Recurrence of Sleep Apnea Without Concomitant Weight Increase 7.5 Years After Weight Reduction Surgery

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In this study we report on a long-term follow-up of 14 morbidly obese sleep apneic patients, 11 of whom were male and 3 female. The mean age was 46 ± 8.5 years. These patients had undergone weight reduction surgery. Before surgery, body mass index (BMI) and apnea index (AI) were 45 ± 7.2 kg/m² and 40 ± 25.8 (SD) h⁻¹, respectively. Four and a half months after surgery (range, 2 to 7 months), both BMI and AI significantly decreased to 33 ± 7.5 kg/m² and 11 ± 16.4 h⁻¹, respectively. Seven and half years after surgery (range, 5 to 10 years), BMI increased only slightly to 35 ± 6.0 kg/m² (p > 0.5), while AI increased significantly to 24 ± 23 h⁻¹ (p < 0.05). There were poor and insignificant correlations between changes in BMI and AI prior to 4.5 months after operation (r=0.23; p > 0.4), and 4.5 months to 7.5 years after operation (r=0.41; p > 0.1). We conclude that morbid obesity is not the only causative factor in the sleep apnea syndrome for these patients. Weight reduction surgery alone does not "cure" their sleep apnea, and they are still at risk.

Methods and Patients

Fourteen morbidly obese patients with a diagnosis of obstructive sleep apnea (OSA) underwent weight reduction surgery (Roux-en-Y gastric bypass and vertical-banded gastroplasty). Previous it has been shown that weight reduction surgery is associated with a dramatic amelioration in the severity of SAS. In some patients, surgically achieved massive weight loss was associated with complete disappearance of the syndrome. The single study reporting on long-term follow-up of 57 obese SAS patients treated by weight reduction surgery indicated that apnea index (AI) fell from 64 ± 39 apneas per hour preoperatively to 33 ± 27 apneas per hour 1 year postoperatively, and then to 32 ± 32 (apneas per hour) 4.5 ± 2.3 years postoperatively. However, only seven patients underwent polysomnography in the long-term follow-up visit. The body mass index (BMI) at those times changed from 56 ± 12 to 37 ± 8 and to 38 ± 9 kg/m², respectively. Our observations, presented in this study, revealed a different course for the long-term effects of weight reduction surgery in a larger group of morbidly obese SAS patients.

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follow-up recordings are presented in Table 1. There were no significant differences in total sleep times between the three studies at the sleep lab. Before operation, all patients were symptomatic, with a mean AI of 40 ± 28.8 apneas per hour. The first postoperative recording (4.5 ± 1.8 months postoperatively; range, 3 to 7 months) revealed that weight reduction surgery was associated with a dramatic decrease in sleep apneas: AI decreased by 72.5%, from 40 ± 28.8 h⁻¹ to 11 ± 16.4 h⁻¹ (p < 0.005). Furthermore, 42.8% of the patients did not have any apneas. At that time, BMI decreased by 26.7% from 45 ± 7.2 kg/m² to 33 ± 7.5 kg/m² (p < 0.005). This was associated with a significant improvement in sleep. The percentage of deep sleep (stage 3 to 4) and rapid eye movement (REM) almost doubled from 12 ± 8% and 13 ± 7%, preoperatively to 23 ± 5 and 21 ± 4% short-term postoperatively (p < 0.005, Table 1).

Seven and a half years after surgery (range, 5 to 10 years), BMI increased slightly to 35 ± 6.0 kg/m², while there was a twofold increase in AI to 24 ± 23 h⁻¹ (p < 0.05). However, compared with preoperative findings, AI was still significantly lower (by 40%, p < 0.05). This was associated with a mild deterioration in sleep quality. The percentage of deep sleep and REM decreased to 18 ± 8% (p > 0.05) and to 18 ± 6% (p > 0.05). Compared with preoperative findings, the percentage of deep sleep and REM were still significantly higher (p < 0.05 for both [Table 1]).

To examine the possibility that the increase in AI 7.5 years postoperatively occurred only in patients who regained weight, correlational analysis was used to determine if there was a meaningful relationship between the amount of changes in body weight and AI.

There were poor correlations between changes in BMI and AI preoperatively to 4.5 months postoperatively (r=0.23; p > 0.4), and between 4.5 months and 7.5 years postoperatively (r=0.41; p > 0.1), indicating that the increase in AI was independent of changes in BMI. This lack of relationship is further demonstrated in Table 2. It shows that in 5 patients there was an increase in AI, 7.5 years postoperatively, in spite of a decrease or no change in BMI.

### Table 1—Body Mass Indices, Apnea Indexes, and Sleep Architecture Before Operation and a Short Time (Mean, 4.5 Months) and a Long Time After Operation (Mean, 7.5 Years)*

<table>
<thead>
<tr>
<th>Period</th>
<th>Body Mass Index, kg/m²</th>
<th>Apnea Index, h⁻¹</th>
<th>% Stage 1-2</th>
<th>% Stage 3-4</th>
<th>% Stage REM</th>
<th>Total Sleep Time, h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preoperative</td>
<td>45 ± 7.2</td>
<td>40 ± 29</td>
<td>76 ± 15</td>
<td>2 ± 8</td>
<td>3 ± 7</td>
<td>6.8 ± 1.0</td>
</tr>
<tr>
<td>4.5 mo postoperative</td>
<td>33 ± 7.5†</td>
<td>11 ± 16†</td>
<td>56 ± 7†</td>
<td>23 ± 5†</td>
<td>21 ± 4†</td>
<td>6.5 ± 0.7</td>
</tr>
<tr>
<td>7.5 yr postoperative</td>
<td>35 ± 6.0†‡</td>
<td>24 ± 23‡</td>
<td>65 ± 13‡</td>
<td>18 ± 8‡</td>
<td>18 ± 6‡</td>
<td>6.9 ± 1.2</td>
</tr>
</tbody>
</table>

*All data are mean ± SD.
†Significant differences are in comparison with preoperative recordings (p < 0.005).
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### Table 2—Number of Patients Classified According to Changes in Body Mass Index and Apnea Index in the Period Between Shortly After Surgery (Mean, 4.5 Months) to a Long Time After Surgery (Mean, 7.5 Years)

<table>
<thead>
<tr>
<th>Bodily Mass Index</th>
<th>Increase, 7.5 yr &gt; 4.5 mo</th>
<th>Decrease, 7.5 yr ≤ 4.5 mo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase, 7.5 yr &gt; 4.5 mo</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Decrease, 7.5 yr ≤ 4.5 mo</td>
<td>5</td>
<td>2</td>
</tr>
</tbody>
</table>

### Discussion

The present study revealed a recurrence of sleep apnea 7.5 years after weight reduction surgery in morbidly obese SAS patients, which could not be accounted for by weight increase. These results disagree with those of Sugarman et al.¹⁴ who reported that AI was lower at 4.5 years than 1 year postoperatively in 7 patients. The recurrence of sleep apneas in the present study agreed with patients’ subjective reports of exacerbated snoring and excessive daytime sleepiness at the time of the second postoperative recording. Consequently, in two of the patients constant positive airway pressure treatment was started shortly after the recordings.

The different courses of BMI and AI after surgery, and the poor correlations between the changes in BMI and AI, indicate two different primary etiologies for OSA in these morbidly obese patients. Our initial finding²,⁸,¹³ that in almost half of the patients massive weight reduction was associated with complete disappearance of the apneas shortly after the operation, which agrees with the short-term effects in the study of Sugarman et al.¹⁴ suggested a causative link between obesity and sleep apnea. Obesity, however, cannot be the single causative factor, since 60 to 80% of morbidly obese patients do not have SAS.⁸,¹⁰,¹⁴ Thus, it appears that obesity constitutes a risk factor only to certain individuals who are likely to have SAS. Previously, we reported that in some patients breathing disorders in sleep are caused by exacerbation of the preexisting tendency of the respiratory system to oscillate in sleep.¹⁹ This exacerbation can be caused by increased weight, increased upper airway resistance, decreased cortical arousability, or other risk factors.
factors. More recent research from our laboratory, as well as that of others, implicates a very significant genetic importance in sleep apnea. Hence, obesity may constitute a risk factor to individuals who are genetically susceptible to OSA. Since SAS rarely appears in people younger than 30 years, age must be considered an additional risk factor. The finding that in 5 of our patients, AI increased without a concomitant increase in BMI, or even in the face of an actual decrease in BMI (n=1), may suggest a shift in the relative risk of aging versus obesity in some individuals. Possibly, as a result of the operation, the patient’s weight was reduced to levels which did not constitute any risk of sleep apnea for that particular age. Seven and half years later, although there was no significant change in body weight, the amount of extra weight that remained, combined with the increased age, was sufficient to induce apneas. If this explanation is true, then it could be anticipated that younger patients would show the largest increase in AI between the second and third recordings. Although not significantly so, there was a tendency in this direction. There was a negative correlation of -0.4 (p <0.17) between the change in AI from the first to second postoperative recordings and the patient’s age. We could not find any other differences (eg, other medical illnesses or upper airway disease) in those five patients who had a higher AI without gaining weight that would have predisposed them to recurrent OSA.

One limitation of the present study that should be considered is that our analysis of apnea severity was based only on AI and clinical symptoms, since we missed some of the preoperative oxygen saturation data. In recent years, a much greater emphasis has been placed upon oxygen desaturation levels and hypopneas. Because postoperative oxygen desaturation values could not be compared with preoperative values, these data have been excluded.

What are the clinical implications of the present findings concerning the efficacy of bariatric surgery to treat morbidly obese SAS patients? In spite of the recurrence of sleep apnea 7.5 years after surgery, AI at that time was still significantly lower than preoperatively (24 vs 40; p<0.05). Considering this fact, and the observation that shortly after the operation about half of the patients were completely free of apneas, we believe the use of this treatment in morbidly obese patients is justified.

In summary, we found that BMI decreased after weight reduction surgery and increased only slightly after 7.5 years. Apnea index dramatically decreased after surgery, but disproportionately increased 7.5 years later. We conclude that both obesity and aging constitute risk factors for sleep apnea in predisposed individuals. Weight reduction surgery is an effective way of reducing BMI and AI for at least 7.5 years. However, despite massive weight reduction, this group of patients is still at risk for OSA possibly due to increasing age or other yet unexplained factors.

REFERENCES
5. Lavie P. Incidence of sleep apnea in a presumably healthy working population: a significant relationship with excessive daytime sleepiness. Sleep 1983; 6:312-18