P E F v ersu s F E V ,

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 to values measured with a microLung function analyser, 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 on 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).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 PE F5 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 methods of expressing 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 have failed to explain why their methods have led to such a large difference in predicted values, with changes in PEF explained as percentage differences in absolute values. The dissimilar manner in which the values were expressed invalidate their comparison and, hence, any conclusions drawn from it; it also underlines the authors’ profound misunderstanding 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 FEV1 after a bronchodilator is “useful and valid” in separating asthma from COPD6 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.7 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 merit the criteria for monitoring ATS.8 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 and variable orifice 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 changes in PEF in absolute values (including absolute improvement of 200 ml) with changes in PEF, both expressed as percentage differences 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 PE F/FVC ratio is a reliable indicator of bronchial obstruction provided the manoeuvre is carried out correctly. This is a problem with hand held spiroimeters, and the recommendations are that the expiratory curve be followed for some 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 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 when 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.

I A N G R E G G
Eynsham, Witney, Oxford, UK


A U T H O R S ’ R E P L Y

Dr Gregg’s remarks on our paper concern four major points: (1) the differences between the Micro medical spiro-meter and the mini-Wright peak flow meter; (2) the prediction of values; (3) the “dissimilar manner” in which changes in PEF were compared with changes in FEV1; and (4) the use of 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 pneumotachograph;9 and the variable orifice peak flow meter yields significantly higher 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 turbine values generally meet the criteria for monitoring 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.

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D S POSTMA
TH W VAN DER MARK
Department of Pulmonary Diseases, University of Groningen, The Netherlands

H A THIADENS
Department of General Practice, Leiden University Medical Center, 2300 CB Leiden, The Netherlands

Correspondence to: Dr H A Thiadens


www.thoraxjnl.com
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⁻⁵; 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 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 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.

AUTHORS’ REPLY Dr Cheng's analysis of the patient we presented is somewhat superficial — that is, a relatively low wedge pressure could lead to an incorrect diagnosis. 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.

D LANGLEBEN
R SCHLESINGER
Faculty of Medicine,
Sir Mortimer B Davis Jewish General Hospital,
Montreal Quebec H3T 1E2
Canada


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 marginalized communities. We wish to draw attention to the issues raised by Partridge which are 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 marginalized 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 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 (95%) 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. 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 examine how best to provide a genuine service. As stated by Richard Smith: “they deserve the best, not the poorest, care”.

A B CHANG
Associate Professor of Paediatrics,
Flinders University Northern Territory Clinical School,
Alice Springs Hospital,
Australia
email: achang@mac.com

C SHANNON
Director of Indigenous Health Program,
Queensland University of Technology,
Australia

I B MASTERS
Deputy Director of Respiratory Medicine,
Royal Children's Hospital,
Brisbane, Australia

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 vary from country to country.

MARTYN R PARTRIDGE
The Chest Clinic,
Whips Cross Hospital,
London E11 1NR, UK
email: mpartridge@whipschest.cdenemon.co.uk

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
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.\(^3\)

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.\(^4\)

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 beehoven upon the pharmaceutical industry to sponsor such studies, asking clinically relevant questions regarding the utility of leukotriene receptor antagonists as second line intravenous treatment.

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.


AUTHORS’ REPLY We would like to thank Drs Dempsey and Lipworth for their concise 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.

TB guidelines

We read the BTS guidelines on the management of tuberculosis 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.


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 for thoracic radiological 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.

PIETER ORMERO
Department of Respiratory Medicine, Blackburn Royal Infirmary Chairman, Joint Tuberculosis Committee

IAN CAMPBELL
Llandough Hospital Secretary, Joint Tuberculosis Committee


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 ‘Nephrototoxic, 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 writing this review but, equally, the prose

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.