LETTERS TO THE EDITOR

PEF versus FEV1

The assertion by Dr Thiadens and colleagues1 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 (FEV1) 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 microplethysmograph, 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 FEV1 for estimating magnitude of airflow limitation, Thiadens et al 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 PEF5 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 two devices in 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 PEF was associated with normal values of FEV1, whereas only 3.3% of patients was a normal value of PEF associated with an abnormally low value of FEV1.6

To evaluate the relative merits of PEF, relative to FEV1 as indices of bronchodilator reversibility Thiadens et al compared changes in PEF,7 expressed as percentage differences in predicted values, with changes in FEV1 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’ proflit 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 PEF, after a bronchodilator is “useful and valid in separating asthmatic from COPD8 since a bronchodilator reveals only immediate reversibility. Moreover, true reversibility 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 common

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' compared with curvilinear regressions of Nunn and Gregg.


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 FEV1; and (4) the use of changes in FEV1, rather than changes in the FEV1/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 pneumotachograph9 and the variable orifice peak flow meter yields significantly higher (200–300 l/min) than a pneumotachograph in the mid region.10 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 airway obstruction.11

(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 and mini-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 this case, 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 FEV1, expressed as percentage differences of predicted values, but also compared differences in PEF expressed as percentage differences in absolute values); we also compared changes in FEV1, 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 FEV1 value at the start and the FEV1 after some weeks of corticosteroid treatment as outcome parameters. This issue was also discussed in the editorial by Professor Jones.

(4) Generally, the FEV1/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 for at least 2 s 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 FEV1 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 useful with 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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Differentiation between mitral stenosis and coexisting PPH

I read with great interest the case report by Langleben et al 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⁻¹; normal = 67 (30)). In the presence of severe mitral stenosis the pulmonary artery wedge pressure, which reflects the left atrial pressure, is usually substantially increased whereas in the presence of high 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 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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AUTHORS’ REPLY Dr Cheng’s analysis of the patient we presented is somewhat superficial. The George Washington University, Flinders University Northern Territory Clinical School, Alice Springs Hospital, Australia


AUTHORS’ 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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1 Partridge MR. In what way may race, ethnicity or culture influence asthma outcomes? Thorax 2000;55:175–6.


AUTHOR’S REPLY Dr Partridge’s recent thought provoking editorial which is timely with recent articles focusing on medicine, poverty, and marginalisation. We wish to add that the issues raised by Partridge are also highly relevant in Australia with respect to indigenous Australians who have unacceptable levels of morbidity and mortality compared with non-indigenous Australians.1 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 serving minority groups.

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 follow-ups 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 revert 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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Caring for indigenous Australian children with asthma

We applaud Dr Partridge’s recent thought provoking editorial which is timely with recent articles focusing on medicine, poverty, and marginalisation. We wish to add that the issues raised by Partridge are also highly relevant in Australia with respect to indigenous Australians who have unacceptable levels of morbidity and mortality compared with non-indigenous Australians.1 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 serving minority groups.

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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 (FEV₁) in patients with chronic persistent asthma.3 We agree with the accompanying editorial, that a study of montelukast in acute severe asthma is now warranted.4 Indeed, few
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published studies have examined the use of leukotriene receptor antagonists in patients with severe persistent asthma.1

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,2 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.1

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.1 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, and non-infectious patients until sensitivity tests were done on patients. Data for Leicestershire (which notified about 200 cases each year) indicate that resistance to ethambutol, eight to pyrazinamide, three to rifampicin, and seven to streptomycin was noted among 20% of patients. Data for the three midlands and the north and is not based on current rates of multidrug resistant tuberculosis.2

The scientific and evidence base for short course chemotherapy was set out in detail in the recommendations3 in the section immediately preceding the detailed treatment recommendations commented on by Cookson et al. References 13–19 of the 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 few drug resistant strains. 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. 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 significant more common isolates of resistance 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 position is also advocated by the American Thoracic Society5 if the isoniazid resistance is >4% and the European Respiratory Society/International Union against Tuberculosis and Lung Diseases/World Health Organisation6 in most cases of tuberculosis. It is also advocated on the basis of bacteriological and controlled trials data by one of the pioneers of short course chemotherapy, Professor J Mitchison, who states: “If resistant strains were

TB guidelines

We read the BTS guidelines on the management of tuberculosis7 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 regi-

mern for all these patients would either have been ineffective or would have led to further resistance developing in about half of the patients. Data from before this phase (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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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 corticosteroids,1 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 con-

trolled trials in adults, paediatric studies or trials with active comparators can be considered.

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found more often, for instance in 3–10% of untreated patients, a fourth drug, usually ethambutol but sometimes streptomycin, is added”. 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 make explicit the need to establish bacteriological confirmation and hence drug susceptibility whenever possible, and the need to be aware of rifampin 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.

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 engineering, workshop and factory conditions’), 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 made explicit the need to study in respiratory disease and also for support for clinical research abroad.

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.
Intravenous montelukast in acute asthma: expensive aminophylline?

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