CASE REPORT

Development of Central Sleep Apnea After Maxillofacial Surgery for Obstructive Sleep Apnea

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Central sleep apnea is a rarely reported complication of surgery for obstructive sleep apnea (OSA). We report the case of a 38-year-old male who developed marked central sleep apnea 3 months after a maxillomandibular advancement for moderate OSA, which spontaneously resolved on his 6-month postoperative polysomnogram. Five prior cases of this postoperative complication have been reported in non-obese individuals after tracheostomy for OSA. Additionally, a recent study demonstrated that patients with atmospheric pharyngeal closing pressures are susceptible to unstable ventilation. We hypothesize that latent high loop gain from chronic OSA, coupled with atmospheric pharyngeal closing pressures, predisposed our patient to develop unstable ventilation after an abrupt postoperative change in his ventilatory load. Our case supports delaying postoperative polysomnography ≥ 6 months in individuals at high risk for this complication.

Keywords: Central sleep apnea; obstructive sleep apnea; maxillomandibular advancement


REPORT OF CASE

A 38-year-old Hispanic male presented with complaints of hypersomnolence, snoring, and witnessed apneas. Physical examination was significant for a BMI of 26, Mallampati class 3 airway, neck circumference of 16 inches, and a normal cardiopulmonary exam. A split-night polysomnogram (PSG) revealed an apnea-hypopnea index (AHI) of 26 events/hour, all obstructive. These events were ameliorated with CPAP, and no central apneas were observed during the treatment portion of the PSG. The patient started CPAP and initially noted a subjective improvement in his daytime hypersomnolence, but was unable to tolerate therapy. He elected to undergo uvulopalatopharyngoplasty followed by radio frequency ablation of the tongue, but his postoperative PSG demonstrated an increased AHI of 32 obstructive events/hour. Six months later the patient underwent maxillomandibular advancement (MMA) with a LeFort I osteotomy, bilateral mandibular sagittal split osteotomy, mandibular genial tubercle advancement of 10 mm, septoplasty, and bilateral inferior turbinectomies. Lateral neck radiographs demonstrated an excellent postoperative result, with enlargement of the posterior airway space from 2 to 11 mm. However, PSG performed 3 months after surgery demonstrated an AHI of 39 events/hour, with the overwhelming majority central in origin (Figure 1). The patient was not taking any medications at the time of the PSG; follow-up echocardiogram was normal, and an MRI of the cervical spine confirmed airway patency in multiple positions. Three months later, repeat PSG revealed resolution of these central events with an AHI of 11 events/hour. Details of polysomnographic studies are summarized in Table 1.

DISCUSSION

CSA describes an apnea that occurs due to lack of respiratory drive.¹ CSA development has been described using the concept of high loop gain—an engineering term that in the respiratory system describes an exaggerated ventilatory response to carbon...
There are no respiratory efforts during the apneas in either the abdominal or thoracic channel. During the central apneas, there are prominent cardiac oscillations. The apneas are terminated by pronounced respiratory efforts demonstrated in both the thoracic and abdominal channels.

**Figure 1**—Five minutes of overnight PSG recording demonstrating central apneas in the CPAP channel (nasal pressure transducer recording).

<table>
<thead>
<tr>
<th>PSG</th>
<th>Total AHI</th>
<th>Max Desat</th>
<th>Obstructive AHI</th>
<th>Mixed AHI</th>
<th>Central AHI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial</td>
<td>26.3</td>
<td>84%</td>
<td>26.3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Post-UPP</td>
<td>32</td>
<td>90%</td>
<td>32</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Post-MMA, 3 mo</td>
<td>39.15</td>
<td>85%</td>
<td>0.78</td>
<td>3.13</td>
<td>35.22</td>
</tr>
<tr>
<td>Post-MMA, 6 mo</td>
<td>11.54</td>
<td>88%</td>
<td>8.73</td>
<td>0.85</td>
<td>1.92</td>
</tr>
</tbody>
</table>

Wellman et al. prospectively studied the effect of pharyngeal closing pressures on ventilatory instability in 25 OSA patients. Loop gain did not correlate with AHI in patients with negative and positive pharyngeal closing pressures, but there was a significant correlation between AHI and loop gain in patients with atmospheric closing pressures. While technically obese (mean BMI 31.9), these patients had the lowest BMI of the 3 study groups. The authors concluded that patients with atmospheric closing pressures were “highly susceptible” to unstable ventilation.

Like the patients in these studies, our patient was younger and less obese than the typical individual with OSA. It is plausible that our patient had an atmospheric pharyngeal closing pressure, which, combined with his latent high loop gain from chronic OSA, predisposed him to develop unstable ventilation after an abrupt change in his ventilatory load following MMA. The fact that his CSA resolved 6 months postoperatively after his ventilatory control stabilized further supports this hypothesis. This case provides unique insight into the ventilatory mechanics of patients following OSA surgery and supports delay-
ing postoperative polysomnographic testing for at least six 6 months after the procedure, especially in patients at high risk for this complication.

DISCLOSURE STATEMENT

The views expressed in this review are those of the authors and do not reflect the official policy or position of the Department of the Army, Department of Defense, or the U.S. Government. This was not an industry supported study. The authors have indicated no financial conflicts of interest.

REFERENCES