

Case reports

Transtracheal air in the treatment of obstructive sleep apnoea hypopnoea syndrome

F Sériès, J La Forge, N Lampron, Y Cormier

Abstract

A 49 year old woman with typical obstructive sleep apnoea hypopnoea syndrome underwent an unsuccessful trial with continuous positive airway pressure (CPAP) followed by uvulopalatopharyngoplasty with septorhinoplasty, treatment with protriptyline, and a second CPAP trial that was abandoned. Transtracheal air was then given and normalised sleep and breathing at a flow rate of 5 l/min. A sustained clinical improvement was observed at follow up visits. Transtracheal air could represent a simple and effective alternative to tracheotomy in patients with obstructive sleep apnoea hypopnoea syndrome in whom conventional treatments fail.

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Keywords: obstructive sleep apnoea; hypersomnolence; transtracheal air; pneumatic splinting

Tracheotomy was the reference treatment for obstructive sleep apnoea hypopnoea syndrome (SAHS) until about 15 years ago,¹ since when other effective treatment methods²⁻⁴ have been developed. However, tracheotomy may still be indicated as the ultimate solution in some patients whose nocturnal breathing disorders persist or who do not tolerate continuous positive airway pressure (CPAP) or mandibular prostheses.

Transtracheal oxygen is an effective treatment of hypoxia in chronic lung disease.⁵ Nocturnal breathing disorders can improve or normalise with transtracheal oxygen given at a flow rate of 2-3 l/min in patients with obstructive SAHS with diurnal hypoxaemia and severe nocturnal desaturation.^{6,7} However, little or no change in the frequency of respiratory events

was found with transtracheal air.⁷ A trial was undertaken using a moderate airflow to determine whether increasing the flow rate could further improve obstructive breathing disorders via pneumatic splinting of upper airway structures.

We report the case of a patient with obstructive SAHS who had been unsuccessfully treated with conventional treatments and whose breathing disorders were completely reversed with transtracheal air.

Case report

A 49 year old obese white woman (body mass index (BMI) 30.3 kg/m²) was first evaluated in May 1989. She was treated for systemic hypertension with verapamil and had undergone an adenoidectomy during childhood. Her symptoms were characteristic of obstructive SAHS. Examination of the upper airway revealed a deviation of the nasal septum, macroglossia, and a posterior position of the soft palate. Thyroid function, pulmonary function tests, and arterial blood gas tensions were normal. A sleep study confirmed the diagnosis of obstructive SAHS (table 1). All apnoeic and hypopnoeic events were obstructive in type.

CPAP was initiated at home at a pressure of 7 cm H₂O but it was rapidly terminated due to mask and pressure discomfort. Uvulopalatopharyngoplasty (UPPP) and septorhinoplasty were then performed but a sleep study carried out four months later showed no improvement in nocturnal sleep disorders (table 1). The patient refused CPAP and a mandibular prosthesis. Protriptyline was initiated in a daily dose of 20 mg with no improvements in clinical symptoms and nocturnal breathing (table 1). Another trial with CPAP was therefore performed at an effective pressure level of 10 cm H₂O (BMI now 32.6 kg/m²). The treatment was well tolerated during the first three

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Table 1 Results of the different sleep studies

| | Diagnosis (July 1989) | First CPAP trial (July 1989) | UPPP (July 1992) | Protriptyline (October 1992) | Second CPAP trial (October 1992) | Follow up (January 1997) | Transtracheal air (January 1997) |
|----------------------------------|--------------------------|---------------------------------|---------------------|---------------------------------|-------------------------------------|-----------------------------|-------------------------------------|
| Total sleep time (TST) (h) | 6.7 | 5.3 | 3.7 | 5.4 | 7.6 | 7.0 | 7.5 |
| Stage I-II (% TST) | 71.1 | 39.1 | 71.9 | 78.2 | 48.7 | 76.0 | 51.2 |
| Stage III-IV (% TST) | 15.5 | 30.5 | 15.5 | 12.0 | 31.0 | 10.1 | 32.7 |
| Stage REM (% TST) | 13.4 | 30.4 | 12.6 | 9.8 | 20.3 | 13.9 | 16.1 |
| Apnoea + hypopnoea index (no./h) | 52.0 | 14.6 | 55.0 | 56.2 | 11.3 | 48.1 | 7.2 |
| Arousal index (no./h) | — | — | 45.0 | 55.0 | 10.0 | 54.7 | 8.6 |
| % TST <90% Sao ₂ | 31.5 | 4.7 | 40.0 | 45.7 | 3.0 | 21.6 | 1.6 |

CPAP = continuous positive airway pressure; UPPP = uvulopalatopharyngoplasty.

months but the patient did not return for the follow up visit.

She was re-evaluated four years later following definitive interruption of CPAP therapy (table 1). The therapeutic alternatives were reviewed and it was decided to evaluate transtracheal air before proceeding to a tracheotomy. A Scoop II[®] catheter was inserted under local anaesthesia.⁶ Repetitive night time oximetry measurements were carried out at different flow levels to determine the effective flow rate. Periodic desaturations persisted at 1.5 l/min and 3 l/min although they improved at the higher flow rate (6% recording time below 90% SaO_2). These events were completely abolished at a flow rate of 5 l/min. At this flow rate the sleep structure and nocturnal breathing disorders normalised. The shape and dimensions of the upper airway measured by computerised tomography during wakefulness did not change with increasing airflow. The patient was sent home with this treatment using an oxygen concentrator without a molecular filter as an airflow generator. Sustained clinical improvement was observed at follow up visits. Unfortunately, several episodes of skin infection around the catheter stoma occurred during the following months that led to withdrawal of the transtracheal catheter after 11 months of use, and a tracheotomy was then performed.

Discussion

Transtracheal air may represent an effective treatment for obstructive SAHS. There may be several reasons for the apparent discrepancy between our findings and previously published results with transtracheal air in obstructive SAHS.⁷ A flow rate of 2 l/min and 3 l/min was used in previous studies. In our patient periodic desaturations⁸ persisted at suboptimal flow rates. This suggests that the effectiveness of transtracheal air in obstructive SAHS is dependent on the flow rate. Another important difference is that the transtracheal flow rate was previously adjusted to maintain SaO_2 above 90%. Since transtracheal air may increase the lung volume, it may act as passive lung inflation and improve apnoea/hypopnoea related falls in SaO_2 with persisting abnormal respiratory disturbances⁹ at a moderate rate of flow.

Previous reports concluded that the effectiveness of transtracheal oxygen is mainly the

result of a stabilisation of chemoreceptor activity. Our results suggest that the mechanical effects of transtracheal air can prevent obstruction of the upper airway. We speculate that it may act as an autocontrolled intrathoracic CPAP. With transtracheal air the changes in intrathoracic pressure should be determined by the spontaneous ventilatory pattern and by expiratory upper airway resistance, excess flow being freely expired through the open upper airway. Inspiratory and expiratory upper airway resistance both progressively increase during the breathing cycles that precede the onset of obstructive apnoea.¹⁰ As soon as these resistances increase, transtracheal air increases intrathoracic and extrathoracic airway pressure, resulting in lung inflation and in pneumatic splinting of the upper airway. Any rise in upper airway resistance will therefore be automatically counterbalanced by an increase in the passive transmural dilating force and maintenance of patency of the upper airway.

Transtracheal air could represent a simple and effective alternative to tracheotomy and could therefore be used in selected motivated patients in whom conventional treatments have failed.

- 1 He J, Kryger MH, Zorick FJ, *et al.* Mortality and apnea index in obstructive sleep apnea. *Chest* 1988;**94**:9–14.
- 2 Sullivan CE, Issa FQ, Berthon-Jones M, *et al.* Reversal of obstructive sleep apnea by continuous positive airway pressure applied through the nares. *Lancet* 1981;**i**:862–5.
- 3 Fujita S, Conway W, Zorick FJ, *et al.* Evaluation of the effectiveness of uvulopalatopharyngoplasty. *Laryngoscope* 1985;**95**:70–4.
- 4 O'Sullivan RA, Hillman DR, Mateiljan R, *et al.* Mandibular advancement splint: an appliance to treat snoring and obstructive sleep apnea. *Am J Respir Crit Care Med* 1995;**151**:194–8.
- 5 Chisiofer KL, Spofford BT, Peturn MD, *et al.* A program for transtracheal oxygen delivery: assessment of safety and efficacy. *Ann Intern Med* 1987;**107**:802–8.
- 6 Chauncey JB, Aldrich MS. Preliminary findings in the treatment of obstructive sleep apnea with transtracheal oxygen. *Sleep* 1990;**13**:167–74.
- 7 Farney RJ, Walker JM, Elmer JC, *et al.* Transtracheal oxygen, nasal CPAP, and nasal oxygen in five patients with obstructive sleep apnea. *Chest* 1992;**101**:1228–35.
- 8 Sériès F, Marc I, Cormier Y, *et al.* Utility of nocturnal home oximetry for case finding in patients with suspected sleep apnea-hypopnea syndrome. *Ann Intern Med* 1993;**119**:449–53.
- 9 Sériès F, Cormier Y, Lampron N, *et al.* Influence of lung volumes in sleep apnoea. *Thorax* 1989;**44**:52–7.
- 10 Sanders MH, Moore SE. Inspiratory and expiratory partitioning of airway resistance during sleep in patients with sleep apnea. *Am Rev Respir Dis* 1983;**127**:554–8.