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 transcricoid 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 2 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 2 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 transcricoid 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 vocal 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 transcricoid 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 transcricoid injection are avoided.

J MacMAHON
Department of Respiratory Medicine, Belfast City Hospital, Belfast BT9 7AB

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 membrane 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 preterm 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 puberty 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.[1-3]

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]

M SILVERMAN
K N CHAN
Department of Paediatrics and Neonatal Medicine, Royal Postgraduate Medical School, London W12 0NN

1 Chan KN, Noble-Jamieson CM, Elliman A, Bryan EM, Silverman M. Lung function in children of low birthweight. *Arch Dis Child* 1989;64:1284–93.
2 Chan KN, Elliman A, Bryan E, Silverman M. Respiratory symptoms in children of low birthweight. *Arch Dis Child* 1989;64:1294–304.
3 Chan KN, Elliman A, Bryan E, Silverman M. Clinical significance of airway responsiveness in children of low birthweight. *Pediatr Pulmonol* 1989;7:251–8.
4 Chan KN, Wong YC, Silverman M. Relationship between infant lung mechanics and childhood lung function in children of very low birthweight. *Pediatr Pulmonol* 1990;8:74–81.

Authors' reply

We thank Drs Chan and Silverman for addressing the question whether abnormalities in lung function, found in survivors of hyaline membrane disease, are due to prematurity itself or to the persistence of unresolved lung injury. Although their impressive data provide evidence for the former hypothesis, our data support the latter.

They emphasise the importance of dysanaptic growth, offering the hypothesis that very premature birth disrupts the normal process of lung growth.[1 2] Our study was designed to detect late sequelae of intensive pulmonary treatment. For that reason we deliberately selected as a main control group children who differed only in severity and treatment of hyaline membrane disease. We thought that the choice of a control group of premature children without hyaline membrane disease was less appropriate, as lack of surfactant is a physiological condition under 34 weeks of gestation. A group of prematurely born infants without hyaline membrane disease in the perinatal period may be confounded either with known factors such as intrauterine growth retardation, prolonged rupture of the membranes, and perinatal infections or with unknown factors.

The main abnormality we found was increased bronchial smooth muscle tone. Although Dr Chan and colleagues studied airway responsiveness to histamine very thoroughly,[3 4] they did not provide data on lung function after bronchodilatation. We await the results of the longitudinal measurements which they intend to perform in their low birth weight group[2] and suggest they also include lung function measurements after bronchodilatation.

M J K de KLEINE
H M JANSEN
Neonatology Centres, Sint Joseph Ziekenhuis, 5500 MB Veldhoven, The Netherlands

1 Green M, Turner JM. Variability of maximal expiratory flow-volume. *J Appl Physiol* 1974;37:67–74.
2 Chan KN, Noble-Jamieson CM, Elliman A, Bryan EM, Silverman M. Lung function in children of low birth weight. *Arch Dis Childh* 1989;64:1284–93.
3 Chan KN, Noble-Jamieson CM, Elliman A, Bryan EM, Aber VR, Silverman M. Airway responsiveness in low birthweight children and their mothers. *Arch Dis Child* 1988; 63:905–10.
4 Chan KN, Elliman A, Bryan E, Silverman M. Clinical significance of airway responsiveness in children of low birth weight. *Pediatr Pulmonol* 1989;7:251–8.

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 too that the medical history of 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 ipecacuanha 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.[3] In such patients Murray (1776)[4] describes additional symptoms from the eyes and the nose occurring immediately after exposure to ipecacuanha dust. From Cullen (1780)[5] 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