Nocturnal oxyhemoglobin desaturation in COPD patients with arterial oxygen tensions above 60 mm Hg.

We studied 152 COPD patients with a daytime PaO₂ ≥ 60 mm Hg using formal polysomnography (EEG, airflow, respiratory muscle movement, ear oximeter) to detect the presence of nocturnal, nonapneic, oxyhemoglobin desaturation. Nine subjects were disqualified by the unexpected discovery of sleep apnea, as were another eight because they could not sleep in the laboratory setting. Of the remaining 135 subjects, 37 (27 percent) desaturated below a baseline sleep saturation of 90 percent for five minutes or more, reaching a nadir saturation of at least 85 percent. Anthropomorphic, pulmonary function, and historic factors comparing desaturators and nondesaturators failed to separate the groups. Awake PaO₂ at rest in the desaturators was significantly lower than in the nondesaturators.
The desaturators was significantly lower than in the non-desaturators. The PaCO$_2$ was higher in the desaturators. Reversibility of the desaturation phenomenon was demonstrated in three patients during subsequent polysomnographic studies following periods of clinical improvement. Continuous oxyhemoglobin monitoring during sleep remains the only reliable tool for detecting nocturnal desaturation.

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