The Ethics of Bilevel Positive Airway Pressure

To the Editor:

Noninvasive mechanical ventilation applied via either a tight fitting nasal or face mask has been used with considerable success in the treatment of acute and chronic respiratory failure arising as a result of a variety of medical conditions that include COPD, pneumonia, chronic heart failure, obstructive sleep apnea, and neuromuscular weakness. The availability of commercial devices that provide a bilevel, differential, positive airway pressure has made bilevel positive airway pressure (BiPAP; Respironics, Inc; Murrysville, Pa) a routine option for critically ill patients at many hospitals and often provides a means to avoid a more invasive approach with endotracheal intubation and conventional mechanical ventilation.

Unfortunately, we have found that many physicians at our institution consider BiPAP to be a routine therapeutic option for patients who have explicitly requested, through living wills or stratification of care documents, that mechanical ventilation not be implemented in the event of severe or life-threatening medical illness. This is compounded by the additional observation that patients frequently require either chemical or physical restraints to tolerate this intervention. The cost, measured in terms of lost patient autonomy, can be profound in this circumstance.

The ethics of this practice and its impact upon patients' rights near the end of life need to be considered. Given the widespread availability of this technology, it would seem prudent to discuss it specifically when issues of cardiopulmonary resuscitation are reviewed with patients and families.

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Sleep Apnea Blown Away by CPAP

To the Editor:

Regular continuous positive airway pressure (CPAP) use has been associated with a reduction in the severity of obstructive sleep apnea (OSA) on the first night post-CPAP, although OSA still remains evident.1,2 We report a patient with moderately severe OSA in whom overnight polysomnography results were entirely normal after regular CPAP use and almost led to a missed diagnosis.

A 48-year-old white man (body mass index, 27 kg/m²) presented with a 3-year history of heavy snoring, witnessed apneas, and daytime somnolence. A consulting respiratory physician prescribed CPAP at an empiric pressure of 7 cm H₂O, pending diagnostic polysomnography. Diagnostic overnight polysomnography (three electroencephalography channels, two electro-oculography channels, submental electromyography, thoracic and abdominal inductance plethysmography, oronasal thermistors, oxygen saturation, intercostal electromyography, and snore sensor [Nellcor Puritan-Bennett (Melville) Ltd; Ottawa, Canada]) was performed 1 year after commencement of CPAP treatment. Despite having received advice to discontinue CPAP for 72 h before the polysomnogram, he had continued to use CPAP until the study night. The polysomnogram results were normal, and there was no evidence of OSA or the upper airway resistance syndrome (Table 1).

The patient protested that after having discontinued CPAP on two previous camping vacations, he experienced unrefreshing sleep and daytime somnolence within 2 to 3 days. Therefore, repeat diagnostic polysomnography was arranged after 10 days of no CPAP treatment. The second overnight polysomnogram revealed moderately severe OSA, requiring CPAP at 8 cm H₂O.

Diagnostic polysomnography was performed a third time, 2 months after CPAP treatment had been reinstated, and results again were entirely normal. There had been no change in the patient's weight, nasal airway patency on clinical exam, sleep staging, or sleeping position, which might account for the difference in OSA severity between the three studies.

Current guidelines for polysomnography do not stipulate the need for patients with suspected OSA to discontinue CPAP before diagnostic polysomnography.3,4 This case history demonstrates the importance of discontinuing CPAP treatment before overnight polysomnography in patients with suspected OSA, because CPAP pretreatment may not only disguise the severity of OSA, but may actually conceal its existence.

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REFERENCES

Table 1—Sleep and Respiratory Data Over Three Diagnostic Polysomnograms*

<table>
<thead>
<tr>
<th>Study</th>
<th>1 (After using CPAP)</th>
<th>2 (After 10 nights of no CPAP)</th>
<th>3 (After using CPAP again)</th>
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<tbody>
<tr>
<td>TST, min</td>
<td>414.5</td>
<td>325.9</td>
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<tr>
<td>Sleep efficiency, %</td>
<td>81.5</td>
<td>73.6</td>
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<td>Stage 1, % TST</td>
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<tr>
<td>Stage 2, % TST</td>
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<td>14.2</td>
<td>31.8</td>
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<tr>
<td>Stage REM, % TST</td>
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<td>AHI</td>
<td>3.2</td>
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</tbody>
</table>

* TST=total sleep time; REM=rapid eye movement; AHI=apnea/hypopnea index.