

LETTERS TO THE EDITOR

Comparison of outcome measures in patients with COPD

Harper *et al*¹ found completion rates and success scaling rates of the St George's Respiratory Questionnaire (SGRQ) lower than those observed in other questionnaires completed by patients with COPD attending an outpatient clinic. Readers may infer from this finding that the SGRQ is a less useful instrument than the Chronic Respiratory Questionnaire (CRQ), but an alternative explanation may lie on some methodological limitations of the study.

Firstly, the CRQ was administered only to an opportunistic subsample and, unfortunately, the authors provide insufficient information to rule out systematic differences with the rest of the patients. Secondly, the claim that SGRQ had lower completion rates may reflect the local condition in which it was applied rather than a general limitation. The CRQ was administered by an interviewer while the SGRQ was self-administered, and it is well established that self-administered questionnaires are more prone to missing items than those interviewer-administered. Moreover, supervision of completion can reduce missing data substantially. In a study of ours in 321 men with COPD,² 75% of patients self-completed the SGRQ and only 22% of individuals with missing items were observed. Missing information was reduced to 0% after the imputation algorithm recommended by the developers of the questionnaire was applied. It is important to note that Harper *et al* did not apply the algorithm of imputation in the SGRQ but they did impute the missing items of the CRQ. An appropriate design to allow for comparisons requires, at least, that the same instruments be administered to all the individuals controlling for the order and type of administration.³

Finally, the small sample sizes used may have influenced their results in general and, in particular, do not allow the authors to be conclusive about the lower (54%) success scaling rate of the SGRQ—that is, the proportion of items which correlate >0.4 with their hypothesised dimension. In our study substantially higher success scaling rates were found (78%).²

In conclusion, we believe that the comparison of the SGRQ with the CRQ and other instruments by Harper *et al* is of interest but is inconclusive. A more accurate and specific comparison is therefore still needed.

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1 Harper R, Brazier JE, Waterhouse JC, *et al*. Comparison of outcome measures for patients with chronic obstructive pulmonary disease

(COPD) in an outpatient setting. *Thorax* 1997;52:879-87.

2 Ferrer M, Alonso J, Prieto L, *et al*. Validity and reliability of the St George's Respiratory Questionnaire after adaptation to a different language and culture: the Spanish example. *Eur Respir J* 1996;9:1160-6.

3 Prieto L, Alonso J, Ferrer M, Antó JM for the Quality of Life in COPD Study Group. Are results of the SF-36 health survey and the Nottingham Health Profile similar? A comparison in COPD patients. *J Clin Epidemiol* 1997;50:463-73.

AUTHORS' REPLY We thank Dr Ferrer and colleagues for their interest in our paper. We examined our results in relation to completion in two ways; firstly, patients who completed or did not complete all items of the Impact dimension and found small and inconsistent differences between them; and, secondly, patients who completed all four questionnaires or completed three or less and found that the only significantly different patient characteristic was in the distance walked. We feel that such differences are unlikely to explain those observed in general for completion and consistency for the SGRQ.

At the time of our study a method of imputing was under development for the SGRQ. Besides, we have doubts about the validity of substitution for high rates of missing data, nor are we convinced that encouraging patients to complete omitted items produces valid responses. More supervision than was available in our study may have increased the level of completion at some cost, but not necessarily the level of consistency. However, for routine clinical use what we need are questionnaires which do not require any supervision, such as a self-complete version of the CRQ.

The low correlation of items with their hypothesised dimensions for the SGRQ may be indicative of our patient group, which was clearly described as elderly and with long-standing disease, but in our experience this group is typical of those attending outpatient clinics in the UK.

We would like to point out that in our concluding sentence we were careful to express reservations about the instruments of choice. We suggested further development of the two condition-specific questionnaires and possible additions to the generic SF-36, and would certainly like to encourage more specific research into patient perceived quality of life measures for this significant patient group.

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CFA with preserved lung volumes

Doherty *et al*¹ seem confident of their ability to distinguish emphysematous spaces from large cystic spaces of honeycomb lung on the basis of a wall thickness on HRCT scanning of greater or less than 1 mm. In cryptogenic fibrosing alveolitis (CFA) we think this is difficult unless an assessment of function (with V/Q scanning or expiratory CT scans) is made at the same time.² The situation is

complicated by the ability of fibrosis in mixed emphysematous-fibrotic areas to support the airways and mitigate the expected gas trapping.

"Emphysematous" changes were seen in only six of 21 patients with CFA and preserved lung volumes (only seven of whom had HRCT scans). Were expiratory CT scans performed? We note the normal RV/TLC and FEV₁/VC ratios. In the absence of functional data for these spaces, we reserve judgement about the pathology.

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1 Doherty MJ, *et al*. Cryptogenic fibrosing alveolitis with preserved lung volumes. *Thorax* 1997;52:998-1002.

2 Strickland N, *et al*. *Am J Roentgenol* 1995;161:719-25.

AUTHORS' REPLY We thank Dr Strickland and Professor Hughes for their helpful comments on the differentiation of emphysema from large cystic changes in CFA. We have tried to stress the limitations of our data in the discussion section of our paper and have tried to emphasise that emphysema was found on CT scans in only six of 21 patients with preserved lung volumes, though this included six of the seven patients who had a comparable CT scan appearance. We also commented on the findings of the normal RV/TLC and FEV₁/FVC ratios.

The CT criteria used in our paper to diagnose emphysema were rather more specific than indicated in their letter in that they included the presence of areas of low attenuation or bullae (air spaces with a 1 cm diameter with a wall frequency of less than 1 mm). The former description of unmarginated areas could not be mistaken for the cystic changes of CFA. The subjects with preserved lung volumes did not show more extensive fibrosis which might otherwise have caused pathological enlargement of the air spaces. All this supports our view that their changes were due to emphysema; neither do we believe that the effects of cigarette smoking on the airways are necessarily confined to pathological alveolar destruction. Co-existing small airways disease provides an alternative and plausible explanation of the physiological findings we noted in our patients with preserved lung volumes. Clearly, this is related to their significant cigarette exposure.

Neither expiratory CT scans nor V/Q scanning were performed in these subjects. This reflects the retrospective nature of the data, many of the subjects having been investigated before the publication of the paper by Strickland and Hughes.

Prospective studies utilising their technique would be helpful in providing further information to support our view that the changes seen in patients with CFA with this pattern of physiological abnormality are indeed due to smoking related obstructive lung disease.

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Serum adenosine deaminase activity in pleural effusion

Pleural effusion is a common complication of many disease processes. Tuberculosis is still one of the most important causes of exudative effusions.¹ Over the last decade many workers have emphasised the importance of estimating adenosine deaminase (ADA) in pleural fluid.² ADA activity in human biological fluid results from the action of two isoenzymes—namely, ADA₁ and ADA₂—with different affinities on two substrates, 2' deoxyadenosine and adenosine, respectively. The deaminase ratio (ADA₁/ADA₂) is of value in the correct differentiation of the aetiology of the disease, whether it is infectious or neoplastic.³

One hundred patients (84 men) with pleural effusions admitted to a chest ward were divided into four groups including 41 patients with tuberculous pleurisy and 15 patients with malignancy. A significant increase in total ADA was observed in those with tuberculous effusion compared with those with benign acellular effusion (98.52 (20.41) vs 16.25 (1.35) IU/l, $p < 0.001$; ADA ratio < 0.28 vs 0.30). In addition, patients with metapneumonic pleurisy had high ADA activity (100.35 (25.65) IU/l) with an ADA ratio of 0.55 (0.05), and those with malignant effusion had an ADA ratio of 0.57 (0.040) with total ADA activity of 37.41 (1.64) IU/l. In patients with tuberculous effusion the test had 90% sensitivity and 87% specificity.

We thus observed significantly raised ADA values in patients with tuberculous, metapneumonic and malignant effusions, but the ADA ratio was < 0.45 in those with tuberculous effusions and > 0.45 in those with malignant or metapneumonic effusions. The ADA₁ isoenzyme is intracellular in location and is essential for the differentiation of lymphoid cells, particularly T cells.³ An increase in ADA₁ can be attributed either to extensive cellular necrosis or to increased turnover of lymphoid cells as occurs in metapneumonic pleurisy and malignancy. ADA₂ is found only in monocyte macrophages and is released into biological fluids (pleural, peritoneal, CSF and serum) when they harbour a micro-organism.³ High total ADA activity and a deaminase ratio of < 0.45 implies an increase in ADA₂ and the patient is most likely to be infected with an intracellular organism—for example, tuberculosis. On the other hand, high total ADA activity and a deaminase ratio of > 0.45 correlated well with malignancy or empyema in the present study. Our findings suggest that the deaminase ratio may be a useful screening test in the diagnosis of exudative pleural effusions.

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- 1 Niwa Y, Kishimoto H, Shimokata K. Carcinomatous and tuberculous pleural effusions. Comparison of tumour markers. *Chest* 1985;87:351–5.
- 2 Banales JL, Pineda PR, Fitzgerald JM. Adenosine deaminase in the diagnosis of tuberculous pleural effusions. *Chest* 1991;99:355–7.
- 3 Gakis C, Naitana A, Ortu AR, et al. Adenosine deaminase activity in the diagnosis of infectious diseases. *Infect Med* 1994;11:219–24, 232.

BOOK REVIEWS

Interpretation of Pulmonary Function Tests: A Practical Guide. Hyatt RE, Scanlon PD, Nakamura M. (Pp 212; £30.50). USA: Lippincott-Raven, 1997. ISBN 0 316 26261 7.

It is the authors' stated aim to produce a concise and practical guide to the interpretation of pulmonary function tests. As learning to interpret pulmonary function tests often appears daunting to both medical and technical trainees, such a book would be a useful addition to the literature on the subject. The sleeve notes say this book is "the only practical guide to the optimal clinical use of pulmonary function tests". I had therefore been expecting a different format from standard texts, but 10 of the 15 chapters are still descriptions of the various tests and how they change in disease. The last four chapters are of a more practical nature, describing changes in lung function in disease including a useful section on which tests are likely to be most helpful in various clinical settings, although there is no mention of AIDS or haematological problems. Chapter 14 comprises a step by step approach to the interpretation of pulmonary function tests; although it gives the correct interpretation to the tests, it covers eight pages of text and figures and is rather difficult to use—perhaps the data could have been incorporated into a flow diagram.

A practical guide to interpreting pulmonary function tests that is easy to read and understand would be very useful; however, this book illustrates that it is difficult to achieve this concisely and still provide an understanding of the physiology involved. The authors suggest that structured approaches to the interpretation of pulmonary function tests have limitations in describing a "gestalt" approach, looking at the spirometric results and using the lung volumes and gas transfer to categorise the anomaly fully. Such methods are used by most experienced reporters of pulmonary function tests, and this method of looking at the whole picture explains why humans are still better than computers at reporting pulmonary function tests.

This is not a bad book and, for anyone wanting an entry level guide to pulmonary function tests, it does make the basic points clearly. However, I feel the reader would soon require a more comprehensive text or an additional volume on physiology.

Lung Function Tests: A Guide to their Interpretation. Kinnear William JM. (Pp 162; £19.50 paperback). Nottingham: Nottingham University Press, 1998. ISBN 1 897676 80 8.

This book adopts a step by step approach to the interpretation of lung function tests. It is aimed at junior doctors specialising in respiratory medicine and clinicians who have contact with patients following lung function assessment. The book deals with the most commonly performed tests but also includes shorter sections on exercise tests and respiratory muscle tests. There is an explanation of predicted values, calculation of normal ranges and standardised residuals (SR). For

the numerous examples of lung function the author comes down firmly on the use of SR to define an abnormal result. As a concession to those who do not calculate the SR, the percentage predicted value is also given. The format guides the reader from the simplistic "within normal limits" to the comprehensive report which recommends additional avenues of investigation and the consideration of likely pathologies. Additional levels of complexity are presented one step at a time and each chapter ends with a useful summary. Test repeatability is dealt with briefly and there is a short chapter on serial lung function tests. This is perhaps too brief and would have benefited from a more detailed assessment and additional examples including some pre and post-treatment changes. The chapter on exercise tests is superficial and perhaps the least helpful. The useful appendix contains 11 worked examples for the reader to test him/herself.

This book is a very accessible introduction to the interpretation of lung function tests. It might easily be used for reference and revision for both measurement practitioners and for the reporting clinician.

Lung Biology in Health and Disease. Volume 111. Dyspnoea. Mahler Donald A. (Pp 432; \$165.00). New York: Marcel Dekker, 1998. ISBN 08247 9814 7.

The preface states that the aim of this volume is to focus on the problem of dyspnoea as a symptom (the manifestation of a pathophysiological condition) and as an illness (the entire range of a person's understanding and response to breathing difficulty).

Fifteen experts in the field have contributed to the 11 chapters in the book. The first presents a conceptual model that considers dyspnoea as a sensation, symptom and illness, and the next chapter explores the language of dyspnoea and the various descriptors that patients with different diseases use to describe their experience of breathlessness. The mechanisms of breathlessness, the diagnosis of the cause, together with how to assess and measure the severity of breathlessness and its impact upon the patient are covered in the chapters that follow. Three further chapters address treatment strategies for relieving breathlessness. These include those specific to the underlying disease process, a very useful chapter on coping strategies, physical modalities such as exercise training and inspiratory muscle training as well as oxygen and other medications. The final chapter evaluates the management of dyspnoea in patients receiving ventilatory assistance. Whilst there is some similarity between the chapters contributed by the same authors to both Mahler's earlier book on dyspnoea (published in 1990 by Futura) and this volume, in each instance the chapters have been expanded to take into account more recent developments. Similarly, whilst some overlap exists with the recent volume on *Respiratory Sensation* in this series, this is largely complementary rather than repetitive.

This book is enjoyable to read. It achieves its stated aims and represents the most comprehensive and up to date summary of knowledge concerning the management of dyspnoea in this format. It is essential reading for professionals involved in research into dyspnoea and is highly recommended to those whose clinical practice largely involves caring for breathless patients.