LETTERS TO THE EDITOR

Chickenpox pneumonia: an association with pregnancy

We read with interest the article by Dr T F Esmonde and colleagues (October 1989;44: 812–5) on the relation between chickenpox pneumonia and pregnancy. We agree with their overall conclusions, though our own observations and those of others[1] suggest that pregnancy may not have been the only risk factor in their patients. Dr Esmonde and colleagues do not record details of their patients' smoking histories, a crucial risk factor.[2]

Over the last two years we have cared for 32 adult inpatients with chickenpox (mean age 24·7 (SD 6·3) years), of whom seven had symptoms and radiological evidence of pneumonia. Of the patients with pneumonia, two were pregnant, and both were smokers. Smoking was the only risk factor in four patients, and one patient was diabetic. We also treated six pregnant or puerperal women without pneumonia (three in the last trimester), of whom only one smoked. Although only four of the 14 smokers with no other risk factor had pneumonia, lung function abnormalities are often found in smokers with chickenpox, despite a normal chest radiograph.[2]

Immunological changes of pregnancy may be responsible for some of the increased risks of pneumonia. Attending to infected children may carry a risk of severe disease in some patients, and pregnancy may be a marker for the presence of infectious children at home. We found that three of seven patients with pneumonia had apparently contracted chickenpox from a child they were caring for, compared with only one of 25 without pneumonia ($p < 0·05$, Fisher's exact test). Measles mortality is associated with overcrowding, close contact, and (by implication) a large infectious dose.[3] A case-control study of the association between exposure to the source case of chickenpox and the severity of the resultant illness would be most interesting.

Finally, although acyclovir is not of proved benefit in chickenpox pneumonia, we would advise its use in the patient with evidence of early or established pneumonitis due to varicella zoster, at a dose of 10–15 mg/kg three times a day.

STEPHEN J ROGERSON
F J NYE
Infectious Disease Unit, Fazakerley Hospital, Liverpool
NICHOLAS J BEECHING
Liverpool School of Tropical Medicine, Liverpool

1 Davidson RN, Lynn W, Savage P, Wansbrough-Jones MH. Chickenpox pneumonia: experience with antiviral treatment. *Thorax* 1988;43:627–30.
2 Ellis ME, Neal KR, Webb AK. Is smoking a risk factor for pneumonia in adults with chickenpox? *Br Med J* 1987;294:1002.
3 Aaby P. Malnutrition and overcrowding/intensive exposure in severe measles infection: review of community studies. *Rev Inf Dis* 1988;10:478–91.

AUTHORS' REPLY We are grateful for these comments. Two of our three patients smoked (10 and 20 cigarettes daily) and one did not. It is an interesting suggestion that pregnancy might be a marker for the presence of infectious children in the home but the form of our study did not allow us to address this question.

We concluded that the role of acyclovir was not proved in the treatment of chickenpox pneumonia and that mortality had not altered since the introduction of this drug. The recommendation of Dr Rogerson and his colleagues to use acyclovir in "early and established" cases (presumably this means all) of chickenpox pneumonia is not based on any factual evidence.

J ESMONDE
G ANDERSON
G HERDMAN
Newport Chest Clinic, Newport, Gwent NP9 4GA

Sedation for fibreoptic bronchoscopy

Dr MP Shelley and colleagues (October 1989;44:769–75) concentrated on the various sedatives used during fibreoptic bronchoscopy but only briefly mentioned the local anaesthetic technique; yet this, perhaps more than the sedative, determines the acceptability of the procedure to the patient.

In the 1986 survey of bronchoscopic practice in the UK, lignocaine was the most commonly used local anaesthetic and only 7% of respondents used cocaine. The anaesthetic was administered via the bronchoscope in most and 15% of respondents used the transcricoid route. In a study primarily concerned with the effect of local anaesthetic agents on lavage yield and macrophage function,[1] we were impressed by the local anaesthesia produced by the transcricoid route and also with the superiority of cocaine (4–6 ml 5% cocaine: 200–300 mg) as the local anaesthetic. This has now become our standard anaesthetic technique in this hospital. The preference for the transcricoid route, for both operator and patient, has recently been confirmed.[2,3] We are unaware of a formal comparison of cocaine and lignocaine as anaesthetic agents at bronchoscopy. We recommend that other centres performing fibreoptic bronchoscopy consider changing to the transcricoid instillation of cocaine.

A C DAVIDSON
R LEACH
N T BATEMAN
Medical Unit, United Medical Dental School, St Thomas's Campus, London SE1 7EH

1 Davidson AC, Barbosa I, Kooner JS, *et al.* The influence of topical anaesthetic agent at broncho-alveolar lavage upon cellular yield and identification and upon macrophage function. *Br J Dis Chest* 1986;80:385–90.
2 Kinnear WJM, Reynolds L, Gaskin D, Macfarlane JT. Comparison of transcricoid and bronchoscopic routes for administration of local anaesthesia before fibreoptic bronchoscopy [abstract]. *Thorax* 1988;43:805P.
3 Hay J, Clague J, Nasar M, Earis JE. Local anaesthesia for fibreoptic bronchoscopy [abstract]. *Thorax* 1989;44:890P.

Postpneumonectomy pulmonary oedema

Our experience with postpneumonectomy pulmonary oedema supports the work of Dr L Verheijen-Breemhaar and colleagues (April 1988;43:323–6). We found no significant differences between three patients with postpneumonectomy pulmonary oedema following right pneumonectomy and 10 patients with uncomplicated postoperative courses in age, preoperative pulmonary function, blood gases, intraoperative blood loss, or operative time. The 24 hour net fluid balance, however, was 86·4 (SD 10) ml/kg for patients with postpneumonectomy pulmonary oedema and 47 (19) ml/kg for those without ($p < 0·008$). We agree that most existing data implicate excessive fluid administration in the pathogenesis of this condition, though application of an equivalent haemodynamic stress by left atrial Foley balloon inflation produced no increased susceptibility to postpneumonectomy pulmonary oedema, suggesting that the decreased oncotic pressures resulting from excessive fluid administration may be important.[1]

These data suggest that postpneumonectomy pulmonary oedema might be prevented if safe guidelines for fluid administration were established. Unfortunately, there appears to be no consensus on this matter. One manual advises that "for the first 24 hours it is wise to limit the baseline fluids to 40 ml/kg given as 5% dextrose in water" after pulmonary operations.[2] A leading anaesthesia text suggests 8 ml/kg/h intraoperatively for thoracotomy.[3] Neither provides specific guidelines for treatment after a pneumonectomy.

Our data suggest important differences in postpneumonectomy fluid management between different institutions. We were struck that one Dutch patient developed postpneumonectomy pulmonary oedema with a net positive balance of only 1000 ml (roughly 15 ml/kg/24 h), yet two of our patients developed no difficulty despite a net balance exceeding 70 ml/kg/24 h. Perhaps individual variations in preoperative hydration, cardiac reserve, residual pulmonary lymphatic capacity, and pulmonary endothelial permeability affect the fluid volume that may be safely given.

We caution that the safe postpneumonectomy fluid threshold is variable and may be less than 15 ml/kg/24 h in some patients. It seems likely that postpneumonectomy pulmonary oedema cannot be completely eliminated, but careful management of fluid intake may lessen its incidence and severity.

M MARGOLIS
Pulmonary Disease Section, Philadelphia Veterans' Administration Medical Center, Philadelphia, Pennsylvania 19104, USA

1 Lee E, Little AG, Hsu WH, *et al.* Effect of pneumonectomy on extra-vascular lung water in dogs. *J Surg Res* 1985;38:568–73.
2 Committee on Pre and Postoperative Care, American College of Surgeons. *Manual of preoperative and postoperative care.* 3rd ed. Philadelphia: Saunders, 1983:376–7.
3 Miller RD. *Anesthesia.* 2nd ed. New York: Churchill Livingstone, 1986:1314–5.

BOOK NOTICE

Crofton and Douglas' Respiratory Medicine. 4th edition. Edited by A Seaton, D Seaton, AG Leitch. (Pp 1215; £79·50.) Oxford: Blackwells, 1989. ISBN 0632 019735.

This well known and excellent textbook has been extensively rewritten by the three new editors. It is in general an extremely well presented and readable text with illustrations, including the many radiographs, of a very

high standard. The book is thoroughly referenced at the end of each chapter. The text is aimed at those who have committed themselves to specialisation in respiratory medicine and also at those embarking on their specialist career. All the important subjects in respiratory medicine are covered, though in many cases those who might dip into this textbook to obtain information on a rare disease or presentation might be disappointed that, although the subject is mentioned, it is only a rather brief annotation. Usually the reference list at the end of the chapter makes up for this. The fact that the three editors have written the entire textbook (with the exception of the chapter on AIDS) adds enormously to the continuity of style and the lack of repetition and makes for easy though intense reading. Inevitably one has one's quibbles. Some important subjects have less space devoted to them than they might deserve—for example, respiratory failure 13 pages, cystic fibrosis 15 pages, sarcoidosis 30 pages.

One of the growing areas of respiratory medicine, sleep abnormalities, is mentioned only as a short review in the chapter on pulmonary physiology and discussion of its treatment is particularly scanty. Chronic bronchitis also has little on its treatment for such a common disease and, although oxygen therapy is referred to in a later chapter (chapter 20), it is in fact barely mentioned. Long term oxygen therapy, a particular problem we are facing today, receives only two paragraphs. The adult respiratory distress syndrome is also discussed too briefly to allow the reader real insight into the plethora of problems it is associated with. Nevertheless, nearly all respiratory problems are presented most adequately and the chapter on drugs in respiratory disease is first class, providing a magnificent broad brush of treatment across the specialty. There is little doubt that those wishing to learn the basics of respiratory medicine, with a view to obtaining a first class background from which they could progress to more exhaustive reading, would get much out of this book, which can be thoroughly recommended to every physician committed to and already in respiratory medicine. My only other comments are that the book finds space to quote one of Dr Gerald Anderson's poorer jokes and the statutory reversed chest radiograph (to make sure the reviewer has done his stuff) is on page 983.—SS

CORRECTION

Decline of the lung function related to the type of tobacco smoked and inhalation

We regret that, owing to a printing error, in tables 2 and 3 of the paper by P Lange *et al* (January 1990;45:24) the rows of figures against "Did not inhale" and "Inhaled" were transposed. The corrected tables are printed below.

Table 2 Average daily tobacco consumption and decline in FEV_1 (ΔFEV_1) according to the type of tobacco smoked and whether smoke was inhaled

	Women			*Men*		
Smoking group	*n*	*Tobacco consumption (g, range)*	*ΔFEV_1 (ml/y, SEM)*	*n*	*Tobacco consumption (g, range)*	*ΔFEV_1 (ml/y, SEM)*
NON-SMOKERS	2417	0	25 (2)	1336	0	30 (3)
SMOKERS						
Plain cigarettes						
Did not inhale	112	11 (1–40)	27 (7)	55	16 (1–55)	53 (13)
Inhaled	591	14 (1–43)	34 (3)	734	18 (1–79)	46 (3)
Filter cigarettes						
Did not inhale	351	10 (1–40)	28 (5)	47	14 (1–45)	30 (17)
Inhaled	1111	14 (1–48)	32 (3)	427	17 (1–50)	42 (5)
Cigar or cheroots						
Did not inhale	315	29 (3–110)	41 (4)	166	31 (3–110)	52 (8)
Inhaled	89	43 (3–110)	44 (8)	141	45 (3–110)	66 (6)
Pipe						
Did not inhale				107	15 (5–50)	32 (11)
Inhaled				126	16 (5–39)	56 (9)

Table 3 Regression analysis of decline in FEV_1 (ml/y) on age, height, alcohol consumption, and the different types of tobacco smoking for smokers and non-smokers of both sexes (decline in non-smokers used as baseline)

	Women		*Men*	
Independent variable	*Regression coefficient (SEM)*	*p*	*Regression coefficient (SEM)*	*p*
Intercept	−96·7		−141·0	
Age (y)	1·2 (0·1)	<0·001	0·8 (0·2)	<0·001
Height (cm)	0·4 (0·2)	0·05	0·7 (0·3)	<0·05
Alcohol drinks/day	0·4 (1·7)	NS	2·1 (0·9)	<0·05
Smoking groups:				
NON-SMOKERS	0		0	
SMOKERS				
Plain cigarettes				
Did not inhale	1·0 (7·8)	NS	18·3 (15·1)	NS
Inhaled	13·3 (3·8)	<0·001	14·3 (5·6)	<0·005
Filter cigarettes				
Did not inhale	7·1 (4·7)	NS	−3·0 (15·6)	NS
Inhaled	11·1 (3·1)	<0·001	14·9 (6·1)	<0·05
Cigars or cheroots				
Did not inhale	8·5 (4·9)	0·09	16·8 (9·0)	0·06
Inhaled	20·6 (9·0)	<0·05	33·6 (9·6)	<0·001
Pipe				
Did not inhale			−2·1 (10·7)	NS
Inhaled			27·9 (10·1)	<0·01

NS indicates p > 0·1.