Minitracheotomy: a simple alternative to tracheostomy in obstructive sleep apnoea

SIR,—Mr A Hasan and colleagues (March 1989;**44**:224–5) describe the use of a minitracheotomy tube in the management of a patient with severe obstructive sleep apnoea and advocate its use in other cases. I was consulted on the management of this patient when he first presented and recommended a formal tracheostomy. There is no doubt that the minitracheotomy he received was sufficient to enable him to recover from right heart failure, but I believe it to have been a less than optimal treatment.

The recordings of oxyhaemoglobin saturation show that, although severe hypoxaemia was prevented, there were still multiple episodes of partial airway obstruction resulting in arousal and subsequent sleep fragmentation. This was confirmed by the patient at subsequent consultations in this clinic. His excessive daytime sleepiness did not resolve until after the nasal surgery and tonsillectomy performed by Mr R S A Thomas at Leicester Royal Infirmary. Airway management during the surgery was complicated by the presence of the minitracheotomy. The minitracheotomy was poorly tolerated by the patient. The 15 mm connector attached to the tube protruded sufficiently to catch on his clothing and its weight caused the intratracheal portion of the tube to move around, causing bouts of coughing. Secretion control was difficult as the tube could not be easily removed for cleaning, unlike a formal silver tube, and humidification was almost impossible. He was unable to return to work with the tube in situ. It was removed by me because of these problems, despite the continuing presence of mild obstructive sleep apnoea at a sleep study after the tonsillectomy. The patient has subsequently relapsed as he has failed to reduce his weight as directed, and is at present well established on nasal continuous positive airway pressure with complete resolution of all his symptoms.

The resistance to airflow of a 5 mm endotracheal tube is much greater than that of the normal upper airway[1] and would be further increased by any secretions. It is possible that the reduction in total airway resistance after its insertion would be sufficient to prevent pharyngeal collapse in some subjects but its efficacy would be difficult to predict. Tracheostomy is advocated only as an initial treatment in patients with life threatening cardiac failure, and it would appear logical to adopt the technique which guarantees an adequate airway at the lowest resistance to airflow and also permits ready institution of intermittent positive pressure ventilation either for correction of respiratory failure or for surgery.

I do not believe the minitracheotomy to be an appropriate form of airway management for patients with obstructive sleep apnoea and life threatening cardiac failure. They should be managed with either nasal continuous positive airway pressure or formal tracheostomy.

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1 Nunn JF. *Applied respiratory physiology*. 2nd ed. London: Butterworth, 1977.

AUTHORS' REPLY We thank Dr Hanning for his comments and acknowledge his contribution to the long term management of this patient. We also accept his view that conventional tracheostomy and nasal continuous positive airway pressure may be superior methods of obtaining airway control in the acute management of obstructive sleep apnoea. It is important to remember, however, that emergency tracheostomy carries considerably more operative risk to the patient than minitracheotomy and is also likely to produce greater morbidity in terms of tracheal stenosis, skin ulceration, sepsis, and secondary haemorrhage, as well as interfering with the function of the glottis; it is therefore a matter not of one method being right and the other wrong but of weighing up the advantages and disadvantages of each. There is also the point that nasal continuous positive airway pressure may not always be available outside special centres.

We do not believe that we made excessive claims for the role of minitracheotomy but reported the case in order to indicate that there is a relatively safe and effective temporary alternative to traditional methods of management. We have not suggested the use of the device for an indefinite period, though we know that other patients have tolerated it for many months. Centres which specialise in sleep disordered breathing may not find it the ideal method of management, but where expert facilities are not available the minitracheotomy may have an important role.

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Alveolitis associated with sulphamethoxypyridazine

SIR,—We were very interested to read of the patient reported by Dr CL Steinfort and others (April 1989;**44**:310–1), who describe the association of alveolitis with sulphamethoxypyridazine, as we have recently treated a similar case.

A 49 year old woman with bullous linear IgA disease was treated with dapsone, which had to be withdrawn owing to a febrile reaction associated with lymphadenopathy and malaise. Subsequently she received sulphamethoxypyridazine 250 mg thrice daily, increasing to 500 mg thrice daily, and prednisolone 15 mg daily for six months, during which time she became increasingly short of breath. At presentation she was tachypnoeic with poor chest expansion, vesicular breath sounds, and basal crackles. Her arterial oxygen tension fell to 6·2 kPa and her FEV_1 and FVC to 0·5 and 0·8 l. Chest radiography showed diffuse interstitial shadowing throughout the lower and mid zones bilaterally. In view of the association of the underlying skin condition with malignancy, especially lymphoma,[1] the patient proceeded to open lung biopsy. There was extensive focal interstitial fibrosis, some oedema, minor lymphocyte infiltration, and the presence of type 2 pneumatocytes.

These findings were thought most likely to represent drug induced lung disease. Sulphamethoxypyridazine was withdrawn and prednisolone increased to 60 mg daily, partly to keep the skin disease in remission. Within five weeks the arterial oxygen tension had returned to 13·6 kPa, the FVC to over 2 l, and the chest radiograph had cleared.