Table 1—Four-Channel Studies, n=207

<table>
<thead>
<tr>
<th>Class</th>
<th>RDI</th>
<th>No.</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>0-5</td>
<td>69</td>
<td>33</td>
</tr>
<tr>
<td>Mild</td>
<td>5-20</td>
<td>66</td>
<td>32</td>
</tr>
<tr>
<td>Moderate</td>
<td>22-40</td>
<td>33</td>
<td>16</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt;40</td>
<td>39</td>
<td>19</td>
</tr>
</tbody>
</table>

nea recordings in the diagnosis and management of obstructive sleep apnea. The comments that followed in the January 1994,5 issue stimulated analysis of our own data in the use of similar recording devices.

Since 1987, we have performed outpatient, unattended, home sleep apnea monitoring, first with the Vitalog system (Vitalog Monitoring, Inc, Redwood City, Calif) and subsequently with the CNS Edentrace (Edentec, Eden Prairie, Minn) four-channel recording device similar to that reported in the aforementioned report. Since that time, we have performed or analyzed over 500 four-channel outpatient polysomnograms. For the years 1991, 1992, and 1993, we analyzed all of our unattended Edentrace four-channel studies. Technically adequate for interpretation were 164/189 (87%). The results are listed in Table 1.

A respiratory index (RDI) of five events per hour or less was considered within normal limits. Thirty-three percent of the studies were normal, 32% of studies revealed mild sleep apnea hypopnea (RDI between 5 to 20), 16% showed moderate sleep disordered breathing (RDI between 20 to 40 events per hour), and 19% revealed severe obstructive sleep apnea hypopnea (RDI greater than 40 events per hour).

Of those patients in the moderate to severe category, nasal CPAP or ventilatory support system therapy was instituted in the home environment in 42/72 cases (58%). All of these patients were successfully titrated in the home environment with an excellent therapeutic response. The remaining patients in the moderate and severe categories elected other forms of therapy or declined therapeutic intervention.

We agree with Dr. Coppola and Mr. Lawee that unattended monitoring has a role in the screening and diagnosis of obstructive sleep apnea hypopnea syndrome. In addition, we have found home titration of nasal CPAP to be effective and well tolerated. We hasten to add and caution that the limitations of four-channel unattended monitoring include the inability to diagnose other sleep disorders such as narcolepsy, periodic leg movement disorders, and idiopathic hypersomnia. In addition, attention to body position is important and failure to achieve delta wave or rapid eye movement sleep may alter the respiratory disturbance index. We have not found this to be a significant problem.

We do, however, believe that extensive clinical screening including a detailed sleep apnea questionnaire and physician interview should precede four-channel apnea monitoring. Since analysis of the aforementioned data, we have embarked on this approach to decrease the percentage of normal studies. We believe this approach is most useful in screening and diagnosing patients with moderate or severe obstructive sleep apnea syndrome where the index of suspicion for disease is high in an effort to control cost. In addition, this approach may be useful in situations where the prior probability for disease is low. We encourage further research and investigation in this area and look forward to these results.

Barry S. Dicicco, MD, FCCP, and Thomas J. LoRusso, MD, FCCP, Pulmonary Diseases and Critical Care Medicine, Fairfax, Virginia

References

2 Sackner MA. Obstructive sleep apnea [letter]. Chest 1994; 104:320
3 Coppola MP. Obstructive sleep apnea [letter]. Chest 1994; 104:320-21

OSA Treatment

UPPP vs N-CPAP

To the Editor:

We have carefully read the article on “Long-Term Survival of Patients With Obstructive Sleep Apnea Treated by Uvulopalatopharyngoplasty or Nasal CPAP” by Keenan et al in the January 1994 issue of Chest.

A hundred and forty-nine patients with obstructive sleep apnea (OSA) were treated by uvulopalatopharyngoplasty (UPPP) and 126 by nasal CPAP (N-CPAP). The apnea index was modestly but significantly higher in the patients treated by UPPP. The authors observed six deaths in the group treated by UPPP and three deaths in the group treated by N-CPAP. They concluded that there is no difference in the cumulative survival between the two treatments.

Several comments are worthwhile concerning this study.

First, the fact that patients unavailable for follow-up were included in the survival analysis is worrisome. All or some of them could have died in the intervening period.

Second, if we exclude deaths due to cancer and pneumonia, clearly unrelated to OSA, and to an accident, although a boating accident could be due to the “driver” falling asleep, we are left with four deaths from myocardial infarction in the group of patients treated by UPPP; in the group of patients treated by N-CPAP, there was one death from postoperative respiratory arrest at a time when the patient was not using his N-CPAP treatment. Postoperative deaths in untreated patients with OSA may be a direct consequence of their disease. Indeed, in the postoperative period, patients with OSA are extremely vulnerable since they are treated with opioids, sedatives, and anesthetics, all of which have a deleterious effect on sleep apnea. Thus, although this patient was in the N-CPAP treated group, he should not be considered as one who died from therapy at the time of death.

Therefore, the data show four patients dying of cardiovascular-related death, which is probably linked to OSA, in the group treated by UPPP, against one death, probably also related to OSA at a time when the patient was not treated, in the group treated by N-CPAP. With these assumptions and excluding patients lost to follow-up, we have performed a x^2 test comparing mortality in the two groups (4 out of 137 vs 0 out of 117). This test showed a significant difference between the two groups (p<0.05).

Third, the absence of cardiovascular-related deaths in the group treated by N-CPAP is even more remarkable, if one considers that patients in this group were significantly more obese (BMI 36 kg/m^2 vs 30 kg/m^2 in the group treated by UPPP).

Fourth, it is unfortunate that the apnea-hypopnea index was not given in the report. Indeed, it has been shown that after UPPP patients with a transpalatal obstruction improved the apnea, but not the hypopnea component of their sleep related breathing disorder, whereas the opposite held true in patients with hypopharyngeal obstruction.

Fifth, the discussion about time of death does not tell the reader...
at what time the myocardial infarction did occur.

Sixth, we fully agree with the authors that UPPP should be mandatorily followed by a polysomnography after 2 to 3 months to assess the result of surgery.

In conclusion, we are left with the impression that mortality related to OSA, ie, cardiovascular death, may well be higher in patients treated with UPPP than in patients treated with N-CPAP.

Daniel Rodenstein, MD, PhD, Philippe Collard, MD, and Genevieve Aubert, MD, PhD; Cliniques Universitaires Saint Luc, Catholic University of Louvain, Brussels, Belgium

REFERENCES

To the Editor:

I am pleased to comment on the points raised by Dr. Rodenstein et al in their letter about our article, “Long-Term Survival of Patients With Obstructive Sleep Apnea Treated by Uvulopalatopharyngoplasty or Nasal CPAP” (Chest 1994; 105:155-59).

First, they express concern over the fact that patients unavailable for follow-up were included in the survival analysis, and all or some of these patients could have died in the intervening period. Twelve of the 149 patients treated by UPPP and 9 of the 126 patients treated by nasal CPAP (N-CPAP) were lost to follow-up. Follow-up loss of patients is unavoidable in any study over 6 years. We are confident that very few, if any, of these patients died, because we submitted their names to the Government of British Columbia Vital Statistics Department. The Vital Statistics Department records the names of those patients who die within British Columbia. Consequently, we believe it was appropriate to include the patients lost to follow-up in the survival analysis.

Second, we do not believe it is appropriate to reanalyze the data excluding five of the nine deaths and only include the four patients dying of a cardiovascular-related death because Dr. Rodenstein and colleagues believe this to be linked to OSA. Patient compliance is the major problem with N-CPAP therapy, and I believe it is quite appropriate to include the patient in the N-CPAP group who died of a respiratory arrest in the immediate postoperative period after unrelated surgery, while not on N-CPAP therapy. This death occurred in another center, and we agree it highlights the importance of complete compliance with N-CPAP, especially while receiving sedation or analgesia. This point was made in the original draft of our manuscript and was removed at the request of one of the reviewers.

Third, their comment that the absence of cardiovascular-related deaths in the group treated by N-CPAP is even more remarkable because the patients in this group were significantly more obese. We believe it is inappropriate to conclude that the four cardiovascular-related deaths were necessarily attributable to OSA. The total number of deaths was small and cardiovascular-related deaths are common in the general population.

Fourth, we provided the apnea index rather than the apnea/hypopnea index so that our data could be compared with the data from He et al who also provided apnea index. It has been our experience that after UPPP, the apnea/hypopnea index decreases proportionally to the same degree as the reduction in the apnea index.

Fifth, in most patients, we established the time of death from their death certificate, and we could not accurately establish the time of the myocardial infarctions.

Sixth, we agree with Dr. Rodenstein et al that UPPP should be mandatorily followed by polysomnography. In the final paragraph of the original version of our manuscript, we stated “follow-up polysomnography is essential after UPPP....” In response to a reviewer’s comment, we modified this recommendation to “follow-up polysomnography may be important after UPPP....”

We thank Dr. Rodenstein et al for their comments and interest in our article but see no reason to change our original conclusion that there is no difference between the long-term survival of patients with OSA who are treated with UPPP and who are treated with N-CPAP. In our article, we acknowledged and discussed the limitations of our study and stated that further prospective controlled clinical studies are required to compare long-term survival between different treatments for OSA.

John A. Fleetham, MB, Department of Medicine, Vancouver Hospital & Health Sciences Centre, Vancouver, British Columbia, Canada

REFERENCE

N-CPAP in Patients With COPD in Acute Respiratory Failure

To the Editor:

We read with interest in the December 1993 issue of Chest the paper of de Lucas et al 1 on the use of nasal continuous positive airway pressure (N-CPAP) in patients with COPD in acute respiratory failure.

We have previously reported that N-CPAP may be used in long-term treatment of various clinical conditions besides obstructive sleep apnea syndrome, such as end-stage lung disease from different causes (COPD, idiopathic lung fibrosis, or lung cancer).2,3

In these conditions, N-CPAP at low pressure (3 to 5 cm H2O) decreases dyspnea, improves oxygenation with lower flux of oxygen in respect to conventional delivery by nasal prongs or Ventimask, and allows us to reduce the hospital stay of severely ill patients.

In particular, we have observed that N-CPAP is a good tool for oxygen administration in COPD patients with severe hypoxia and