

Short reports

Minitracheotomy: a simple alternative to tracheostomy in obstructive sleep apnoea

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ABSTRACT A patient with obstructive sleep apnoea sufficiently severe to cause papilloedema was managed temporarily with a minitracheotomy. This allowed arterial oxygen saturation to return to normal while he lost weight, before surgery for his enlarged tonsils.

Most patients with obstructive sleep apnoea are managed successfully with a combination of non-surgical measures. Surgery is limited to the removal of obvious physical obstructions, the occasional plastic surgery procedure, and formal tracheostomy, which may still be necessary in the initial treatment of severe cases or when other treatment has failed. In view of the risks and disadvantages of tracheostomy, we report a patient with severe obstructive sleep apnoea who was managed successfully by the temporary insertion of a minitracheotomy tube.

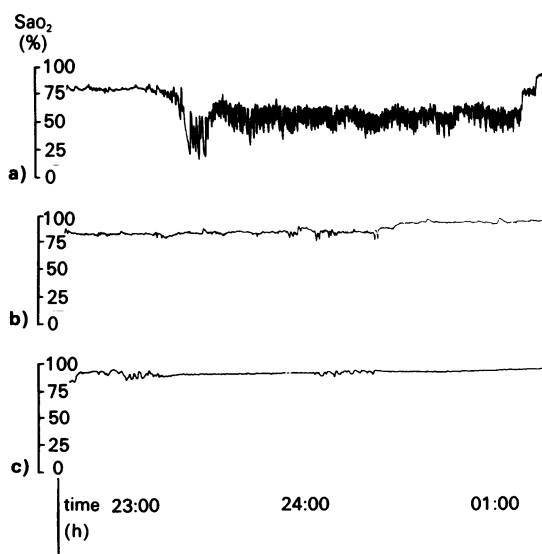
Case report

A non-smoking Asian man, aged 44 years, was admitted to hospital for investigation of headaches and papilloedema. For the previous eight years he had been treated with regular venesection for polycythaemia, which was discovered during a pneumonic illness. He had a choanal polypectomy for nasal obstruction at the age of 33 and bilateral anterior turbinectomies at the age of 36. During his present admission his relatives volunteered a history of loud snoring and periodic apnoea and arousal during sleep. The patient complained of daytime somnolence and a socially inappropriate sleep pattern.

On examination he was obese (weight 102 kg, height 146 cm, body mass index 47.9) and had peripheral oedema, central cyanosis, bilateral papilloedema, a poor gag reflex, and grossly enlarged tonsils. No other abnormality was found from systemic examination. The haemoglobin concentration was 19.6 g/dl, leucocyte count $7.3 \times 10^9/l$, and packed cell volume 0.67. His biochemical profile, including thyroxine concentration, was normal. His lung function test results were essentially normal: FEV 2.46 l (83% predicted), forced vital capacity (FVC) 3.19 l (88%), FEV₁/FVC 77%, total lung capacity 5.21 l (85%), transfer factor for carbon monoxide (TlCO) 9.2 (97%), and Kco 1.94. His chest

radiograph showed cardiomegaly and prominent hila and the electrocardiogram showed peaked P wave and T wave inversion across the chest leads. Arterial blood gas analysis with the patient breathing air and awake showed hypoxaemia and hypercapnia (arterial oxygen tension (Pao₂) 5.46 kPa, arterial carbon dioxide tension (Paco₂) 7.70 kPa). Overnight oximetry (Hewlett Packard, 47201A) on the first night in hospital showed a dramatic fall in oxygen saturation (Sao₂) from a baseline of 80% to a minimum recorded value of 20% (figure). Arterial blood gas samples taken during the night also showed increasing hypercapnia (Paco₂ 12.0 kPa).

A clinical diagnosis of cor pulmonale secondary to obstructive sleep apnoea was made. Urgent airway control was considered appropriate in view of the severity of the condition and the coexisting nasal obstruction. Nasal continuous positive airway pressure was not available and an endoscopic ear, nose, and throat examination showed



Representative portions of overnight oximetry traces on three occasions: (a) Before treatment. (b) Twenty four hours later with a 5 mm minitracheotomy tube, 5 cm H₂O continuous positive airway pressure, and 30% oxygen. For the remainder of the night the arterial oxygen saturation (Sao₂) was over 80%. (c) One month later, with the minitracheotomy only, the Sao₂ remained above 90% for the whole night.

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Accepted 6 December 1988

obstruction of the posterior choana by nasopharyngeal lymphoid tissue. Control was obtained via a minitracheotomy tube (Portex UK Ltd, internal diameter 5 mm) inserted through the cricothyroid membrane under local anaesthesia, the open technique being used.

Oxygen was administered directly into the trachea through the cannula, initially with a continuous positive airway pressure of 5 cm H₂O. The continuous positive airway pressure was applied via a connector and T piece with a fresh gas flow of 16 l/min to a water trap. The mixture of air and oxygen was adjusted to give an inspired oxygen concentration of 30%. This achieved an immediate improvement in oxygenation (Pao₂ 9.7 kPa), stability of Sao₂ (figure), and a profuse diuresis of 7 l in 24 hours. As the signs of cor pulmonale and papilloedema resolved continuous positive airway pressure and supplemental oxygen were withdrawn. One month after admission his nocturnal oxygen saturation was normal (figure) and he had lost 10 kg in weight. He was discharged home with the minitracheotomy tube in place. Two months later he had a tonsillectomy and the minitracheotomy tube was removed after two weeks. At the six month follow up the patient was back at work with no symptoms of obstructive sleep apnoea and with resolution of the electrocardiographic changes.

Discussion

The management of obstructive sleep apnoea has evolved during the last decade owing to a better understanding of the underlying pathophysiology. Formal tracheostomy used to be required frequently but has now been largely superseded by the pneumatic support of the airway with nasal continuous positive airway pressure. Tracheostomy may still be necessary, however, when the condition is severe or when nasal continuous positive airway pressure cannot be tolerated.¹ Conventional tracheostomy as a permanent treatment has cosmetic disadvantages and impairs speech and cough. It may also produce appreciable morbidity and even mortality in obstructive apnoea if the neck is obese and short.² Intratracheal delivery of oxygen through a small cannula has been shown to maintain nocturnal oxygenation in patients with obstructive sleep apnoea, though it cannot prevent obstructive episodes.³ The 5 mm minitracheotomy tube provides an airway and a route for oxygen delivery with little risk. It was developed to provide access for suction in sputum retention⁴ but can if necessary support spontaneous ventilation.⁵ In patients with a short, fat neck the procedure should be performed under direct vision by the open method of cannulation to ensure correct placement.⁶

This case of obstructive sleep apnoea is unusual in its severity, presenting with polycythaemia and cor pulmonale. The response to relief of the upper airway obstruction was complete, and disclosed no underlying pulmonary abnormality. The development of nocturnal obstruction was presumably due to a combination of nasal obstruction, tonsillar enlargement, and obesity. The mechanism of airflow limitation in these patients may be a complex interaction of physical narrowing, reduction in muscle tone, and increased compliance leading to airway collapse on inspiration. The effectiveness of the minitracheotomy was not solely due to diversion of the tidal volume since the presence of even a small leak beyond the obstruction may have reduced the pressure gradient sufficiently to have prevented airway collapse. In addition, the minitracheotomy was a conduit for secure oxygenation and the provision of internal pneumatic splinting with continuous positive airway pressure.

Although we would not necessarily recommend minitracheotomy for long term treatment, this airway was tolerated for four months, allowing the patient to retain speech and cough, until definitive treatment was effective. We believe that the minitracheotomy may provide a simple temporary alternative to tracheostomy in obstructive sleep apnoea.

We wish to express our grateful thanks to Dr D E Stableforth for permission to report this case.

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