LETTERS TO THE EDITOR

Local anaesthesia for fibreoptic bronchoscopy

I read with interest the report by Dr A R Webb and colleagues (June 1990;45:474-7), in which the transcranial injection of lignocaine was compared with "spray as you go." Drs B R O'Driscoll and P V Barber (December 1990;45:984) describe a modification of the latter technique in which lignocaine is introduced into the subglottic space as a bolus during inspiration. They indicate, however, that this technique requires an experienced bronchoscopist and that in difficult cases an assistant is required.

For the last 10 years I have been using a combination of "spray as you go" and direct injection of lignocaine into the trachea via a catheter passed through the channel of the bronchoscope. Lignocaine 2% gel is applied to the nasal mucosa and the structures at the back of the mouth are sprayed with local anaesthetic (lignocaine 10% spray, 10 mg/ dose). The tip of the bronchoscope is then positioned above the vocal cords and lignocaine 2% (one or two ml doses) is sprayed directly on to the cords. A catheter (PR-2B, supplied with Olympus bronchoscopes) is passed down the channel of the bronchoscope and advanced through the cords. Lignocaine 2% (one or two ml doses) is then injected directly into the trachea via the catheter. The effect of the intratracheal injection is usually to stimulate coughing. As in both transcranial injection and the technique of Drs O'Driscoll and Barber, the lignocaine is likely to be deposited on the inferior and medial surfaces of the tracheal cords, producing more effective anaesthesia for bronchoscopy than simple "spray as you go."

Although the methods have not yet been compared directly, I believe that this technique is as effective as transcranial injection in producing good conditions for bronchoscopy. It may take slightly more time but the bronchoscopist need not be particularly experienced, an extra assistant is not required, and the occasional complications of transcranial injection are avoided.

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Lung function 8-18 years after intermittent positive pressure ventilation for hyaline membrane disease

The work which has gone into the follow up study by Drs M J K de Kleine and others of lung function in children of preterm birth (December 1990;45:941-6) has been thorough and exhaustive. We would, however, question the basis on which the work was carried out. Follow up studies may be subject to tremendous bias, particularly in the selection of control subjects. For instance, the authors chose children with hyaline mem-

brane disease who had not been ventilated for comparison with children who had bronchopulmonary dysplasia, matching for gestational age and sex. It is therefore unlikely that they would be able to identify gestational age or sex as risk factors for subsequent chronic respiratory disease. One of the other control groups, the 25 premature children who had neither hyaline membrane disease nor other perinatal respiratory problems, seem to have been a specific group enrolled to test the effect of antenatal corticosteroids. No perinatal data were obtained for this reference group. Finally, the full term controls consisted merely of 39 pupils at a local school, including some children who had passed through hospital and for whom, as the authors themselves admit, adequate reference data allowing for the pubertal growth spurt are not available.

In our own more comprehensive study of a complete cohort of low birth weight children, together with a large unselected group of local schoolchildren, we came to different conclusions from those of Dr de Kleine and his colleagues. Our data clearly showed that, independently of perinatal disease and its management, birth weight and to a lesser extent gestational age were by far the greatest risk factor for chronic respiratory handicap at the age of 7. Male sex, maternal smoking, and the duration and degree of oxygen therapy in the newborn period were also significant risk factors. 1, 2, 3

Whereas it is quite clear that neonatal mechanical ventilation is associated with early lung injury, it would seem that later in childhood, after healing and repair, the residual effects may largely be due to the period of lost or abnormal lung growth resulting from prematurity. 4

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Ipecacuanha asthma: more lessons

I read with interest the report by Professor A Seaton (December 1990;45:974), who expresses concern about "a decline in interest among doctors about the primary causes of diseases as opposed to the mechanisms." He uses the example of ipecacuanha asthma and asks: "Why was it forgotten between 1850s and the 1980s?" I fully agree with the view that mechanisms alone have been given too much weight lately in asthma research. I think that the way asthma continues to be a rich source of important facts about the disease. Astute observers such as Henry Hyde Salter 1 should always be consulted, whether it is about the nature of the disease or its treatment. 2 A key to other authors, particularly on ipecacuanha asthma, can be found in the excellent Geschichte der Allergie by Schadewaldt. 3 It seems evident that there are more lessons to be learned from the long history of asthma. This disease had been described by 1662 and it was not forgotten after 1850.

During the eighteenth and nineteenth centuries ipecacuanha induced asthma was frequently reported as an occupational risk for people in the pharmaceutical and medical professions. 4 In such patients Murray (1776) 5 describes additional symptoms from the eyes and the nose occurring immediately after exposure to ipecacuanha dust. From Cullen (1780) 6 we learn that a pharmacist after working with this substance is sufficiently contaminated to provoke an attack of asthma in his wife.

There is a precedent for the 1884 publication that prompted Professor Seaton's report. In the Boston Medical and Surgical Journal of