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Response to CPAP and UPPP in Apnea

Frank J. Zorick, MD,* Timothy Roehrs, PhD,* William Conway, MD,† George Potts,‡ and Thomas Roth, PhD*

Ninety-two consecutive patients with obstructive sleep apnea syndrome (OSAS) were studied before and six weeks after treatment with either nasal continuous positive airway pressure (CPAP) or uvulopalatopharyngoplasty (UPPP) (n = 46 per group). Assignment of patients to treatment was based on clinical considerations and patient preference. Patients were assessed by nocturnal polysomnography and performance on the Multiple Sleep Latency Test (MSLT) the following day. Before treatment, the CPAP and UPPP groups did not differ in sleep-related respiratory disturbance, oxygenation during sleep, fragmentation of sleep, or the degree of excessive daytime sleepiness indicated by the MSLT. Both treatments produced significant improvement on all measures. However, improvement in UPPP patients was significantly less consistent than that of CPAP patients. To the extent that UPPP successfully reversed the respiratory disturbance (i.e., 50% reduction in respiratory events index), sleep continuity and daytime sleepiness were improved to a degree comparable to that of patients treated with CPAP. (Henry Ford Hosp Med J 1990;38:223-6)

Obstructive sleep apnea syndrome (OSAS) is a disorder characterized by frequent, recurrent episodes of upper airway obstruction during sleep, associated with hypoxemia, bradycardia, and brief cortical arousal to resume breathing (1). OSAS presents typically as excessive daytime sleepiness (EDS), obesity, and heavy snoring, which occurs predominantly in men. In severe and advanced stages OSAS can lead to life-threatening cardiac arrhythmias, right heart failure, high blood pressure, and functionally incapacitating EDS (2,3). Consequently, untreated OSAS is recognized as a serious and potentially lethal disease (4).

The two most frequently used treatments of OSAS are nasal continuous positive airway pressure (CPAP) and uvulopalatopharyngoplasty (UPPP). CPAP is the application of continuous positive pressure to the airway through an airtight nosemask, thereby maintaining patency during sleep. UPPP is designed to enlarge the oropharyngeal airspace by excision of redundant soft tissues of the soft palate, uvula, and posterior lateral pharyngeal wall (6,7). Successful UPPP reduces or eliminates the airflow obstruction that occurs during sleep.

The effectiveness of each of these treatments has been evaluated independently in a number of case series (8,9). While treatment chosen for a given patient is based on anatomical and medical considerations as well as patient preference, no published study has compared changes in the signs and symptoms of OSAS following CPAP or UPPP.

Methods

Subjects

Study subjects included 92 consecutive patients with OSAS treated by either CPAP (n = 46) or UPPP (n = 46) (Table 1). Each patient presented to our Sleep Disorders Center with a history of EDS and loud snoring. Diagnosis of OSAS was based on the diagnostic evaluation described below. Each patient underwent treatment (CPAP or UPPP) and was reevaluated six weeks later.

Assignment to treatment was based on patient preference, anatomical findings, and medical indications. Specifically, patients without oropharyngeal collapse (indicated by the otolaryngologic examination) were directed to CPAP treatment. Patients who were poor surgical risks because of age or other medical complications also were directed to CPAP. Patients were otherwise free to select their treatment. While not randomly assigned to the two treatment groups, no significant differences were noted between patients of the two groups in age, body weight, and sex distribution (Table 1).

Procedure

All patients received a complete sleep and medical history, physical examination, and otolaryngologic examination. The latter included direct nasopharyngoscopy using a Machida nasopharyngoscope. Patients were examined while awake, in both the upright and supine positions. Upper airway dynamic changes during the Mueller maneuver were observed fiberoptically and patients rated as to site and degree of airway compromise (10).

Nocturnal polysomnogram (8 hours) was followed by a Multiple Sleep Latency Test (MSLT) the next day. The nocturnal
polysomnogram included the central (C3/C4) and occipital (Oz) EEG, right and left electrocorticograms (EOGs), and submental and tibialis electromyograms (EMGs) with continuous ECG recording (11). Respiration was monitored with a thermistor at the nose and mouth to detect airflow and by thoracoabdominal strain gauges to detect respiratory effort (12). Oxygen saturation was measured continuously by a Biox ear oximeter.

Information was recorded by Grass model 78-D or Nihon Kohden (models 4312 and 4212) polygraphs. The Grass polygraphs were calibrated with a pen deflection of 50 pV = 7.5 mm and paper speed was 10 mm/sec.

The pretreatment and posttreatment measures of sleep-related respiratory disturbance, oxygenation during sleep, stages of sleep, and level of EDS (MSLT, average sleep latency in minutes) are presented in Table 2. Before treatment, the CPAP and UPPP patients did not differ in any of these parameters. Both treatments were associated with significant improvement in sleep-related respiratory events and oxygenation. Respiratory events index (REI) was reduced significantly (F = 214.68, P < 0.001), as was the apnea index (AI) (F = 108.34, P < 0.001). Oxygenation expressed as minutes of SaO2 < 85% was improved significantly (F = 45.84, P < 0.001).

These data supported the use of CPAP treatment for sleep-related respiratory disturbance and oxygenation. These improvements were not due to a placebo effect, as the UPPP group in this study did not differ from the CPAP group in any outcome measures. The CPAP was effective in reducing both apnea and hypopnea, and it improved oxygenation and sleep-related respiratory events.

**Table 1**

Characteristics of the Treatment Groups

<table>
<thead>
<tr>
<th></th>
<th>CPAP</th>
<th>UPPP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>51.67</td>
<td>47.24</td>
</tr>
<tr>
<td>SD</td>
<td>(10.3)</td>
<td>(13.7)</td>
</tr>
<tr>
<td>Males</td>
<td>37</td>
<td>39</td>
</tr>
<tr>
<td>Females</td>
<td>9</td>
<td>7</td>
</tr>
<tr>
<td>Body mass index*</td>
<td>38.32</td>
<td>35.53</td>
</tr>
<tr>
<td>SD</td>
<td>(8.49)</td>
<td>(7.01)</td>
</tr>
</tbody>
</table>

*Body mass index = weight (kg)/height² (cm) x 10,000.

<table>
<thead>
<tr>
<th></th>
<th>CPAP Pretreatment</th>
<th>Posttreatment</th>
<th>Preoperative Postoperative</th>
</tr>
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<tbody>
<tr>
<td>Respiratory Measures:</td>
<td>76.0 (23)</td>
<td>7.0 (9)</td>
<td>71.0 (30)</td>
</tr>
<tr>
<td>Apnea index</td>
<td>49.0 (26)</td>
<td>4.0 (9)</td>
<td>42.0 (28)</td>
</tr>
<tr>
<td>Minutes SaO2 &lt;85%</td>
<td>12.0 (12)</td>
<td>1.0 (6)</td>
<td>14.0 (16)</td>
</tr>
<tr>
<td>Sleep Measures:</td>
<td>57.0 (24)</td>
<td>16.0 (10)</td>
<td>54.0 (21)</td>
</tr>
<tr>
<td>% Stage 1</td>
<td>31.0 (21)</td>
<td>53.0 (11)</td>
<td>33.0 (18)</td>
</tr>
<tr>
<td>% Stage 2</td>
<td>1.0 (3)</td>
<td>9.0 (8)</td>
<td>4.0 (6)</td>
</tr>
<tr>
<td>% Stage REM</td>
<td>10.0 (6)</td>
<td>21.0 (7)</td>
<td>10.0 (5)</td>
</tr>
<tr>
<td>Daytime Sleepiness:</td>
<td>4.4 (0.9)</td>
<td>10.3 (1.1)</td>
<td>4.1 (1.0)</td>
</tr>
</tbody>
</table>

Data are means and (standard deviations).

**Results**

The pretreatment and posttreatment measures of sleep-related respiratory disturbance, oxygenation during sleep, stages of sleep, and level of EDS (MSLT, average sleep latency in minutes) are presented in Table 2. Before treatment, the CPAP and UPPP patients did not differ in any of these parameters. Both treatments were associated with significant improvement in sleep-related respiratory events and oxygenation. Respiratory events index (REI) was reduced significantly (F = 214.68, P < 0.001), as was the apnea index (AI) (F = 108.34, P < 0.001). Oxygenation expressed as minutes of SaO2 < 85% was improved significantly (F = 45.84, P < 0.001).

The measures of sleep staging also improved toward normal. The percentage of light sleep (stage 1) was reduced (F = 100.83, P < 0.001) and the percentage of deep slow wave sleep increased set at 70 Hz. All electrode impedences were less than 10,000 ohms and paper speed was 10 mm/sec.

The MSLT was conducted at 10 AM, 12 PM, 2 PM, and 4 PM following standard procedures (13). Subjects were in bed in a darkened room and instructed to try to sleep while EOGs, submental EMG, and EEGs, always including an Oz placement, were recorded. The recording was terminated after 1 minute of unambiguous stage 1 sleep, after the first signs of stage 2 or rapid eye movement (REM) sleep, or after 20 minutes of continuous wakefulness (10).

Six weeks after UPPP, operated patients returned to the sleep laboratory for follow-up polysomnogram and MSLT. CPAP patients underwent an adjustment night to determine optimum CPAP pressure. The home CPAP apparatus (Respironics Sleep Easy) was then set at the lowest effective pressure to prevent airway obstruction during the 8-hour adjustment night. These patients returned six weeks later for a polysomnogram while using the CPAP apparatus, followed the next day by a MSLT without the apparatus.

All nocturnal polysomnograms were scored for sleep stages according to the standard criteria of Rechtschaffen and Kales (11). The respiratory tracings were scored for episodes of apnea (cessation of airflow and/or effort for 10 sec or more) or hypopnea (50% reduction in airflow and/or effort for 10 sec or more). These were classified as central apneas (cessation of both oronasal airflow and respiratory effort), obstructive apneas (cessation of oronasal airflow with continued respiratory effort), or mixed apneas (episodes with central and obstructive components). The lowest level of oxygen saturation (SaO2) and the frequency and duration of hypoxia (SaO2 less than 85%) were recorded.

These outcome measures were submitted to two factor mixed design analyses using the Statistical Analysis System Institute general linear model analysis. Data from the two treatment groups determined the between-group factor, and pretreatment versus posttreatment scores represented the within-group factor. Conservative P levels corrected by the Greenhouse-Geisser method were used for all pretreatment and posttreatment comparisons. The between-group posthoc comparisons were made with the Duncan test.

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**Table 2**

Treatment Effects on Respiration and Sleep

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Data are means and (standard deviations).
In all patients with CPAP treatment. Restoration of the continuity of sleep leads to a normalization of daytime alertness.

Patients receiving UPPP did not improve uniformly. In patients who experienced a marked reduction in the number of respiratory events, the amount of stage 1 sleep was reduced proportionately. Improvement in sleep continuity was associated with improvement in the level of daytime sleepiness/alertness in such patients. Such was not the case for patients in whom UPPP was unsuccessful. The apnea continued as did both the sleep fragmentation and the daytime sleepiness. This linear relationship

Discussion

These data show that CPAP is a more uniformly successful treatment for OSAS than UPPP. CPAP prevents respiratory disturbance during sleep, restoring its continuity and consequently correcting EDS in all patients. To the extent that UPPP successfully reverses the respiratory disturbance, sleep continuity is improved and daytime sleepiness normalized.

These findings extend and replicate our previous research demonstrating a relationship between fragmentation of sleep and EDS in OSAS. Sleep fragmentation refers to a brief (1 to 10 sec) ECG arousals which occur at the termination of an apnea episode. The arousals disrupt the continuity of sleep and reduce its restorative capacity which leads to EDS.

CPAP with pressure set to maintain a patent airway prevents apnea, the consequent EEG arousals, and fragmentation of sleep. In this study the increased episodes of stage 1 sleep seen in OSAs (reflecting the fragmentation of sleep) returned to normal
between daytime sleepiness/alertness and sleep fragmentation is illustrated in the Figure. Note that the REI indicates the number of arousals and hence the fragmentation of sleep.

**References**