Pressure Support Ventilation

To the Editor:

We read with interest the report by Piper and Sullivan in the February 1994 issue of Chest. They described 13 patients with obstructive sleep apnea (OSA) and hypercapnia who were treated with nasal intermittent positive pressure ventilation (NIPPV); most were successfully switched to nasal continuous positive airway pressure after 7 to 18 days of NIPPV. The report leaves unanswered the important questions of method of diagnosis and treatment modality for alveolar hypoventilation. Alveolar hypoventilation is more prevalent than is commonly appreciated, and it may occur in up to 71% of sleeping patients, as defined by end-tidal CO₂ > 45. Up to 32% of cases may exhibit similar levels of daytime hypercapnia. Routine use of end-tidal CO₂ monitoring is a noninvasive and sensitive method of diagnosis.

We frequently use timed pressure support ventilation (BiPAP, Respiration) as the initial treatment modality for alveolar hypoventilation. We retrospectively identified 42 patients with coexisting OSA and alveolar hypoventilation and found that timed pressure support ventilation was used in 22 of 42 patients. Ventilation improved in polysonomography in 18 of these 22 patients, which suggests that pressure support ventilation provides adequate inspiratory pressures to many patients. Piper and Sullivan do not state the specific pressures needed to ventilate their 13 patients with NIPPV. Waldhorn describes a range of pressure support ventilation pressures from 14 to 22 cm of water, but only two of his eight patients were treated with timed pressure support ventilation. Pressure support ventilation is less expensive than NIPPV; therefore, timed pressure support ventilation should be evaluated to see if it is effective therapy for individual patients.

It is noteworthy that Piper and Sullivan show a more rapid improvement in ventilation control than had been previously reported. This is an important consideration when deciding to resudy patients to determine the efficacy of less costly treatment with nasal continuous positive airway pressure.

In summary, important issues remain unresolved in the diagnosis and treatment of alveolar hypoventilation in OSA. Routine use of end-tidal CO₂ monitoring is a noninvasive and sensitive method of diagnosis. Timed pressure support ventilation is an effective treatment modality in many patients with alveolar hypoventilation.

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REFERENCES
3 Waldhorn RE. Nocturnal nasal intermittent positive pressure ventilation with bi-level positive airway pressure (BiPAP) in respiratory failure. Chest 1992; 101:516-21

To the Editor:

We agree with the comments of Drs. Claman and Votteri about the underrecognition of hypoventilation in many patients with obstructive sleep apnea. For this reason, our routine clinical practice is to measure carbon dioxide levels both by evening and morning blood gases as well as continuous monitoring of transtcutaneous carbon dioxide levels. We have found such measurements useful during diagnostic as well as treatment nights, reflecting the degree of hypoventilation occurring during spontaneous unassisted breathing as well as being an additional measure of the patient’s clinical response to therapy. We have found that most patients use of continuous positive airway pressure and pressure support ventilation (BiPAP, Respironics) is sufficient either to either prevent or minimize this rise in CO₂, keeping it within an acceptable range, ie, 4 to 7 mm Hg above daytime spontaneous values. The degree of hypoventilation occurring in the patients reported in our study was both substantial and unresponsive to continuous positive airway pressure. They represent the extreme end of the spectrum of obstructive sleep apnea with marked hypercapnia (mean daytime CO₂: 62 mm Hg, mean rise in REM sleep 16 mm Hg).

Our intent in this article was to emphasize the degree to which both spontaneous daytime and nocturnal breathing could be improved with short-term full nocturnal ventilatory support. In particular, relief from severe hypoventilation in sleep can and does lead to an improved spontaneous ventilatory drive both awake and asleep. With an improved drive to breathe by use of this technique, simpler and less expensive devices can then be used on a long-term basis.

We agree that timed pressure support ventilation can be an effective treatment modality for many patients with alveolar hypoventilation, and we have used it extensively ourselves in the management of patients with both acute and chronic hypercapnic respiratory failure. However, we, like other authors, have found pressure support ventilation can be less effective than volume cycled ventilation in patients with severe disease or those requiring high peak inspiratory pressures. In the patients studied in our report, peak inspiratory pressures ranged from 20- to 45-cm H₂O using a volume cycled device. Importantly, such pressures were not static either night-to-night or even throughout a single night. As volume cycled devices can accommodate these variations in both upper airway and chest wall compliance by delivering a preset tidal volume despite changes in peak inspiratory pressures, such devices retain an advantage over pressure preset devices in the very difficult patient. Although we agree that pressure support ventilation is easier for both the patient and clinician to use, volume cycled ventilation remains a therapeutic option if pressure support ventilation fails to give satisfactory results.

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REFERENCES