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 al1 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 al1 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