LETTERS TO THE EDITOR

Local anaesthesia for fiberoptic 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.

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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 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

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- 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 group² and suggest they also include lung function measurements after bronchodilatation.

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- 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 Child* 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 Salter¹ 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

1843 Turner reported on his "discovery" of ipecacuanha induced asthma. Turner, who also suffered from asthma, had to convince himself repeatedly that minute atmospheric amounts of ipecacuanha dust were capable of inducing a violent attack of asthma "because so new and singular was the fact . . ." The other side of the Atlantic may even today have a reputation for medical rediscoveries but, as Professor Seaton's communication makes clear, the rediscoveries may equally well take place within Europe. Importantly, Schadewaldt³ gives Turner credit for discovering that asthma can be provoked by oral intake of the allergen: "On two occasions I took a single laxative pill, without being aware that each of them contained about a grain of ipecac., yet in both instances, after an interval of eight or 10 hours, a severe paroxysm of asthma followed their exhibition."

Towards the end of the nineteenth century, there was a great interest in the hypothesis that asthma could be induced by a neurogenic nasal reflex mechanism.⁴ Lenhartz (1899) regarded ipecacuanha asthma as supporting the idea of a neurogenic reflex induced asthma starting in olfactory nerves.²

In his book on asthma Salter¹ characterised ipecacuanha asthma together with the asthma that was specifically induced by animal emanations (particularly from the "fur of animals"), and with that "curious affection known as hay fever" (which Salter, before Blackley, blamed on "flowering grass" instead of hay). Among Salter's own patients ipecacuanha asthma affected three medical students. Salter remarked on the singular cause of this asthma: "In none of them does asthma occur under any other circumstances —no other irritant will produce it." He seemed to exclude a psychogenic component: "They were always obliged to leave the room when it [ipecac.] was employed." Finally, Salter concluded from the fact that they were all medical students: "If more people were exposed to the peculiar exciting cause the number of instances of this kind of asthma would probably be much greater."

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1 Salter HH. *On asthma: its pathology and treatment*. 2nd ed. London: Churchill, 1868.

2 Persson CGA. On the medical history of xanth-

ines and other remedies for asthma. A tribute to H H Salter. *Thorax* 1985;40:881-8.

3 Schadewaldt H. *Geschichte der Allergie*. München: Dustri, 1983.

4 Murray JA. *Apparatus medicaminum tam simplicium quam praeparatorum et compositorum*. Göttingen, 1776.

5 Cullen W. *First lines of the practice of physic*. Edinburgh, 1776.

6 Turner U. Asthma caused by ipecacuanha. *Boston Med Surg J* 1843;29 (see also *Ann Allergy* 1960;18:110).

7 Persson CGA. On the medical history of asthma and rhinitis. In: Mygind N, Pipkorn U, Dahl R, eds. *Rhinitis and asthma. Similarities and differences*. Copenhagen: Munksgaard, 1990.

8 Lenhartz H. Asthma nervosum, s. bronchiale. In: W Ebstein, ed. *Handbuch der praktischen Medicin*. Bd 1. Stuttgart: 1899:392.

BOOK NOTICE

Imaging of Diseases of the Chest. Edited by P Armstrong, A G Wilson, P Dee. (Pp 914; £127.50.) London: Wolfe, 1990. ISBN 0-8151-0350-6.

This new book has all the potential to be a success. It fills an important place between the established but weighty volumes of Fraser and Paré and the numerous smaller books on chest radiology. The book covers the subject of chest imaging in a single volume and in a remarkably comprehensive way. It is written from both sides of the Atlantic, which is a winning formula. Peter Armstrong is professor of radiology at St Bartholomew's Hospital but spent many years in the University of Virginia. His coeditors are Alan Wilson from London and Paul Dee from Virginia; the three editors have written most of the chapters but with important contributions from other American authors. The book very successfully combines current practice and thinking from both sides. The illustrations are generally of very good quality and the text is a pleasure to read. There is a good introductory section on basic principles and the normal chest, and this is particularly well written for the trainee. There then follow systematic sections on diseases of the lung,

pleura, and mediastinum, and finally useful chapters on chest trauma and on interventional procedures. The book will be enjoyed by general radiologists and physicians but there is also much to satisfy the specialist. There is a good blending of radiology, medicine, and pathology, which adds greatly to the book's value. I particularly like the tables for classification of disease and differential diagnosis, and the large number of references, mainly to British and American publications. This is a very good book and I expect it to establish itself as a standard work. I hope that it will be bought by all medical and radiological libraries, but individuals will also find it indispensable and well worth the purchase price.—BCO

NOTICES

British Thoracic Society winter meeting

The British Thoracic Society's winter meeting will be held at Kensington Town Hall, London, on 4-6 December 1991.

This meeting will be open to non-members. Abstract forms can be obtained from the administrative director, 1 St Andrews Place, London NW1 4LB, from 2 August 1991 (tel 071 486 7766; fax 071 224 2635). The closing date for acceptance is 20 September 1991 (first post).

International Symposium on Cardiopulmonary Urgencies and Emergencies

The 7th International Symposium on Cardiopulmonary Urgencies and Emergencies will take place on 19-22 November 1991 in Rotterdam. Details may be obtained from Dr O Prakash, Thorax Centre, Dijkzigt Hospital, Dr Molewaterplein 50, 3015 GD Rotterdam, The Netherlands (tel 31-10-463-5230; fax 31-10-463-5240).