that changes in osmolality during nebulisation could have led to bronchoconstriction is not borne out by the failure of nebulised saline control to cause any reduction in $\dot{V}$maxFRC.

It is possible that the low pH of salbutamol solution could have induced bronchoconstriction. The pH of our normal saline ampoles is 6·0 and of salbutamol nebules is 4·0 (not 7·6 and 6·25, as found by Dr O'Callaghan). However, neither histamine solution in low concentrations (below that which induced bronchoconstriction—pH around 5) nor ipratropium bromide nebuliser solution (pH 4·0) have caused a significant decline in $\dot{V}$maxFRC in our studies.[1 2] The hypothesis that the pH of the nebuliser solution is a critical factor therefore remains speculative. More research in infants is clearly required.

In answer to the comments of Dr Beasley and colleagues, the salbutamol respirator solution used in the study performed by O'Callaghan *et al* and the salbutamol "nebules" in our study contain benzalkonium chloride. Thus the reduction in $\dot{V}$maxFRC seen 15 minutes after salbutamol nebulisation could be preservative induced.

We have performed a further study[2] of change in airway function in 17 wheezy infants after nebulised ipratropium bromide, which also contains benzalkonium chloride. At 15–20 minutes after nebulisation of 1 ml of ipratropium bromide in 1·5 ml 0·9% sodium chloride there was a significant reduction in specific airway resistance with no significant change in $\dot{V}$maxFRC. Thus benzalkonium chloride, at least when present in nebulised ipratropium bromide, does not appear to induce bronchoconstriction in these infants.

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1 Prendiville A, Green S, Silverman M. Ipratropium bromide and airways function in wheezy infants. *Thorax* 1987;**42**:92–9.
2 Prendiville A, Green S, Silverman M. Ipratropium bromide and airways function in wheezy infants. *Arch Dis Child* 1987;**62**:397–400.

Book notices

Recent Advances in Respiratory Medicine. No. 4. Eds DC Flenley, T Petty. (Pp 285; £37.50, hardback) Edinburgh: Churchill Livingstone, 1986. ISBN 0-443-034117.

The difficulty about reviewing an anthology is that, almost always, the contributions are of varying standard, varying interest, and varying degrees of suitability for inclusion. There is no such problem here; all of the 16 chapters are well prepared, stimulating, and relevant to the practising clinician. This is the fourth volume of *Recent Advances in Respiratory Medicine* to appear after a regular interval of three years and many of the "plum" subjects for review have already been covered in earlier numbers. It is all the more remarkable that Professor Flenley and Dr Petty have managed to obtain such an interesting collection of informative reviews. Topics and authors are spread widely. The epidemiology of asthma is reviewed from New Zealand, airways responsiveness from Canada, and cell receptors and airway function in asthma from the United Kingdom. Pulmonary manifestations of AIDS are reviewed from the United States (San Francisco), the use of cephalosporins in lung disease from Italy, and the control and surveillance of tuberculosis from The Netherlands. It will be apparent already that this is a much more international collection than any of the previous volumes. Some of the contributions are condensed overall reviews of the subject rather than commentaries limited to recent advances and this is the case with the chapters on sarcoidosis, cryptogenic fibrosing alveolitis, pulmonary thromboembolism, and pleural effusion. The editors' particular interests are reflected in the next few chapters on the early pathogenesis and identification of chronic obstructive airways disease, various topics in chronic bronchitis and emphysema (diagnosis of emphysema, protease-antiprotease theory, pulmonary vasodilators, improving airflow limitation, inspiratory muscle training), domiciliary and ambulatory oxygen treatment in chronic respiratory insufficiency, and breathing during sleep in adults. There are two contributions on lung cancer—one on early identification and one on staging. Overall this is an excellent collection of reviews, which is well up to the standard of the previous volumes and which will be much thumbed over the next few years by clinicians with an interest in respiratory medicine.—RALB

Acute Lung Injury. Pathogenesis of adult respiratory distress syndrome. H Kazemi, AL Hyman, PJ Kadowitz. (Pp 270; £27.50, hardback.) Massachusetts: PSG Publishing Company, 1986. ISBN 0-88416-536-6.

This book summarises a symposium on the pathogenesis of the adult respiratory distress syndrome (ARDS) held in 1984 under the auspices of the Cardiopulmonary Council of the American Heart Association. In 19 chapters it reviews areas of lung injury research which shed light on the pathogenesis of ARDS. The authors, 43 in total, are a cross section of active researchers in ARDS in the United States. They give detailed and relatively up to date reviews of their own research and that of allied workers. There are good reviews of the pathology and pulmonary haemodynamics of human ARDS from the Boston group, but most of the chapters concentrate on experimental work in animal models or in vitro systems and have as yet little direct clinical application. The organisers of the symposium are to be congratulated for not allowing the role of the neutrophil to dominate, so that in this volume the neutrophil is viewed in the context of other mechanisms. There are two extremely good chapters on alveolar epithelial function, including active transport mechanisms and permeability and its assessment, which are balanced by chapters discussing the role of the pulmonary endothelium in vasodilatation and the generation of cyclic GMP by vascular smooth muscle, and others on the regulation of fluid balance in the lungs. Animal models of injury,