potential for confounding by severity when an unmatched control group is used. A similar problem occurred in the Saskatchewan study,[7] which also used an unmatched control group. When this problem is corrected, however, either by using an appropriate control group (group A) or by adjusting for markers of asthma severity (table, top of p574), then the association of asthma drugs in general with deaths from asthma tends to disappear, whereas the findings for fenoterol remain firm (a similar pattern occurred in the Saskatchewan data[7]). The table shows that control group A provides an adequate match for asthma severity, whereas some confounding exists in the unadjusted results for control group B. We drew this conclusion in the published paper,[5] and Dr Lanes and his coworkers have simply repeated our analysis but misrepresented our conclusions.

When the hazards of fenoterol are being considered it is important that all of the evidence should be considered. There is now a wealth of epidemiological, experimental, and clinical evidence that fenoterol is more hazardous than other commonly used β agonists.[2] The second New Zealand mortality epidemic started when fenoterol was introduced in 1976, and continued until our first study was published in 1989; the death rate then fell by one half, and is now similar to that in other countries. It is important to search for alternative explanations, but the evidence increasingly indicates that confounding by severity is not a plausible explanation, and that the association between fenoterol and deaths from asthma is likely to be causal.

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Pleural abrasion: a new method of pleurodesis?

Pleural abrasion, as a means of pleurodesing the lung, is not a new technique, as implied by the paper of Mr UU Nkere and others (August 1991;**46**:596–8). We and most thoracic surgeons in Australia have been performing transaxillary thoracotomies, apical bullae stapling and abrasive pleurodesis for at least 20 years. At the Prince Charles Hospital—a cardiothoracic hospital serving Queensland—in the period January 1985–December 1990, 320 patients were operated on in our thoracic surgical service for spontaneous pneumothorax. The mean age was 28 years and M:F ratio was 1·4:1.

Surgery was performed via the following surgical approaches: transaxillary thoracotomy (TAT) 244 patients, bilateral TAT 12 patients, lateral thoracotomy 52 patients, anterior thoracotomy 6 patients, posterolateral thoracotomy 6 patients. Pleurodesis was achieved thus: pleural abrasion 185 patients, talc with or without abrasion 42 patients, pleurectomy 84 patients, talc with or without pleurectomy 4 patients, other or unknown 5 patients. The mean postoperative hospital stay was four days. There were recurrences requiring surgery in 20 patients and recurrences not requiring surgery in three patients.

I think you must agree that from our experience pleural abrasion is *not* a new method. We agree, however, with the authors that it is a highly suitable technique with good results. If combined with a transaxillary approach — often an incision no more than 2 inches (5 cm) wide — it is a cosmetically acceptable form of management for spontaneous pneumothorax, and we will continue to use this procedure.

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AUTHORS' REPLY We are grateful to Drs Cole and Matar for sharing their extensive experience of surgery for pneumothorax with us. It was with some misgivings that we accepted the editorial decision to change the original title of our paper from "A safe and effective method of pleurodesis" to "New method . . . ." The only aspect of the technique which, as far as we are aware, has not been previously described is the use of a domestic pan scourer to achieve pleural abrasion and even this is not our invention, as it was being used by Mr Angus MacArthur at King's College Hospital 20 years ago. Despite the fact, however, that pleural abrasion has been in widespread use in North America and, as we now know, in Australia for many years not many surgeons using the technique routinely have published their results, and in the United Kingdom there remains the belief outside a small circle of thoracic surgeons and enlightened chest physicians that surgery for pneumothorax calls for a full pleurectomy through a large and painful incision. Indeed, it was the inaccurate and sometimes alarming perception that many of our patients had appeared to receive that prompted us to put our experience together, and in that the subject seems now to have received a wider medical airing than before[1] our principal objective has, in part, been fulfilled.

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Shunting through a patent foramen ovale

The phenomenon of shunting through a patent foramen ovale, as recently reported by Dr L Berry and others (January 1992;**47**:60–1), has interested me for some time.[1]

Those authors emphasised the important point that the shunt flow was detected by transoesophageal echocardiography but *not* by transthoracic echocardiography — this is not recognised by all echocardiologists. A characteristic feature of this syndrome is platypnoea and orthodeoxia — that is, more pronounced breathlessness and hypoxaemia in the upright than in the supine position. Thus the shunt measurements should probably be made with the patient in the upright position if possible.

The incidence of probe patient foramen ovale is about 25%,[2] but so long as left atrial pressure exceeds right atrial pressure the valve leaflet acts as a closing flap. In the fetus the inferior vena cava empties anatomically and functionally through the foramen ovale into the left heart. In the adult a probe patent foramen ovale is most easily penetrated with a cardiac catheter passed from a femoral vein, *not* from the brachial vein. Unfortunately Dr Berry and her colleagues did not state which approach they used in their catheterisation.

For two reasons I doubt their explanation that the mechanism for the shunt is caused by mediastinal distortion caused by right pneumonectomy. Firstly, as mentioned above, the normal anatomy favours blood flow from the inferior vena cava to the left heart when the flap is pressed open. Secondly, this type of shunt occurs also after left pneumonectomy[3] and after severe respiratory failure in chronic obstructive lung disease.[4] A more likely mechanism of the different shunting in different positions is the changing relation between the right and the left atrial pressures. This, in turn, depends on the function curves of the right and left ventricles[5]: apparently they cross, so that at low "venous return" right atrial pressure exceeds left atrial pressure, and at higher "venous return" left atrial pressure exceeds, or approaches, right atrial pressure.

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Persistent alveolar increased permeability to ^{99m}Tc DTPA in patients with advanced HIV infection

In their paper regarding the diagnostic value of lung clearance of ^{99m}Tc DTPA in *Pneumocystis carinii* pneumonia Dr D S Robinson and his colleagues (October 1991;**46**:722–6) emphasised the specificity of the shape of the clearance curve by noting that none of their patients who did not have pneumocystis pneumonia had a biphasic curve in both the upper and the lower zones of the lung.

We have observed three HIV infected homosexual men who did not have pneumocystis pneumonia in whom ^{99m}Tc DTPA transfer time (mean (SE) T_{50}) ranged from 3·1 to 4·6 (mean 4·3) minutes and was biphasic in the upper, mid and lower zones over a follow up period of four, 18, and 31 weeks. This compares with a mean ^{99m}Tc DTPA transfer time in five HIV patients with pneumocystis pneumonia of 3·1 (1·4) (range 1·8–9·6) minutes and is significantly lower than transfer times in HIV positive patients with various non-pneumocystis pneumonia chest condi-

tions (60·3 (10·4, range 13·6–191) min; n = 19; p < 0·001).

The CD4 counts on the patients before the first ^{99m}Tc DTPA transfer were 120, 130, and 170 respectively. All three were smokers, as were nine of 19 with various non-pneumocystis pneumonia chest conditions, and all three took nebulised pentamidine, 300 mg monthly, as primary prophylaxis for pneumocystis pneumonia, as did eight of 19 patients without pneumocystis pneumonia. Bronchopulmonary lavage (all three) and transbronchial biopsy (two patients) had negative results. Open lung biopsy in the first two patients did not show any opportunist infection. Both patients died—10 and 12 months after the initial ^{99m}Tc DTPA transfer. Postmortem examination in the first patient showed cytomegalovirus and toxoplasma brain disease. The third is symptom free 10 months after the first test.

It is concluded that a rapid biphasic ^{99m}Tc DTPA transfer may be seen in advanced HIV infection in the absence of pneumocystis pneumonia.

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BOOK NOTICES

Asthma 3rd ed. T J H Clark, S Godfrey, T H Lee. (Pp 622; £65.) London: Chapman and Hall, 1991. ISBN 0 412 35900 6.

This is the third edition of this text and many readers of *Thorax* will have purchased or have access to the first two editions. Readers of this review therefore need to know whether enough has been changed to justify a further purchase. The 1977 edition contained 409 pages, and by the time of the second edition in 1983 the subject merited an increase in length to 519 pages. This new edition is considerably longer and the original two editors have been joined by Tak Lee, professor of asthma and allergy at Guy's Hospital. The text is essentially new and this is really a new book rather than an update of the previous edition. In only four or five chapters is the author the same as in a previous edition. The chapter on physiology remains as powerful and authoritative as previously but it is now followed by a series of new and very well referenced reviews of airway responsiveness, neural mechanisms, mediators, and inflammation. The latter two are particularly strong and provide a very clear summary of current understanding in a didactic but fair manner. The diagrams and electronmicrographs are particularly clear and well presented in the section on inflammation. The previous edition's chapter on pathology has now been replaced by a short but very readable section on both pathology and cytology, and this contains a useful description of the bronchial circulation. One of the most useful chapters in the first two editions was that by Ian Gregg on epidemiology and it bravely tackled the problem of international comparisons at some length. This has been replaced in this edition by a different but no less useful chapter, which looks carefully at both genetic and environmental influences on the prevalence of asthma. The sections on smoking, pollution, and diet are particularly good, and well referenced up to 1990. An excellent, newly written summary of occupational asthma is rather awkwardly placed between the chapter on epidemiology and five very good chapters on pharmacology. That on β agonists was written recently enough to encompass most of the current controversies (but not necessarily the answers), and the chapters on steroids and other prophylactic agents are clear and provide a good summary of the current position. The chapter on methylxanthines has been rewritten by one of the previous authors, but the subject begins to look rather historical and there are few references beyond the mid 1980s. The last 100 pages are on the more obviously clinical aspects of asthma—that on childhood asthma has been updated rather than rewritten but the summary on adult asthma is completely new. This is well written and referenced but let down by rather unimaginatively produced algorithms. This chapter includes a useful section on the interface and relationships between the general practitioner and the hospital doctor, but in any future edition the editors might wish to consider pulling together a separate chapter that looks at the specific question of delivery of care. I suspect that this book is used most by clinicians who require a source of information on basic mechanisms, epidemiology, and pharmacology rather than being purchased for its clinical content. As such it more than fulfils its role and it is well produced and extremely well referenced. It is essentially a new book rather than a new edition and it can be strongly recommended to all who have any responsibility for those with this common condition.—MRP

History of tuberculosis in Australia, New Zealand and Papua New Guinea. Edited by A J Proust. (Pp273; A$37.50.) Curtin, Australia: Brolga Press, 1991. ISBN 1 875495 02 9.

This book is a delightful kaleidoscope of anecdote, story, and experience. It is written mainly by those engaged in the study and treatment of tuberculosis from the war years until relatively recently in Australia, New Zealand, and the Melanesian Islands. With over 40 contributors, the book has a wealth of variety and expertise, including "personal views" of lay patients. It is divided into 12 chapters, each comprising several separate essays by different authors. Though topics seem to be arranged fairly randomly, moving from tuberculosis in Australia, then to New Zealand, on to Papua New Guinea, and back to Australia again, this in no way detracts from its ability to maintain interest right to the end. It is a book that both lay people and medical professionals will find stimulating and informative. For the epidemiologist, there is some vital information that may not be easily accessible through normal literature searches. In particular, I found the chapter on tuberculosis in Papua New Guinea of interest. Tuberculosis did not affect the population in the central highlands of New Guinea until relatively recently, when epidemiological methods and data processing had reached a reasonably sophisticated state, so that a thorough scientific appraisal of the effect of tuberculosis on a totally non-immune population was made possible. Perhaps because it is written by older and wiser heads, much experienced in tuberculosis, the writing becomes at times almost prophetic. "Recent economic events in X have widened the gap between rich and poor. The increase in poverty and unemployment is likely to result in an upsurge of tuberculosis over the next decade. Furthermore, the advent of AIDS will be associated with an increase in tuberculosis, especially in racial groups with a high incidence of previous infection." Though this was actually written of New Zealand, does it necessarily matter which country in the world X refers to? Again, "no chest surgeon or physician can now hope to obtain the tuberculosis experience of our pioneers. We need to ensure that they have at least read what these remarkable Doctors achieved and to learn the principles they derived from their experience." What better self advertisement could one have for such a delightful read?—PDOD

NOTICES

British Society for Allergy and Clinical Immunology

The annual conference of the British Society for Allergy and Clinical Immunology will be held in Southampton on 7–9 September 1992. The main subjects will be allergens: biology and control; current advances in rheumatoid arthritis; and β agonists and steroids. Details from Conference Associates and Services Ltd, BSACI 1992, Congress House, 55 New Cavendish Street, London W1M 7RE.

German Society for Pneumonology

The 35th scientific congress of the Deutsche Gesellschaft für Pneumologie will be held in Wiesbaden on 23–26 September 1992. The main subjects will be inflammation; operative measures in respiratory diseases; respiratory diseases in the immunocomprised; and mycobacterioses. Details from Professor Dr J Meier-Sydow, Theodor-Stern-Kai 7, D-6000 Frankfurt (Main) 70, Germany (fax (069) 6301 7391).

Continuing medical education and training in Europe

An international conference entitled "Continuing medical education and training in Europe: the future" will be held in London at the Royal College of Physicians on 1 and 2 October 1992. Details from Dr M W N Nicholls, Conference Office, c/o Fellowship of Postgraduate Medicine, 6 St Andrew's Place, London NW1 4LB (tel 071 935 5556, fax 071 224 3219).

Clinical applications of pulmonary function testing

A two day course will be held on 2–3 November 1992 at Hammersmith Hospital, with lectures, demonstrations, and case discussions on physiological background, methods, and application of the common and not so common pulmonary function tests, aimed at doctors and technicians who work in pulmonary function laboratories or who engage in physiological research. The organisers are Dr J M B Hughes and Professor N B Pride. The course fee is £130. There will be some bursaries of £130 for pulmonary function technicians and non-medical staff who apply with a letter of support from their consultant. Application forms and further details from the Wolfson Conference Centre, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0NN (tel 081 740 3117/3245, fax 081 740 4950).