Upper airway surgery to rescue the “untitratable” patient with OSA and obesity

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This is a case report of an 41-year-old male with obesity (body mass index 90 kg/m²), severe obstructive sleep apnea (OSA), and an apnea-hypopnea index of 90 events/h despite high bilevel positive airway pressure (BPAP). He presented to the PAP Alternatives Clinic and underwent tonsillectomy, expansion sphincter pharyngoplasty, and partial uvulectomy to improve positive airway pressure effectiveness. Postoperative BPAP retitration resolved the patient’s OSA. The patient is currently using BPAP therapy at home with improvement in both objective and self-reported OSA outcomes.

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INTRODUCTION

Failed positive airway pressure (PAP) titration for obstructive sleep apnea (OSA) despite elevated pressure does occur; however, the prevalence is unknown. Cited reasons for high-pressure requirements include upper airway anatomical abnormalities (eg, tonsillar hypertrophy, deviated septum) and pathophysiological conditions (eg, allergic rhinitis), all potentially necessitating otolaryngology evaluation.1 Surgical treatment of OSA may be indicated for patients in whom primary treatments such as weight loss and PAP therapy have failed. Surgical procedures include tracheostomy, skeletal surgery (eg, maxillomandibular advancement), soft-tissue surgery (eg, uvulopalatopharyngoplasty), and upper airway stimulation. Although multilevel pharyngeal surgery has been shown to reduce OSA severity, it can also facilitate reduced PAP level requirements in patients with previous continuous positive airway pressure (CPAP) intolerance.2 No previous studies have demonstrated the use of upper airway surgery to rescue a previously “untitratable” patient. We present a case report of a patient with obesity and severe residual OSA on PAP who underwent soft-tissue surgery and achieved PAP therapy effectiveness.

REPORT OF CASE

A 41-year-old African-American male with obesity (body mass index 90 kg/m²), diabetes, hypertension, asthma, and past pulmonary embolism on apixaban was referred by his otolaryngologist for evaluation and possible surgical treatment of OSA. OSA was first diagnosed 7 years prior; however, at no time was the patient able to use CPAP consistently. His original sleep study results were unobtainable. CPAP data download revealed severe residual OSA on 13 cm H2O: an apnea-hypopnea index (AHI) of 75.7 events/h despite minimal mask leak (Table 1).

Physical examination findings included 4+ palatine tonsils and a Friedman III tongue position. Treatment options were discussed including weight loss, bariatric surgery, tracheostomy, and PAP re-trial. He was recommended to undergo a repeat PAP titration with transcutaneous CO2 monitoring and a mask fitting.

PAP titration failed to establish an optimal pressure despite high CPAP and bilevel positive airway pressure (BPAP) levels (Table 1). Residual AHI was 90.1 events/h on BPAP therapy: inspiratory positive airway pressure (IPAP) of 30 cm H2O and expiratory positive airway pressure (EPAP) of 15 cm H2O. The large pressure support gap resulted from titration of IPAP for hypopneas because obstructive apneas were not observed after EPAP reached 15 cm H2O. Titration protocol was not strictly followed because the maximum recommended IPAP-EPAP differential is 10 cm H2O.

At the follow-up visit, surgical treatment options were discussed further and tracheostomy was recommended; however, the patient declined due to procedure morbidity. He instead elected to proceed with tonsillectomy, expansion sphincter pharyngoplasty, and partial uvulectomy followed by PAP re-trial. The patient was optimized by the preoperative team to be cleared for anesthesia and surgery. Surgery was performed without complications. Of note, tonsillar volume was significant at 10 cc measured by water displacement. He experienced a short episode of self-limited bleeding on postoperative day 14 requiring discontinuation of apixaban for 2 days. Upon resumption of apixaban, he experienced no further issues.

The patient resumed CPAP therapy at 13 cm H2O immediately following surgery. His data download revealed a residual AHI of 15.6 events/h, reduced from 75.7 events/h before surgery. BPAP retitration was then performed with resolution of OSA (AHI = 1.3 events/h) on IPAP = 30 cm H2O, EPAP = 20 cm H2O. He required supplemental oxygen due to hypoxemia despite absence of apneic events, likely attributable to obesity hypoventilation although CO2 levels were not obtained.
Case report

H2O, and 3 L/min supplemental O2. Residual AHI on this with obesity. Both in-laboratory ef

This is the first published case to demonstrate the use of OSA surgery to improve PAP effectiveness in a patient with obesity. Both in-laboratory efficacy and home effectiveness of PAP therapy are demonstrated in Table 1. Although the patient required high bilevel pressures following surgery, there was resolution of sleep-disordered breathing (AHI < 5 events/h), increased PAP adherence, and an improved self-reported outcome.

Several mechanisms may explain failed PAP titration despite maximal pressures; however, this scenario is relatively uncommon and has not been systematically studied. Kim et al demonstrated a positive correlation between body mass index, tonsil grade, and CPAP requirements. Upper airway anatomical abnormalities such as deviated septum and enlarged turbinates can reduce nasal patency and restrict PAP airflow. Nasal pathology including allergic rhinitis and nasal polyposis can also cause airflow limitation, thus requiring higher PAP treatment levels. In some cases, CPAP may worsen airway obstruction by causing tongue base or epiglottic collapse.

The two most likely contributors to initial PAP failure in our patient were obesity and palatine tonsillar hypertrophy. Schwartz et al suggested that excessive parapharyngeal fat secondary to obesity may contribute to upper airway narrowing.5 Furthermore, central obesity has been shown to decrease lung volume, which causes increased upper airway collapsibility and thus higher PAP requirements.5,6 Our patient underwent expansion sphincter pharyngoplasty, which increases oropharyngeal lateral wall tension, thus partially counteracting obstruction. Tonsillectomy was performed simultaneously resulting in 10 cc of tissue removed, thereby increasing oropharyngeal airspace.

Previous literature highlights the role of upper airway surgery in facilitation of PAP therapy. Two meta-analyses of patients who underwent upper airway surgery found significant reductions in average CPAP requirements and improved average therapy adherence.7,8 Ayers et al reviewed 11 articles totaling 323 patients and found a mean reduction in titrated CPAP of 1.40 cm H2O with a 0.62-hour improvement in average therapy adherence.7 Camacho et al reviewed 82 patients and reported a mean reduction of 2.66 cm H2O and a 2.5-hour improvement in average therapy adherence following isolated nasal surgery.8 Collectively, the data support a multidisciplinary approach for evaluating and treating OSA in patients in whom PAP therapy has failed.

Our case report demonstrates the importance of otolaryngology evaluation for this high-risk patient with severe OSA. Although the patient was willing to use PAP therapy, his OSA was untreated in part because of upper airway abnormalities. Soft-tissue surgery addressed these abnormalities and contributed to PAP effectiveness.

### DISCUSSION

This is the first published case to demonstrate the use of OSA surgery to improve PAP effectiveness in a patient with obesity. Both in-laboratory efficacy and home effectiveness of PAP therapy are demonstrated in Table 1. Although the patient required high bilevel pressures following surgery, there was resolution of sleep-disordered breathing (AHI < 5 events/h), increased PAP adherence, and an improved self-reported outcome.

### ABBREVIATIONS

- AHI, apnea-hypopnea index
- BPAP, bilevel positive airway pressure
- CPAP, continuous positive airway pressure
- OSA, obstructive sleep apnea
- PAP, positive airway pressure

### REFERENCES


### Table 1—Comparison of questionnaire and positive airway pressure treatment data before and after upper airway surgery.

<table>
<thead>
<tr>
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<th>Preoperative</th>
<th>Postoperative</th>
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<tbody>
<tr>
<td><strong>BMI (kg/m²)</strong></td>
<td>90.0</td>
<td>87.4</td>
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<td><strong>ESS score</strong></td>
<td>5</td>
<td>5</td>
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<tr>
<td><strong>FOSQ-10 score</strong></td>
<td>1.6</td>
<td>3.3</td>
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<tr>
<td><strong>PAP titration</strong></td>
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<tr>
<td>AHI (events/h)</td>
<td>90.1</td>
<td>1.3</td>
</tr>
<tr>
<td>IPAP (cm H2O)</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>EPAP (cm H2O)</td>
<td>15</td>
<td>20</td>
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<tr>
<td><strong>PAP download</strong></td>
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<tr>
<td>CPAP AHI (events/h)</td>
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<tr>
<td>Mean use (h/night)</td>
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<tr>
<td>Use ≥ 4 h/night (%)</td>
<td>23</td>
<td>50</td>
</tr>
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</table>

*PAP titration AHI is the residual AHI on BPAP therapy, that is, no baseline data. **PAP titration IPAP and EPAP refer to the maximum titrated pressure. †PAP download AHI on 13 cm H2O CPAP. ‡PAP download AHI on auto BPAP: maximum IPAP = 25 cm H2O, minimum EPAP = 15 cm H2O, maximum pressure support = 8 cm H2O, minimum pressure support = 4 cm H2O. AHI = apnea-hypopnea index, BMI = body mass index, BPAP = bilevel positive airway pressure, CPAP = continuous positive airway pressure, EPAP = expiratory positive airway pressure, ESS = Epworth Sleepiness Scale, FOSQ-10 = Functional Outcomes of Sleep Questionnaire, IPAP = inspiratory positive airway pressure, PAP = positive airway pressure.


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**DISCLOSURE STATEMENT**

All authors have seen and approved the manuscript. Work for this study was performed at Emory University. The authors report no conflicts of interest.