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 Mullee¹, who compared a similar Microlab turbine spirometer with a micro-aerotachograph, 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 expressed observed values of each as percentage predicted, using the reference values for each sex recommended by the European Respiratory Society (ERS).¹ 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,¹ which an ERS Working Party on PEF² 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 values with a micro-aerotachograph. Thiadens et al considered that, in 19.2% of their patients, low values of PEF were associated with normal values of FEV₁, whereas in only 3.3% of patients was a normal value of PEF associated with an abnormally low value of FEV₁.³

To evaluate the relative merits of PEF, relative to FEV₁ as indices of bronchodilator reversibility Thiadens et al compared changes in PEF, expressed as percentage differences in predicted values of PEF 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’ prolific 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 asthma from COPD” 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 values (200–300 l/min) than a pneumotachograph in the mid region.¹ 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 values measured generally met the criteria for monitoring airway obstruction in ATS.¹ 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.

(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 (micro-aerotachograph) and variable orifice peak flow meter 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 either of the two cases, 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 of changes 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 parameter. 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 spiro meters, and the recommendations are that the expiratory curve be followed for a full second to ensure that a true beginning and end of the forced expiration is detected. This is not possible with hand held spiro meters 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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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 pneumotachograph⁴ and the variable orifice peak flow meter was significantly higher

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 curve-linear regressions of Nunn and Gregg.⁵

Letters to the editor

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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. 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 (90%) at follow-up 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.

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


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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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. We agree with the accompanying editorial, that a study of montelukast in acute severe asthma is now warranted. Indeed, few
The recommendation for a four drug regimen is graded A (requires at least one randomised control trial). Two references are given for the statement. One, a conference report, lists ethnic risk factors for single and multidrug resistance. The other is the report on the 1993 tuberculosis survey in England and Wales which may be unnecessary in other parts of the UK.

The recommendation for a four drug regimen to deal with isoniazid resistant tuberculosis identified in 1993–8 and is not based on current rates of multidrug resistant tuberculosis. The 1998 recommendations were made with the knowledge of the drug resistance rates and epidemiology from Mycobact, both published in the UK since 1990, with the omission of ethambutol from the initial phase regimen that is the best way to prevent progression to multidrug resistance and isoniazid resistance.

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 in the section immediately preceding the detailed treatment recommendations commented on by Cookson et al. References 13–19 of the 1998 recommendations commented on by Cookson et al. were controlled trials largely with a four drug initial regimen, although giving evidence that a three drug initial phase is satisfactory in those with drug resistance. 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 significantly higher rates of isoniazid resistance.

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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 elicit a characteristic 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


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 ‘Nephrotic, 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.

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. —SK


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.

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Differentiation between mitral stenosis and coexisting PPH

TSUNG O CHENG

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