weeks before FOB were included. Bronchoscopies were performed transnasally, and low-to-moderate doses of IM midazolam and atropine were used as premedication in all patients.

Oxygen saturation was monitored during the procedure with pulse oximetry (PO) [model 305; Palco Laboratories; Santa Cruz, CA], and arterial blood gas levels were measured before and after FOB. For predicting the lowest saturation value during FOB, age, gender, primary disease, presence of pleural effusion and/or atelectasia, time of FOB procedure, and basal saturation values were taken as independent variables.

No difference in saturation values was found between arterial blood gas levels and PO analysis both before and after FOB. This finding suggests monitoring of saturation with PO during FOB is a noninvasive and reliable method. Saturation values (mean ± SD) were significantly decreased after FOB (from 96.5 ± 1.0% to 91.6 ± 3.6%, p < 0.001), and desaturation (arterial oxygen saturation < 90%) was detected in 22 of the patients (50%) during the procedure.

REFERENCES


To the Editor: