

### **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<sup>1</sup> 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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<sup>1</sup> 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 sulphamethoxy-pyridazine**

SIR,—We were very interested to read of the patient reported by Dr CL Steinfors and others (April 1989;44:310–1), who describe the association of alveolitis with sulphamethoxy-pyridazine, 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 sulphamethoxy-pyridazine 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<sub>1</sub> 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,<sup>1</sup> 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. Sulphamethoxy-pyridazine 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.

Long acting sulphonamide type drugs may be associated with alveolitis more commonly than is currently realised. Minor degrees of dyspnoea may not be picked up by doctors unaware of the association. Routine three monthly spirometry and possibly chest radiography may be indicated for people taking these drugs. Two particular aspects of our patient's history are the previous dapsone reaction, suggesting generalised hypersensitivity to sulphur drugs, and the onset of lung disease despite her taking corticosteroids.

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#### Cardiovascular and hypokalaemic effects of inhaled salbutamol, fenoterol, and isoprenaline

SIR,—In their paper (February 1989;44:136–40) Dr J Crane and coworkers concluded that fenoterol evoked more pronounced hypokalemia and stronger ( $\beta_1$  mediated) inotropic responses than did salbutamol. We would, however, like to raise some questions regarding interpretations and conclusions which have not been addressed by Dr Crane and his colleagues.

Results concerning the main parameter of the study (plasma potassium concentration) are presented in terms of changes from baseline but with no baseline values given. Placebo caused a substantial increase in plasma potassium, indicating that baseline values were not stable. Were baseline values identical on the different days? The increase in potassium after placebo was actually greater than the reduction after salbutamol—would this increase have been identical on the different days? As  $\beta$  adrenoceptors can be up or down regulated (time and dose dependent changes) and receptors in different tissues may respond differently (see, for example, Harvey *et al*)<sup>1</sup> it would also have been of interest to know if there were possibilities for carry over effects between treatments—what time span was required between studies and are there data on effects on sodium-potassium ATPase activity on renewed  $\beta$  stimulation after provocation with a  $\beta$  agonist? Tolerance may well have developed after a dose of a long acting agonist such as fenoterol.

The authors state that the greater cardiac stimulation apparently afforded by fenoterol was due to more pronounced  $\beta_2$  adrenoceptor activation. The human heart, however, is richly endowed with  $\beta_2$  adrenoceptors, which in most studies comprise about one third of the cardiac  $\beta$  adrenoceptor population<sup>2</sup>; functionally these  $\beta_2$  adrenoceptors seem to be even better coupled to adenylate cyclase than are the  $\beta_1$  adrenoceptors (see, for example, Kauman and Lemoine<sup>3</sup>). Thus fenoterol may well have stimulated the heart via  $\beta_2$  adrenoceptor stimulation. Furthermore, earlier studies did not show any difference in  $\beta_2$  selectivity between salbutamol and fenoterol given in therapeutic doses.<sup>4</sup>

The time dependence of the effects, seen in relation to the pharmacokinetics of the drugs, has not been adequately discussed. Lipid solubility (which is greater for fenoterol) and local metabolism (which is extensive for the rapidly *O* methylated catecholamine isoprenaline) are factors to be considered. Cumulation of the three drugs may have been quite different with the protocol used. No plasma concentration-effect evaluation was performed. Furthermore, the authors provide no evidence that fenoterol and salbutamol are equipotent on a milligram for milligram basis with regard to bronchodilatation (the recommended dosages for fenoterol are lower than those for salbutamol in Sweden)—were equipotent doses studied?

Most importantly, however, we think that it is unjustified to extrapolate findings with single doses of the drugs given to healthy volunteers to chronic treatment in asthmatic subjects. The study does not allow any conclusions about chronic effects of fenoterol or salbutamol (in adequate therapeutic dosages) in the long term—that is, when receptor adaptation, etc, has taken place and most likely modified the various, in our opinion mainly  $\beta_2$  mediated, responses studied.

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- 4 Larsson S, Svedmyr N. Cumulative dose-response curves for comparisons of oral bronchodilating drugs. A study of salbutamol and fenoterol. *Ann Allergy* 1977;39:362–6.

**AUTHORS' REPLY** Drs Larsson and Hjemdahl raise questions regarding interpretations and conclusions not addressed in our paper comparing the extrapulmonary effects of fenoterol, salbutamol, and isoprenaline.

With regard to hypokalaemia, mean (SEM) baseline values were similar on the four study days: placebo 4.0 (0.12) mmol/l, salbutamol 4.2 (0.12) mmol/l, fenoterol 4.0 (0.08) mmol/l, isoprenaline 4.1 (0.12) mmol/l. Studies were undertaken a week apart and treatments administered randomly according to a balanced Latin square design. This design minimises any systematic carry over effect and the time interval makes altered  $\beta$  receptor regulation unlikely, particularly as it followed a single administration. The rise in plasma potassium following placebo and, more importantly, the resting state is of interest. We have repeatedly observed it