Non-apnoeic REM sleep induced nocturnal oxygen desaturation treated by nasal continuous positive airway pressure

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ABSTRACT Non-apnoeic oxygen desaturation related to rapid eye movement (REM) sleep in a patient with hypothyroidism, obesity, respiratory failure, and cardiac failure was improved by treatment with nasal continuous positive airway pressure of 10 cm H₂O.

Sleeping non-apnoeic arterial oxygen desaturation occurs frequently in patients with chronic obstructive or restrictive pulmonary disease.¹ The largest falls in oxygen saturation (SaO₂) occur during rapid eye movement (REM) sleep. These episodes of arterial desaturation are believed to have an important role in the development of pulmonary hypertension and right heart dysfunction.²

The occurrence of oxygen desaturation during REM sleep may be explained by the loss of skeletal muscle tone.³ Reduced activity of intercostal and other accessory muscles at night may lead to alveolar hypoventilation in patients with lung disease.⁴ Loss of upper airway muscle tone can also increase upper airways resistance and inspiratory work load,⁵ and may reduce alveolar ventilation further. Changes in muscle tone during REM sleep may lead to a reduction in functional residual capacity (FRC) to below closing capacity, resulting in an increase in ventilation-perfusion inequalities and intrapulmonary shunting.⁶

We wondered whether nocturnal continuous positive airway pressure (CPAP) could reduce episodes of REM sleep related oxygen desaturation. CPAP could improve oxygen saturation by splinting the upper airways. This should prevent any increase in resistance of the upper airways, by increasing FRC and reducing venous admixture. We report a patient with nocturnal non-apnoeic arterial oxygen desaturation who was improved by 10 cm H₂O nasal continuous positive airway pressure.

Case report

A 64 year old man with severe hypersomnia was referred for sleep studies. He was very obese (198% of ideal body weight) and was an habitual loud snorer. He had smoked 1 pack of cigarettes a day for 40 years. Symptoms included chronic cough and sputum production, grade III/IV dyspnoea, morning headaches, and daytime hypsomnolence. Physical examination showed a short neck and a hypertrophic tongue and uvula with a reduction in the oropharyngeal aperture.

There were diffuse rhonchi and signs of right heart failure. Attempts to carry out pulmonary function tests were unsuccessful: the patient could neither understand the instructions nor coordinate his efforts. Arterial blood gases analysis (sitting position) showed oxygen tension (Pao₂) to be 5-6 kPa, carbon dioxide tension (Paco₂) 8-8 kPa, pH 7-34, and bicarbonate 35-5 mmol/l. Thyroid function tests indicated primary hypothyroidism: thyroxine 2-7 (normal range 78–158) mmol/l, triiodothyronine 0-78 (1-54–3-07) mmol/l, thyroid stimulating hormone 43 (7–0-4) mU/l. The electrocardiogram (ECG) showed right axis deviation and partial right bundle branch block. The patient was treated with bronchodilators and diuretics. An initial polysomnographic study, performed with the patient breathing room air, included a determination of sleep stages, measurement of oxygen saturation with an ear oximeter (Biox II, Ohmeda), nasal and mouth flows (thermocouples), thoracoabdominal movements (inductance vest calibrated by the least square method), and a continuous ECG. Sleep stages and abnormal respiratory events were defined by standard criteria.⁷ The results are reported in the table. Episodes of apnoea occurred only during stage I–II and were exclusively obstructive in type. They were associated with episodes of ventricular tachycardia. The largest falls in SaO₂ were recorded during REM sleep, and were not related to apnoea. As SaO₂ fell below 60%, the lower limit of linearity of our oximeter, the falls could not be quantified. The percentage of REM sleep time spent at an SaO₂ below 90% illustrates the severity of these non-apnoeic desaturations (fig). The ECG remained normal throughout REM sleep.

Results of the diagnostic and continuous positive airway pressure (CPAP) polysomnographic studies

<table>
<thead>
<tr>
<th></th>
<th>Diagnostic night</th>
<th>CPAP (10 cm H₂O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sleep time (TST) (h)</td>
<td>5-1</td>
<td>5-4</td>
</tr>
<tr>
<td>Sleep period time (SPT) (h)</td>
<td>7-9</td>
<td>7-3</td>
</tr>
<tr>
<td>Sleep efficiency (%)*</td>
<td>64-5</td>
<td>73-9</td>
</tr>
<tr>
<td>Stage I–II sleep (% TST)</td>
<td>78-0</td>
<td>84-0</td>
</tr>
<tr>
<td>Stage III–IV sleep (% TST)</td>
<td>4-1</td>
<td>4-5</td>
</tr>
<tr>
<td>REM sleep (% TST)</td>
<td>17-9</td>
<td>11-5</td>
</tr>
<tr>
<td>Apnoea index†</td>
<td>3-6</td>
<td>2</td>
</tr>
<tr>
<td>Apnoea hypopnoea index†</td>
<td>29-2</td>
<td>19-8</td>
</tr>
<tr>
<td>Mean apnoea duration (S)</td>
<td>42</td>
<td>27</td>
</tr>
<tr>
<td>Lowest REM SaO₂ (%)‡</td>
<td>&lt;60</td>
<td>69</td>
</tr>
<tr>
<td>Lowest postapnoea SaO₂ (%)</td>
<td>66</td>
<td>73</td>
</tr>
</tbody>
</table>

*The ratio total sleep time/time in bed.
†Number of episodes per hour of sleep.
‡The lowest levels of SaO₂ were reached during REM sleep and were not related to apnoea.

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A second polysomnographic study was performed with the patient receiving nasal continuous positive airway pressure (Sleep Easy II Nasal CPAP, Respironics). There were no other changes in treatment between the two sleep studies; both were performed before thyroid replacement treatment. The positive pressure was increased progressively from 0 to 10 cm H₂O. Non-apneic oxygen desaturation during REM sleep diminished progressively with increasing nasal continuous positive airway pressure. The lowest Sao₂ value during REM sleep was 63% with 6 cm H₂O continuous positive airway pressure and 69% with 10 cm H₂O. The improvement in oxygen saturations with nasal continuous positive airway pressure is illustrated in the figure. Continuous positive airway pressure of 10 cm H₂O did not eliminate non-REM sleep apnoeic events (table) but the patient was unable to tolerate higher pressures. The ECG abnormalities related to apnoeic events disappeared with continuous positive airway pressure.

Nasal continuous positive airway pressure resulted in clinical improvement, abolishing hypersomnia and morning headache and subjectively improving the quality of sleep. Severe respiratory abnormalities persisted with continuous positive airway pressure, however, so it was not considered for home treatment. After two months of thyroid replacement treatment the patient’s sleep related breathing disorders improved: apnoea-hypopnoea index was 25 and the lowest Sao₂ reached during REM sleep was 60%.

Discussion

Our patient had morbid obesity, hypothyroidism, and a sleep apnoea syndrome with severe non-apneic arterial oxygen desaturation during REM sleep. The association of an obstructive sleep apnoea syndrome and non-apneic falls in Sao₂ during REM sleep has been reported in patients with coexistent chronic obstructive lung disease. Non-apneic oxygen desaturation during REM sleep is well described in obstructive and restrictive lung diseases. Nocturnal oxygen desaturations secondary to obstructive apnoea or hypopnoea has been reported in hypothyroidism. We believe that our patient had pulmonary disease (severe dyspnoea, hepatojugular reflux, diffuse rhonchi, hypoaxaemia with hypercapnia, and respiratory acidosis).

The percentage of total sleep time spent in REM sleep fell with continuous positive airway pressure (19-9% during the initial study, 11-5% with nasal continuous positive airway pressure). We do not believe that this was the cause of the improvement in oxygen saturation because we observed a progressive improvement in Sao₂ during one REM cycle after the continuous positive airway pressure level had been increased.

The mechanism underlying oxygen desaturation during REM sleep is similar to obstructive and restrictive pulmonary diseases; similar mechanisms could therefore account for the improvement in the fall in Sao₂ during REM sleep with nasal continuous positive airway pressure regardless of the underlying lung disease. The benefit of continuous positive airway pressure could be due to an increase in lung volume with improvement in VA/Q inequalities or to a pulmonary splinting effect of the upper airways.

The place of continuous positive airway pressure in the treatment of nocturnal oxygen desaturations will have to be defined by further studies; it could possibly replace or reduce the need for nocturnal supplementary oxygen in some of these patients.

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References