LETTERS TO THE EDITOR

PEF versus FEV_1

The assertion by Dr Thiadens and colleagues[1] that identification of airflow limitation and estimation of its reversibility by a bronchodilator is less reliable when measured by peak expiratory flow (PEF) than by forced expiratory volume in one second (FEV_1) cannot be allowed to go unchallenged. They measured both values with a Microlab 3300 turbine spirometer, disregarding the fact that, in primary care, PEF is almost always measured by peak flow meters of variable orifice type which employ an entirely different principle and give considerably higher values. Jones and Mullee,[2] who compared a similar Microlab turbine spirometer with a mini-Wright meter, found that values of PEF measured by the latter were, on average, 87 l/min higher. Hence, the values reported by Thiadens *et al* would have been much higher if they had been measured with a peak flow meter.

To compare the reliability of PEF and FEV_1 for estimating magnitude of airflow limitation, Thiadens *et al* expressed observed values of each as percentage predicted, using the reference values for each sex recommended by the European Respiratory Society (ERS).[3] Those for predicting PEF were derived from regression equations which describe a linear fall with age and give predicted values much lower than curvilinear regressions such as those of Nunn and Gregg,[4] which an ERS Working Party on PEF[5] subsequently judged to be the most satisfactory reference values for prediction. The difference in l/min between predicted values derived from the latter and those from the ERS regressions was roughly equal in each sex (fig 1) to the difference between the turbine measured observed values and the values which would have been obtained with a peak flow meter. Since they are of opposite direction, they obscure the spuriously low absolute values measured by the turbine spirometer. Nevertheless, Thiadens *et al* considered that, in 19.2% of their patients, low values of PEF were associated with normal values of FEV_1 whereas in only 3.3% of patients was a normal value of PEF associated with an abnormally low value of FEV_1.

To evaluate the relative merits of FEV_1 and PEF as indices of bronchodilator reversibility Thiadens *et al* compared changes in FEV_1, expressed as percentage differences in predicted values, with changes in PEF expressed as percentage differences in absolute values. The dissimilar manner in which the values were expressed invalidates their comparison and, hence, any conclusions drawn from it; it also makes the authors' prolix discussion of the sensitivity and specificity of their findings wholly irrelevant.

There is no justification for stating that a cut off value for a rise in FEV_1 after a bronchodilator is "useful and valid . . . in separating asthma from COPD" since a bronchodilator reveals only immediate reversibility. Moreover, true irreversibility does not necessarily signify COPD since it may be present in patients with longstanding asthma in whom structural damage of the bronchi has occurred. The most convenient and reliable test in primary care for distinguishing between potentially reversible and truly irreversible airflow limitation is still twice or thrice daily monitoring of PEF during a course of corticosteroid treatment.[6]

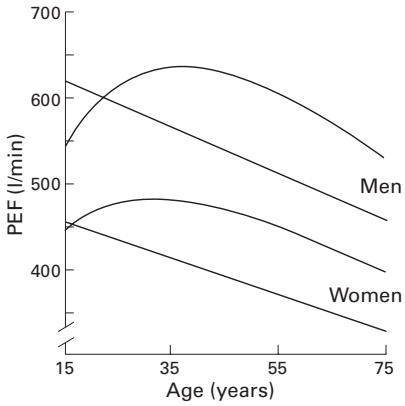

Figure 1 Linear regressions of peak expiratory flow with age in men and women of height 175 cm and 160 cm, respectively, of the ERS[3] compared with curvilinear regressions of Nunn and Gregg.[4]

The conclusion by Thiadens *et al* that FEV_1 is more reliable than PEF for assessment of airflow limitation and its reversibility is not supported by their findings. Although I am very reluctant to criticise their study, attention needs to be drawn to its faults because the prominence given to their study by *Thorax* is likely to persuade general practitioners that its findings are valid and its conclusions are authoritative.

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1 Thiadens HA, De Bock GH, Van Houwelingen JC, *et al*. Can peak expiratory flow measurements reliably identify the presence of airway obstruction and bronchodilator response as assessed by FEV_1 in primary care patients presenting with a persistent cough? *Thorax* 1999;**54**:1055–60.
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4 Nunn AJ, Gregg I. New regression equations for the prediction of peak expiratory flow in adults. *BMJ* 1989;**298**:1068–1070
5 Quanjer PhH, Lebowitz MD, Gregg I, *et al*. Peak expiratory flow: conclusions and recommendations of a Working Party of the European Respiratory Society. *Eur Respir J* 1997; **10**(Suppl 24):2–8s.
6 Gregg I. Reversibility of airways obstruction in asthma. *Respiration* 1969;**26**(Suppl):123–30.

AUTHORS' REPLY Dr Gregg's remarks on our paper concern four major points: (1) the differences between the Micro medical spirometer and the mini-Wright peak flow meter; (2) the choice of predicted values; (3) the "dissimilar manner" in which changes in PEF were compared with changes in FEV_1; and (4) the use of changes in FEV_1 rather than changes in the FEV_1/FVC ratio.

(1) It is true that there are differences between the two devices based upon the different principles—turbine flow measurement and variable orifice peak flow measurement. As we stated in the discussion part of the paper, the turbine flow meter yields slightly lower values than a pneumotachograph[1 2] and the variable orifice peak flow meter shows significantly higher values (200–300 l/min) than a pneumotachograph in the mid region.[3] In both cases the pneumotachograph value is considered the reference value. Without pertaining to the brand of the portable spirometer used, it appears that the turbine values generally meet the criteria for monitoring devices set by the ATS.[4] In any case, the differences between the devices are systemic and should not interfere with the results of the study, provided that the same device is used throughout it.

(2) In the discussion this issue has also been questioned. The choice of the predicted values depends in part on the equipment used. The ERS predicted values for PEF are obtained from a mixture of pneumotachograph data and Wright peak flow data whereas the values proposed by Dr Gregg are obtained from mini-Wright peak flow meters. The values produced by the turbine spirometer come closest to the pneumotachograph values. In view of this, the ERS values are probably the best choice.

(3) This issue was referred to in the discussion of our paper. We did not only compare the changes in FEV_1 expressed as percentage differences of predicted values (with changes in PEF expressed as percentage differences in absolute values); we also compared changes in FEV_1 as percentages of the initial values (including absolute improvement of 200 ml) with changes in PEF, both absolute and percentage, to the initial values (see table 3). We agree that measuring longitudinal reversibility with a corticosteroid is the best method, although we prefer to use the FEV_1 value at the start and the FEV_1 after some weeks of corticosteroid treatment as outcome parameters. This issue was also discussed in the editorial by Professor Jones.[5]

(4) Generally, the FEV_1/FVC ratio is a reliable indicator of bronchial obstruction provided the manoeuvre is carried out correctly. This is a problem with hand held spirometers, and the recommendations are that the expiratory curve be followed in real time to ensure that a true beginning and end of the forced expiration is detected. This is not possible with hand held spirometers and inevitably leads to falsely low FVC values. In our opinion, therefore, it is wise to exclude this parameter from analysis.

Although Dr Gregg is very definitive in his opinion about the value of peak flow measurements, especially using the mini-Wright meter, he has not been able to convince us, nor has he referred to validity studies about the accuracy of this device in demonstrating (reversible) airflow limitation.

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1 Richter K, Kannies F, Mark B, *et al*. Assessment of accuracy and applicability of a new electronic peak flow meter and asthma monitor. *Eur Respir J* 1998;**12**:457–62.
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Differentiation between mitral stenosis and coexisting PPH

I read with great interest the case report by Langleben *et al*[1] of a woman with coexisting mitral stenosis and primary pulmonary hypertension (PPH) or plexogenic pulmonary arteriopathy who succumbed to operation for relief of her mitral stenosis. The failure of the patient's pulmonary hypertension to decrease postoperatively led to her fatal outcome because the coexisting primary pulmonary hypertension was not recognised preoperatively.

I would like to call attention to the fact that the diagnosis should have been suspected preoperatively because her pulmonary artery wedge pressure was only modestly raised (16 mm Hg; normal = 12) and her pulmonary vascular resistance was extremely high (1823 dynes s cm^{-5}; normal = 67 (30)). In the presence of severe mitral stenosis the pulmonary artery wedge pressure, which reflects the left atria1 pressure, is usually substantially increased whereas the pulmonary vascular resistance is usually normal or mildly raised in the presence of "reactive" as well as "passive" pulmonary hypertension. On the other hand, in primary pulmonary hypertension the pulmonary artery wedge pressure is usually normal and the calculated pulmonary vascular resistance is extremely high.

Case reports like that of Langleben *et al*[1] illustrate the importance of careful analysis of the haemodynamic data obtained at cardiac catheterisation "in order to identify plexogenic pulmonary arteriopathy (or primary pulmonary hypertension) obscured by or masquerading as other disorders" such as mitral stenosis.

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1 Langleben D, Lamoureux E, Marcotte F, *et al*. Mitral stenosis obscuring the diagnosis of plexogenic pulmonary arteriopathy and familial pulmonary hypertension. *Thorax* 2000; **55**:247–8.

AUTHORS' REPLY Dr Cheng's analysis of the patient we presented is somewhat superficial from several aspects. Firstly, by current convention and by definition, a mean pulmonary capillary wedge pressure of more than 15 mm Hg is not consistent with the diagnosis of primary pulmonary hypertension. With that finding one must begin to suspect other diagnoses. The detection of severe mitral valvular disease on an echocardiogram also precludes a diagnosis of primary pulmonary hypertension. Secondly, we agree that most patients with mitral stenosis have greatly increased wedge pressures making the diagnosis obvious and easy. However, there is a subset of patients with severe mitral stenosis who present with a "markedly reduced cardiac output and a low transvalvular pressure gradient"[1]—that is, a relatively low wedge pressure. Thus, the absence of a very high wedge pressure does not preclude the diagnosis of mitral stenosis, particularly when the cardiac output is low. Casual dismissal of a relatively low wedge pressure could lead to a failure to detect mitral stenosis. Thirdly, Dr Cheng's statement that the pulmonary vascular resistance is "usually normal or mildly raised" may refer to mild mitral stenosis, but it has been recognised for 50 years that pulmonary vascular resistance can rise disproportionately to the left atrial pressure in humans.[2] Moreover, it has been recognised that extreme elevations in pulmonary vascular resistance can occur in mitral stenosis in at least 10% of patients in many series.[3 4] This probably reflects a genetic variation within the population as we discuss in our case report. In addition, the literature describes cases of extreme elevation of pulmonary vascular resistance in mitral stenosis, even in the presence of a "low" wedge pressure.[5] Our patient fits this profile.

Thus, this case was much more complex than Dr Cheng implies. Had we ignored the echocardiographic data and initially assumed, as he does, that she obviously had primary pulmonary hypertension, then administration of currently accepted therapy for that disease—that is, vasodilators—would probably have killed her by producing pulmonary oedema from an inability of the lung to drain through a stenosed mitral valve. That potential outcome suggests that, while careful analysis of haemodynamic data obtained at cardiac catheterisation is, of course, essential, a superficial perusal of the subtleties of pulmonary vascular disease is equally dangerous.

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1 Braunwald E. Valvular heart disease. In: Braunwald E, ed. *Heart disease: a textbook of cardiovascular medicine*. 4th ed. Philadelphia: W B Saunders, 1992: 1009.
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Caring for indigenous Australian children with asthma

We applaud Dr Partridge's recent thought provoking editorial[1] which is timely with recent articles focusing on medicine, poverty, and marginalised groups.[2] We wish to add that the issues raised by Partridge are also highly relevant in Australia with respect to indigenous Australians who have unacceptably high levels of morbidity and mortality compared with non-indigenous Australians.[3] Also, in addition to the influence of the doctor/patient relationship on health care, we wish to question the model of care used by doctors and other health care providers when servicing minority groups.[4]

We have had the privilege of providing a paediatric respiratory outreach service to remote indigenous communities in far north Queensland over the last three years. In these children we found a high rate of persistent asthma and non-optimal use of asthma devices as well as poor asthma knowledge. Also, by using the community controlled primary health care model instead of the standard practice of servicing through the hospital system, we were able to achieve very high attendance rates (98%) at our clinics[4] as well as in our recently completed prevalence study (95%) (unpublished). Although high attendance rates may not necessarily equate to better care, they do provide a greater opportunity for addressing important elements of health maintenance such as health education and preventative medicine in contrast to an "acute medicine" approach.

It is easy for doctors to resort to a defeatist approach when providing care to minority groups—put the onus on the patients and blame culture and language differences. It is harder to examine and question one's interaction with one's patients and critically to examine how best to provide a genuine service. As stated by Richard Smith: "they deserve the best, not the poorest, care".[2]

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AUTHOR'S REPLY Professor Chang and Drs Shannon and Masters make important points that widen the issue to remind us all that there are other sectors of society who need special attention. My personal view is that the problems that affect asthma care delivery are the same throughout the world; it is only the magnitude of the individual problems that varies from country to country.

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Intravenous montelukast in acute asthma: expensive aminophylline?

We read with interest the recent paper by Dockhorn *et al* comparing the effects of single doses of intravenous and oral montelukast on forced expiratory volume in one second (FEV_1) in patients with chronic persistent asthma.[1] We agree with the accompanying editorial, that a study of montelukast in acute severe asthma is now warranted.[2] Indeed, few

published studies have examined the use of leukotriene receptor antagonists in patients with severe persistent asthma.[3]

There are several good reasons why an intravenous leukotriene receptor antagonist might be effective in acute severe asthma, including evidence that high dose oral steroids do not affect leukotriene synthesis in vivo,[4] and that induced sputum cysteinyl leukotriene concentrations are significantly higher in subjects with acute severe asthma than in patients with milder asthma and normal controls which suggests that leukotrienes may be more functionally important in patients with acute severe asthma.[5]

The question that most clinicians wish to answer is whether adding an intravenous leukotriene receptor antagonist will produce further improvements in patients with acute severe asthma who have already received conventional first line treatment including nebulised high dose salbutamol, ipratropium bromide, and systemic corticosteroids.[6] In other words, is an intravenous leukotriene receptor antagonist any better as second line intravenous treatment than aminophylline or salbutamol? The results of such studies are awaited with keen interest. It is behoven upon the pharmaceutical industry to sponsor such studies, asking clinically relevant questions regarding the use of leukotriene receptor antagonists as second line intravenous treatment.

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Conflict of interests: The Asthma & Allergy Research Group have received funding and educational support from the following companies: Astra-Zeneca, GlaxoWellcome, Aventis, Merck, Sharp & Dohme, and Schering-Plough.

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6 Lipworth BJ. Treatment of acute asthma. *Lancet* 1997;**350**(Suppl 2):8–23.

AUTHORS' REPLY We would like to thank Drs Dempsey and Lipworth for their concise thoughts on the topic of the additive effects of antileukotriene drugs in acute asthma.

In chronic asthma antileukotriene drugs have been shown to have additive benefits with β agonists,[1–3] corticosteroids,[4] and theophylline.[2] Since acute asthmatic episodes are included in the spectrum of the asthmatic response, it is highly likely that antileukotriene drugs will provide at least similar additive effects in this setting.

Adult studies with antileukotrienes in acute asthma in addition to usual treatment will be required. Once safety and efficacy have been demonstrated in adequately controlled trials in adults, paediatric studies or trials with active comparators can be considered.

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1 Altman LC, Munk Z, Seltzer J, *et al.* A placebo-controlled, dose-ranging study of montelukast, a cysteinyl leukotriene-receptor antagonist. *J Allergy Clin Immunol* 1998;**102**:50–6.
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TB guidelines

We read the BTS guidelines on the management of tuberculosis[1] with great interest. For the most part the paper is an excellent summary of best practice and a good reference for a number of difficult situations. We were, however, less happy about the recommendation to move to a four drug regimen for most patients. We wonder if it is legitimate to generalise a practice which may be sensible in London with a significant refugee problem but which may be unnecessary in other parts of the UK.

The recommendation for a four drug regimen is graded as A (requires at least one randomised control trial). Two references are given for the statement. One, a conference report,[2] lists ethnic risk factors for single and multidrug resistance drawn from the UK reference laboratory reporting service for tuberculosis. The other[3] is the report on the 1993 tuberculosis survey in England and Wales and draws attention inter alia to the small but rising incidence of drug resistance between 1988 and 1993. Neither is a controlled trial.

Single drug resistance has been with us from the earliest days of chemotherapy and three drug regimens have not been found wanting in the succeeding 50 years. Thus, the statement justifying a four drug regimen to counter this problem is surprising.

Multidrug resistance (to isoniazid and rifampicin) is another matter. However, data from the UK reference laboratory reporting system for tuberculosis (Mycobnet) give reason to pause. Of 93 isolates of multidrug resistant bacteria for 1997–8, 23 were also resistant to ethambutol, eight to pyrazinamide, and 15 to both. Thus, a four drug regimen for all these patients would either have been ineffective or would have led to further resistance developing in about half of the patients. Data for Leicestershire (which notifies about 200 cases each year) indicate that isolates from all seven cases of multidrug resistant tuberculosis identified in 1993–8 were resistant to at least one other drug.

In view of these results, it seems better to concentrate on obtaining bacteriological proof of resistance whenever possible, the use of more rapid methods for detecting resistance, and even withholding treatment in well non-infectious patients until sensitivity tests are available. A wholesale move to a four drug regimen will increase side effects, decrease compliance, and may not do much to counter the problem of multidrug resistance.

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1 Joint Tuberculosis Committee of the British Thoracic Society. Chemotherapy and management of tuberculosis in the United Kingdom: recommendations 1998. *Thorax* 1998;**53**:536–48.
2 Hayward AC, Bennettt DE, Herbert J, *et al.* Risk factors for drug resistance in patients with tuberculosis in England and Wales 1993–94. *Thorax* 1996;**51**(Suppl 3):S32.
3 Kumar D, Watson JM, Charlett A, *et al.* Tuberculosis in England and Wales in 1993: results of a national survey. *Thorax* 1997;**52**:1060–7.

AUTHORS' REPLY Dr Cookson and colleagues question the recommendation to move to a four drug initial phase regimen for most patients in the 1998 guidelines on the management of tuberculosis.[1] A four drug initial phase regimen has been advised in the UK since 1990, with the omission of ethambutol from the initial phase recommended to those at low risk of isoniazid resistance.[2] The 1998 recommendations were made with the knowledge of the drug resistance rates and epidemiology from Mycobnet, both published and unpublished.

These data show that drug resistance is not confined to London but occurs in many geographical areas, with significant rates of isoniazid resistance in defined groups—that is, those with a history of prior treatment (irrespective of ethnic group), in ethnic minority groups, and in those who are HIV positive (irrespective of ethnic group).

The scientific and evidence base for short course chemotherapy was set out in detail in the recommendations[1] in the section immediately preceding the detailed treatment recommendations commented on by Cookson *et al*. References 13–19 of the 1998 recommendations were controlled trials largely with a four drug initial regimen, although giving evidence that a three drug initial phase is satisfactory in those with *fully sensitive organisms.* The only controlled trial of six month short course chemotherapy for pulmonary disease in the UK (reference 13 of the recommendations) was with four drug initial regimens.[3] The recommendation for an initial phase of four drugs is thus clearly based on multiple clinical trials and is therefore an A grade recommendation as stated. This recommendation is to cater for the significantly more common isoniazid resistance[4] and is not based on current rates of multidrug resistant tuberculosis.[4] The British Thoracic Society is not alone in recommending a four drug initial regimen for those at significant risk of isoniazid resistance. This policy is also advocated by the American Thoracic Society[5] if the isoniazid resistance is >4% and the European Respiratory Society/International Union against Tuberculosis and Lung Diseases/World Health Organisation[6] for most cases of tuberculosis. It is also advocated on the basis of bacteriological and controlled trial data by one of the pioneers of short course chemotherapy, Professor D Mitchison, who states: "If resistant strains are

found more often, for instance in 3–10% of untreated patients, a fourth drug, usually ethambutol but sometimes streptomycin, is added".[7] There is also no evidence from published national audit studies or from programmatic data to support the statement by Cookson *et al* that a four drug regimen will "increase side effects or decrease compliance".

The recommendations[1] make explicit the need to obtain bacteriological confirmation and hence drug susceptibility whenever possible, and the need to be aware of rifampicin resistance and the use of molecular methods for detecting its presence. In this part, the recommendations meet the comments of Dr Cookson and colleagues.

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BOOK REVIEWS

Hunter's Diseases of Occupations. PJ Baxter, PH Adams, T-C Aw, A Cockcroft, JM Harrington, Editors. (Pp 1001, hardback; £155.00). UK: Arnold, 2000. ISBN 0 340 67750 3

In 1955 the first edition of Donald Hunter's book *Diseases of Occupations* was published. He said that he wanted to emphasise the clinical aspects of occupational disease and this remains true in the ninth edition of "Hunter's". The five main editors of this edition and the majority of contributors are UK based. However, given that its primary focus is clinical information, its contents ought to be valid worldwide. Clearly, for issues relating to country-specific health legislation, you may need to look elsewhere (although UK readers are catered for reasonably well).

The 1001 pages of this edition of Hunter's are divided into 11 chapters (parts) with each part being divided into further subsections. Five of the parts are covered sufficiently with one or two subsections ('Reproduction at work', 'Occupational cancer', 'Occupational diseases of the skin', 'Diseases associated with microbiological agents', and 'Diseases related to ergonomic and mechanical factors'), but the chapter on 'Diseases associated with physical agents' requires nine subsections. The larger chapters include 'Diseases associated with chemical agents' (over 200 pages), 'Diseases associated with physical agents' (170 pages), and 'Occupational lung disorders' (132 pages). This edition adds in a chapter on 'Nephrotoxic, neurotoxic, hepatotoxic and haemopoietic effects of workplace exposures' that is useful in compiling a differential diagnosis list for work related possibilities. The index itself is a healthy 74 pages with a reasonable amount of cross referencing but, if the book was available as a CD-ROM, it would be even better; reference books should embrace this useful technology.

Hunter's has some particularly readable sections which give the salient facts and information embellished with nuggets of background or historical data on the condition or disease. For example, the section on 'Hand-arm vibration' covers diagnosis, treatment/management, current techniques for objective testing with comments on specificity and sensitivity, and just enough on the physics of vibration. It also points out that the adverse health effects were recognised by 1918—all this is contained in eight pages supported by four figures, three tables, and 100 references.

What was the book like over a three month period of use? I would dip into it for a specific query and find myself enticed into further pages of reading. Perhaps this was because of writing this review but, equally, the prose around the primary information held my attention. Questions I asked included "Do mobile phones cause cancer?" Hunter's says "no . . . causal link", "electric and magnetic fields does (sic) not function as either a mutagen or a complete carcinogen" and "it is clear that EMF do not pose a large public health or occupational hazard". This was useful when dealing with a worried well patient.

What is missing from this book? A CD-ROM version, email addresses for the contributors, and a list of established occupational health internet sites. Hunter's is a "must have" for the bookshelf of any self-respecting occupational physician—I had already obtained my own copy before receiving the review copy. It should be useful for GPs and hospital physicians dealing with a possible occupationally related diagnosis or help to exclude it. Lawyers will have a copy and, if you are involved in tribunals or medicolegal reports, so should you. Its strength is to be clinically comprehensive both in range (from the very obscure to the more common occupational disorders) and in depth, as well as offering practical advice on management. Hunter's is a good book worthy of a personal (albeit expensive) copy. Buy it if you are frequently or infrequently asked the question "Is it my work doctor?".—SK

Cystic Fibrosis Medical Care. David M Orenstein, Beryl J Rosenstein, Robert C Stern. (Pp 365, paperback). USA: Lippincott Williams & Wilkins, 2000. ISBN 0-7817-1798-1.

Cystic Fibrosis Medical Care is too big to fit into a clinician's pocket and too small to justify a place on a reference book shelf. It is described as a practical and easy to use reference book, and by the authors as an introduction to the principles and practices of cystic fibrosis medical care. In doing so it has missed its target audience and has fallen between two stools. Some chapters provide an excellent overview of difficult issues surrounding cystic fibrosis care, such as Chapter 3 on the diagnosis of cystic fibrosis, while others, particularly Chapter 4 on the treatment of pulmonary exacerbations, did not address the problem in any depth.

In addition, there is a strong transatlantic emphasis on practical care which may not always be applicable to European cystic fibrosis clinics. As such, this book will appeal to North American practitioners who, accepting its limitations, may wish only to dip into some of the complex issues surrounding cystic fibrosis care.—KHVT

NOTICE

Dr H M (Bill) Foreman Memorial Fund

The trustees of the Dr H M (Bill) Foreman Memorial Fund invite applications for grants relating to study in respiratory disease and allied fields. Limited funds are available for registered medical practitioners to assist in travelling to countries other than their own to study respiratory disease, and also for support for clinical research abroad.

Intending applicants should write for further details to: Dr Brian H Davies, Llandough Hospital, Penarth, Vale of Glamorgan CF64 2XX, UK.