Long-Term Survival of Patients With Obstructive Sleep Apnea Treated by Uvulopalatopharyngoplasty or Nasal CPAP*


Patients with obstructive sleep apnea (OSA) have decreased long-term survival. Treatment of OSA with either nasal continuous positive airway pressure (CPAP) or tracheostomy improves survival, but the effect of uvulopalatopharyngoplasty (UPPP) on survival is unclear. We attempted to contact all patients with OSA treated with either UPPP or nasal CPAP over a 6-year period to compare long-term survival between these two treatments. One hundred fifty-four patients had a UPPP during this period. Five of these patients were later treated with nasal CPAP and were excluded from the analysis. Twelve of the remaining 149 were unavailable for follow-up but were included in the analysis. Follow-up polysomnography was performed on 140 (94 percent) of these patients; 114 (81 percent) had either a postoperative apnea index <5/h or >50 percent reduction in apnea index. Two hundred eight patients were started on a regimen of nasal CPAP during the same period. Eighty-two patients discontinued nasal CPAP therapy and were excluded from analysis. Nine of the remaining 126 were unavailable for follow-up but were included in the analysis. Six patients treated with UPPP died. Three of these six patients had a 3-month follow-up polysomnogram that revealed apnea indices of 1/h, 5/h, and 23/h. Three patients treated with nasal CPAP died. There was no difference in the long-term survival between the two treatment groups. We conclude that there may be no difference in the long-term survival of patients with OSA between those treated with UPPP and those treated with nasal CPAP. This study emphasizes the importance of follow-up polysomnography in all patients after UPPP.

\[ \text{Al} = \text{apnea index}; \text{CPAP} = \text{continuous positive airway pressure}; \text{OSA} = \text{obstructive sleep apnea}; \text{UPPP} = \text{uvulopalatopharyngoplasty} \]

METHODS

We reviewed all patients with OSA who were treated with either UPPP or nasal CPAP at our center between January 1984 and April 1990. Obstructive sleep apnea was defined as a pretreatment apnea index (AI) of \( \geq 1 \) or an apnea and hypopnea index of \( \geq 15 \) determined during overnight polysomnography.

In the early stages of the 6-year period, patients with OSA were treated by conservative therapy or UPPP. Nasal CPAP has been our primary treatment for symptomatic OSA since the advent of commercial nasal CPAP. Patients underwent UPPP in the latter part of the study period if they refused to consider or continue nasal CPAP treatment.

We first attempted to contact patients by phone. If this was unsuccessful we next attempted contact through their family physician. We then wrote to those who remained unavailable for follow-up at their last known address and submitted their names to the Government of British Columbia’s Vital Statistics department. The Vital Statistics department records the names of those people who die within British Columbia along with the cause and time of death.

In patients who had died, we attempted to determine the cause and time of death from either the details on their death certificate or through contact with their family physician, last attending physician, or family members. In the UPPP group, we identified those patients who had prior treatment with nasal CPAP to address the possible bias introduced by this crossover in treatment. We reviewed patient records to determine the number of patients with follow-up polysomnograms after treatment in both UPPP and nasal CPAP groups. We examined whether follow-up polysomnography had an effect on survival. Patients whose post-UPPP AI was \( \geq 5 \) or had decreased by a minimum of 50 percent 3 months following UPPP when compared with the preoperative polysomnogram were...
Table 1—Patient Characteristics*

<table>
<thead>
<tr>
<th>N</th>
<th>Sex, M/F</th>
<th>Age, yr</th>
<th>BMI, kg/m²</th>
<th>AI, /h</th>
<th>Follow-up, mo</th>
<th>Deaths</th>
<th>5-yr Survival†</th>
</tr>
</thead>
<tbody>
<tr>
<td>UPPP all patients</td>
<td>149</td>
<td>134/15§</td>
<td>50 ± 11</td>
<td>30 ± 5</td>
<td></td>
<td>25 ± 17]</td>
<td>43 ± 13]</td>
</tr>
<tr>
<td>UPPP A1&gt;20</td>
<td>73</td>
<td>70/38</td>
<td>50 ± 10</td>
<td>30 ± 5]</td>
<td>38 ± 13]</td>
<td>51 ± 21]</td>
<td>4</td>
</tr>
<tr>
<td>CPAP all patients</td>
<td>126</td>
<td>100/26</td>
<td>52 ± 12</td>
<td>36 ± 9]</td>
<td>20 ± 14]</td>
<td>28 ± 13]</td>
<td>3</td>
</tr>
<tr>
<td>CPAP A1&gt;20</td>
<td>48</td>
<td>33/9</td>
<td>51 ± 13</td>
<td>34 ± 8]</td>
<td>35 ± 12]</td>
<td>29 ± 12]</td>
<td>1</td>
</tr>
<tr>
<td>He et al‡</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Untreated A1&lt;20</td>
<td>142</td>
<td>142</td>
<td>51 ± 11</td>
<td>31 ± 8]</td>
<td>9 ± 6]</td>
<td>N/A</td>
<td>3</td>
</tr>
<tr>
<td>Untreated A1&gt;20</td>
<td>104</td>
<td>104</td>
<td>53 ± 12</td>
<td>33 ± 10]</td>
<td>46 ± 20]</td>
<td>N/A</td>
<td>11</td>
</tr>
<tr>
<td>UPPP</td>
<td>60</td>
<td>60</td>
<td>48 ± 11</td>
<td>36 ± 7]</td>
<td>60 ± 25]</td>
<td>N/A</td>
<td>8</td>
</tr>
<tr>
<td>CPAP</td>
<td>25</td>
<td>25</td>
<td>50 ± 12</td>
<td>38 ± 9]</td>
<td>57 ± 20]</td>
<td>N/A</td>
<td>0</td>
</tr>
</tbody>
</table>

*BMI = body mass index; AI = apnea index; N/A = not available; UPPP = uvulopalatopharyngoplasty; CPAP = continuous positive airway pressure. Values are means ± SDs.
†Probability of 5-year survival ± SE.
‡Mortality and apnea index in obstructive sleep apnea.
§p<0.01 vs CPAP.
||p<0.001 vs CPAP.
|^p<0.01 vs untreated A1>20.

classified as responders. Those whose post-UPPP AI had decreased by less than 50 percent were classified as nonresponders.

Statistical Analysis

We used an unpaired two-tailed t test for continuous variables and a x² test for categorical variables to compare anthropometric and polysomnographic data between patients treated by nasal CPAP and by UPPP, and in subgroups with AI > 20/h. Survival curves were constructed for patients who had nasal CPAP and UPPP and their respective subgroups with AI > 20/h using the biomedical data program BMDP statistical package 1L (life table method). In addition, survival curves were constructed for nasal CPAP and the UPPP subgroup of patients that had not received prior treatment with nasal CPAP. We compared survival between groups using the generalized Wilcoxon statistic calculated using BMDP 1L.

RESULTS

One hundred fifty-four patients were treated with UPPP and 208 were treated with nasal CPAP during the 6-year period. Five of the 154 patients treated with UPPP were subsequently treated with nasal CPAP because of an unsuccessful response to surgery, and they were excluded from further analysis. Follow-up was complete in 137 of the remaining 149 patients. Twelve patients were unavailable for follow-up but were included in the survival analysis. Six deaths occurred among the remaining 137. Eighty-two (40 percent) of the 208 patients initially treated with nasal CPAP were no longer using this form of treatment at the time of follow-up. Forty-four of these 82 elected to have a UPPP, 3 a tracheostomy, 6 an intraoral appliance, and 29 were not using any specific therapy. Thirty-two of the 44 patients going on to UPPP were included in our UPPP group, the other 12 had their surgery after conclusion of the study period or in another center. Only the 126 patients who were using nasal CPAP at the time of contact were included in the initial analysis. Nine were unavailable for follow-up but were included in the survival analysis. Three deaths occurred among patients treated with nasal CPAP.

There was no difference in age between patients treated with UPPP and nasal CPAP (Table 1). There were more men among the patients treated with UPPP (p < 0.01), and these patients had a lower mean body mass index (p < 0.001), a higher mean AI (p < 0.01), and a longer mean duration of follow-up (p < 0.001).

There was no significant difference in survival between those patients treated by nasal CPAP and UPPP when either total patient groups (Fig 1) or subgroups with AIs greater than 20 (Fig 2) were considered. Also, there was no difference in survival between patients treated by nasal CPAP and the UPPP subgroup of patients that had not received prior treatment with nasal CPAP. Furthermore, no difference in survival was found between either UPPP responders and nonresponders, or between those with postoperative AIs greater than or less than 5/h.

![Graph showing probability of cumulative survival for patients with obstructive sleep apnea and nasal CPAP](http://publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21688/ on 06/16/2017)
In the six patients who died after UPPP, four died of a myocardial infarction, one died of massive hemoptysis secondary to lung cancer, and one in a boating accident. The three deaths in the nasal CPAP group were due to pneumonia, prostate cancer, and postoperative respiratory arrest (Table 2). Five of the patients died between 8 AM and 4 PM, two between midnight and 8 AM, and the remaining two at unknown times.

One hundred forty of the 149 UPPP patients (94 percent) had a follow-up polysomnogram. Eighty-nine of the 140 (63 percent) had a postoperative AI of less than 5 and a further 25 (18 percent) had a greater than 50 percent reduction in AI giving an overall 81 percent response rate. Patients who did not respond well to UPPP (< 50 percent decrease in AI) tended to be older (mean ± SD age, 55 ± 10 years vs 50 ± 11 years, p < 0.02); however, their body mass index and preoperative Al were not significantly different (29.7 ± 4.6 kg/m² for responders vs 30.8 ± 5.7 kg/m² for nonresponders and 25.6 ± 17.4/h vs 25.0 ± 14.0/h, respectively). All patients with nasal CPAP had an AI less than 5/h on follow-up polysomnography. There was no significant difference in posttherapy awake or asleep arterial oxygen saturation between survivors and non-survivors in either the patients with UPPP or nasal CPAP.

In the UPPP patient group, 3 of the 9 patients (33 percent) without a follow-up polysomnogram died compared with 3 deaths in the 140 patients (2.1 percent) with a follow-up polysomnogram. The follow-up AIs of the 3 patients with UPPP who died and had a follow-up polysomnogram were 23/h, 5/h, and 1/h. The patients in both treatment groups who died were older (mean age, of 60 ± 9.3 years, p < 0.001). But initial AI (mean of 34.3 ± 25/h) and body-mass indices (31.1 ± 9.2 kg/m²) were not significantly different from the survivors.

**DISCUSSION**

Our results suggest there may be no difference in long-term survival between patients with OSA treated with UPPP and nasal CPAP. Myocardial infarction was the most common cause of death, and daytime was the most common time of death. Mortality was much more common in the patients treated with UPPP who failed to return for follow-up polysomnography.

Patients with OSA appear to have decreased long-term survival. Partinen and colleagues reported decreased 5-year survival in patients with untreated OSA compared with both patients treated by tracheostomy and the US age-adjusted survival curve. He and coworkers demonstrated a decreased survival in patients with untreated OSA with an AI > 20/h compared with those patients treated by tracheostomy or nasal CPAP. This difference was most evident in patients younger than 50 years old. However, Gonzalez-Rothi and associates found no difference in survival between treated and untreated patients with OSA and a group of control patients. Unfortunately, the controls were not well matched for sex, weight, and other factors.

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**Table 2—Patient Deaths**

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age, yr</th>
<th>BMI, kg/m²</th>
<th>Pre-Rx AI, /h</th>
<th>Post-Rx AI, /h</th>
<th>Follow-up, yr</th>
<th>Cause of Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>UPPP</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 M</td>
<td>56</td>
<td>35</td>
<td>47</td>
<td>5</td>
<td>0.3</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>2 M</td>
<td>66</td>
<td>30</td>
<td>55</td>
<td>5</td>
<td>4.6</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>3 M</td>
<td>40</td>
<td>29</td>
<td>38</td>
<td>23</td>
<td>4.5</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>4 M</td>
<td>65</td>
<td>33</td>
<td>62</td>
<td>1</td>
<td>1.8</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>5 M</td>
<td>65</td>
<td>27</td>
<td>5</td>
<td>7</td>
<td>1.6</td>
<td>Lung cancer</td>
</tr>
<tr>
<td>6 M</td>
<td>64</td>
<td>22</td>
<td>7</td>
<td>0</td>
<td>2.6</td>
<td>Postoperative respiratory arrest</td>
</tr>
<tr>
<td>CPAP</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 F</td>
<td>76</td>
<td>18</td>
<td>68</td>
<td>0</td>
<td>2.6</td>
<td>Pneumonia</td>
</tr>
<tr>
<td>2 F</td>
<td>64</td>
<td>49</td>
<td>12</td>
<td>0</td>
<td>0.9</td>
<td>Prostatic cancer</td>
</tr>
<tr>
<td>3 M</td>
<td>62</td>
<td>37</td>
<td>16</td>
<td>0</td>
<td>0.5</td>
<td></td>
</tr>
</tbody>
</table>

*UPPP = uvulopalatopharyngoplasty; BMI = body mass index; Rx = treatment; AI = apnea index; CPAP = continuous positive airway pressure.
and the presence of significant comorbidity. One explanation for our finding of no difference in long-term survival between patients treated with UPPP and nasal CPAP is that untreated OSA may not decrease long-term survival. There is some support for this explanation in that we found no difference in survival between UPPP responders and nonresponders. In elderly patients the effect of OSA on long-term survival is less clear. Ancoli-Israel and colleagues showed an association between OSA and decreased survival in elderly women but not in men. Blwise and coworkers demonstrated no difference in mortality in a group of treated and untreated elderly patients with OSA compared with controls.

The major cause of increased mortality in OSA appears to be cardiovascular in nature. Partinen and associates found an age-adjusted odds ratio of vascular mortality of 4.7. Long-term follow-up of the same patients has shown a relative risk of developing new vascular problems of 2.3 (95 percent confidence interval of 1.5 to 3.6). Obstructive sleep apnea is associated with significant cardiac arrhythmias and cyclical variation of systemic blood pressure during sleep. Both chronic snoring, which usually precedes the onset of florid OSA, and OSA have been shown to be significant risk factors for the development of cardiovascular and cerebrovascular disease. The combined effects of systemic hypertension, hypoxemia, and increased sympathetic activity during sleep are thought to promote the development of atherosclerosis.

Cardiovascular death predominated in our study, with four of nine patients dying of myocardial infarction. One patient in the nasal CPAP group died of a respiratory arrest in the immediate postoperative period following unrelated surgery while not receiving nasal CPAP therapy. The other deaths do not appear to have been directly related to OSA. Although patients with OSA are more prone to cardiovascular-related deaths, it would be inappropriate to conclude that the four deaths in the UPPP group due to myocardial infarction and the one death in the nasal CPAP group due to postoperative respiratory arrest were attributable to OSA. The total number of deaths was small, and myocardial infarction-related deaths are common in the general population. The majority of our patients died during the daytime. This is similar to the findings of Gonzalez-Rothi and coworkers. However, this is different from the studies by Guilleminault and associates and Ancoli-Israel and coworkers, which found that patients with OSA were more likely to die during the night.

Nasal CPAP is generally established as the primary treatment for symptomatic OSA that has not responded to conservative measures. Nasal CPAP is not a realistic long-term treatment for some patients with OSA. Compliance rates vary considerably depending on the criteria for measuring compliance, experience in initiating treatment, adequacy of follow-up, types of CPAP machine and mask, and availability of other treatment modalities. Uvulopalatopharyngoplasty was proposed as a surgical treatment for OSA by Fujita and associates. The success of this operation varies considerably, and some of this variability has been accounted for by differences in patient selection or surgical technique. Both nasal CPAP and UPPP have been shown to reduce systemic blood pressure in patients with OSA.

In contrast to our study, He and associates found improved survival in those patients with OSA treated by nasal CPAP or tracheostomy but not in those treated by UPPP when compared with untreated patients. However, there are major differences between the two studies. In the study by He and associates the patients had more severe OSA, and only 40 percent had a successful surgical outcome compared with 81 percent in our study, using similar criteria to define success. Furthermore, the proportion of patients who underwent follow-up polysomnography after UPPP differs. In the study by He and associates 79 percent of patients had follow-up polysomnography compared with 94 percent in our study.

Our study's design has certain limitations. It, like all previous studies, is retrospective. Prospective controlled clinical studies are required to compare long-term survival between different treatments for OSA. However, such studies would currently be difficult to accomplish because of potential problems with both randomization and blinding. Furthermore, a long-term control untreated group would not be considered ethical as it is generally accepted that OSA is associated with increased mortality and morbidity. The issue of comorbidity confounds our results. The patients treated with nasal CPAP were more obese, and therefore at higher risk for associated systemic hypertension and ischemic heart disease. However, the patients treated with UPPP had more severe OSA, which would tend to counter the effect of increased body weight on mortality in the patients treated with nasal CPAP. Some of our patients were treated with nasal CPAP prior to UPPP and it could be argued that this nasal CPAP therapy had an impact on survival. We believe this is unlikely as the majority of these patients were only treated with nasal CPAP for 1 to 2 months and when we excluded them from the analysis, there was still no significant difference in long-term survival between patients treated with UPPP and nasal CPAP. Finally, the number of deaths was small, and our study may lack the power to establish differences in long-term survival between UPPP and nasal CPAP. Larger long-term studies may be required; however, our numbers are much larger (UPPP, 149 vs 60; nasal CPAP, 120 vs 25) than the study by He and associates, in which they demonstrated a significant decrease in
survival in patients treated with UPPP.

Follow-up polysomnography may be important after UPPP to identify those patients who require additional treatment because of ongoing OSA due to an unsatisfactory response to UPPP. Snoring, which is the hallmark symptom of OSA, is usually relieved by UPPP, but silent severe apnea may persist. Thirty-three percent (3/9) of our patients and 30 percent (6/20) of the patients in the study by He and associates who did not have follow-up polysomnography died.

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