LETTERS TO THE EDITOR

Who should look after asthma?

The article by Professor Tattersfield and Dr Holmes (June 1995;50:597-9) was full of pertinent and wise statements and observations but we strongly disagree with one of their opinions - namely, that concerning who should look after patients admitted to hospital with severe asthma. They suggest that "... admission under a respiratory physician is likely to be in the patient's interest", but then go on to argue that this may be less good for medical education of juniors and students, implying that asthmatic patients should be admitted to general medical wards rather than specialist units. They also point out that at present there are not enough respiratory physicians. Many audit studies published in the last few years have shown that respiratory physicians deliver a higher quality of inpatient care than do their general physician colleagues. In particular, general physicians prescribe anti-inflammatory treatment less often, are less good at planning to prevent future episodes, and fail to follow up over 40% of cases.¹ A recent article² pointed out that, in cardiac disease, such process measures are probably a more sensitive indicator of standards of care than are direct measures of outcomes, and this conclusion is likely to apply to asthma too since, if asthma prophylaxis is not even prescribed, the patient cannot hope to gain benefit from it.

Tattersfield and Holmes argue that medical students and junior doctors need to see and treat asthma and are fearful that some doctors could fail to learn about it. We agree that all juniors and students need to learn about asthma, but would suggest that it is better that they should rotate through respiratory teams and see a lot of asthma managed well than being exposed to a smattering of asthmatic patients managed in various sub-optimal ways from a range of general medical units. Since respiratory medicine accounts for a quarter of all acute medical admissions,³ it should be possible to organise for house officer and senior house officer rotations to include the speciality and for all medical students to spend some time in it. We would stress that we are not attacking our general physician colleagues: we accept the reverse logic of our arguments in non-respiratory conditions.

The theme of the editorial is the need for a collaborative approach across the primary/secondary interface and again we agree with this; however, it is likely that most general physicians will have other speciality interests and so will not have either the time or the enthusiasm to develop a rapport with general practitioners for the care of patients with asthma. We believe that the patient's interest must come first and that other interests such as education must be subservient. The "competence and consistency" that Tattersfield and Holmes recommend is only likely to occur if inpatient care of asthmatic subjects is provided by respiratory physicians (ideally with respiratory nurse support) who have

active liaison with their local general practitioner colleagues.

A further reason for our view concerns research not mentioned in the editorial. Advances in the management of this, one of the most important of medical emergencies, will be impaired if patients are scattered amongst all the general physicians and around all the medical wards. There are proportionately many more respiratory cases than there are respiratory physicians but this should not deter us from aiming for the best deal for the patient - even if it means having to strive for more respiratory posts. On call care will have to be shared with others, but the person with the responsibility should be a respiratory physician.

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- 2 Mant J, Hicks N. Detecting differences in quality of care: the sensitivity of measures of process and outcome in treating acute myocardial infarction. *BMJ* 1995;311:793-6.
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Community-acquired *Chlamydia pneumoniae* pneumonia

The study by Dr Kauppinen and colleagues (February 1996;51:185-9) contains two interesting clinical aspects: (1) *Chlamydia pneumoniae* caused pneumonia frequently in association with other microorganisms, mainly *Streptococcus pneumoniae*; and (2) the course of this infection was unrelated to the use of appropriate antimicrobial treatment. In addition, asymptomatic carriers have been found by others.¹ All these findings might question the role of *C. pneumoniae* as a pathogenic agent responsible for community-acquired pneumonia.

We recently performed a study to determine the aetiology of community-acquired pneumonia in Lleida (Spain). Traditional diagnostic methods, including paired serum samples for microimmunofluorescence to detect *C. pneumoniae*, were used in combination with polymerase chain reaction (PCR) tests performed on samples obtained directly from lung parenchyma by transthoracic needle aspiration to avoid confounding results. PCR has improved the ability to detect many microbial agents, including *C. pneumoniae*, with a higher sensitivity and specificity than conventional procedures.² Furthermore, transthoracic needle aspiration is a very specific method of obtaining uncontaminated pulmonary samples.

With this method 14 of 119 patients (12%) had a diagnosis of *C. pneumoniae* pneumonia. Serological criteria established the diagnosis in 11 cases and PCR in seven (both methods were positive in four patients). Of these patients three had a dual infection, associated with *S. pneumoniae* in two cases and *S. viridans* in one. The outcome of the patients was retrospectively evaluated in correlation with

the treatment; seven received β -lactam agents only and seven received macrolides, alone or combined with β -lactams. The clinical course of the illness (duration of fever, time in hospital, and incidence of complications) did not differ between the two groups.

We found *C. pneumoniae* in the lung parenchyma of our patients with pneumonia using specific methods, and the clinical results were comparable with those of Kauppinen. Thus, we believe that *C. pneumoniae* is a real pathogen which causes pneumonia. Furthermore, we support the opinion that chlamydial infections can be successfully treated with alternative regimens, particularly β -lactam agents. Prospective studies are needed to explore this possibility.

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- 2 Black CM, Fields PI, Messmer TO, Berdal BP. Detection of *Chlamydia pneumoniae* in clinical specimens by polymerase chain reaction using nested primers. *Eur J Clin Microbiol Infect Dis* 1994;13:752-6.

Fibreoptic bronchoscopy for diagnosis of isolated tuberculous mediastinal lymphadenopathy

We read with interest the report by Baran *et al* (January 1996;51:87-9) on the role of rigid bronchoscopy in the diagnosis of intrathoracic tuberculous lymphadenopathy without parenchymal lesions in a series of 17 adults. Fifteen patients (88%) were found to have various endobronchial abnormalities. Bronchial or transbronchial biopsy specimens were diagnostic in nine (53%). The authors attributed this high diagnostic yield to the use of wide bore needles with the rigid bronchoscope.

We recently reported similar results in a series of 12 HIV negative adults with isolated tuberculous mediastinal lymphadenopathy using fibreoptic bronchoscopy.¹ Isolated tuberculous mediastinal lymphadenopathy was defined as mediastinal lymphadenopathy as the sole detectable manifestation of tuberculosis with negative smear sputum examination. Endobronchial abnormalities were present in nine patients (75%): tracheal, main or segmental bronchus extrinsic compression in eight; tracheal, main or segmental bronchus mucosal inflammation contiguous to an enlarged lymph node in four; endobronchial inflammatory mass contiguous to an enlarged hilar lymph node in three; and endobronchial node fistulisation in three. Diagnosis was obtained by bronchial biopsy in seven cases (58%), mediastinoscopy in four, and computed tomographic-guided transthoracic needle aspiration in one. None of our patients underwent transbronchial biopsy.

This high diagnostic yield of bronchoscopy in patients with isolated tuberculous media-

stinal lymphadenopathy is not surprising. Paediatric series have emphasised the usefulness of bronchoscopy for the diagnosis of endobronchial disease associated with mediastinal lymph node tuberculosis.² In adults Auregan *et al* reported that only three from a series of 140 patients with tuberculous mediastinal lymphadenopathy had a normal fiberoptic bronchoscopic examination and that 135 patients had one or more bronchial fistulae.³

We therefore agree with Baran and colleagues that bronchoscopy is of significant diagnostic value in patients with isolated tuberculous mediastinal lymphadenopathy and may avoid more invasive procedures such as mediastinoscopy. However, rigid bronchoscopy with large sampling and/or transbronchial biopsy is probably not necessary in most patients. We believe that fiberoptic bronchoscopy with bronchial biopsy specimens should be considered as a first line examination in patients with apparently isolated tuberculous mediastinal lymphadenopathy.

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- 2 Powel DA. Tuberculous lymphadenopathy. In: Schlossberg D, ed. *Tuberculosis*. New York: Springer Verlag, 1988:99–108.
- 3 Auregan G, Ali B, Chakib S, Etienne B, Miguères J. Les adénopathies médiastinales tuberculeuses à Djibouti. Rapport de 159 cas. *Rev Pneumol Clin* 1990;46:216–20.

Measuring systemic effects of inhaled beclomethasone

The use of the urine cortisol/creatinine ratio for measuring the effects of inhaled beclomethasone on the hypothalamic-pituitary-adrenal axis described by McIntyre and colleagues (December 1995;50:1280–4), although less cumbersome than measuring the 24 hour urinary free cortisol excretion, nevertheless suffers from the limitation that it is an indirect assessment of plasma cortisol dependent, to a large extent, on an assumption about renal function which cannot be extrapolated to patient subgroups other than those studied by the authors. The use of the low dose adrenocorticotropin test¹ not only circumvents these difficulties, but also appears to be more sensitive than the conventional short synacthen test in revealing impaired adrenal function in asthmatic subjects treated with inhaled corticosteroids. Of 46 subjects tested with 1–24 tetracosactrin in a dose of 0.5 µg/1.73 m², 16 failed to reach a peak plasma cortisol level of > 500 nmol/l following a course of inhaled beclomethasone

dipropionate or budesonide. These 16 patients also had a significantly lower ($p < 0.001$) mean 24 hour urinary free cortisol excretion than patients who responded normally to this test dose. Nonetheless, 15 of these poor responders responded normally to the conventional 250 µg test dose utilised in the short synacthen test.

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BOOK REVIEWS

High Resolution CT of the Chest – Comprehensive Atlas. E J Stern, S J Swenson. (Pp 304; £81.50). Philadelphia: Lippincott-Raven, 1995. 0 397 51451 4.

High Resolution CT of the Lung. W R Webb, N L Muller, D P Naidich. Second Edition. (Pp 336; £96.00). Philadelphia: Lippincott-Raven, 1995. 0 7817 0217 8.

HRCT scanning has made an important contribution to the assessment of intrathoracic disease in the last 10 years. These two American books on HRCT from the same publisher approach the topic in different ways.

The atlas by Stern and Swenson is intended for “private radiology practitioners” and radiology trainees. The authors note that a disease process varies in appearance according to the individual patient and the stage of the disease. They have therefore attempted to present several examples of each condition to give a wider view than the classic presentation. There is a brief chapter on HRCT technique and another on anatomy. Unfortunately, there are no line diagrams to clarify the basic anatomy that HRCT displays. The subsequent 12 chapters deal with specific related types of conditions such as fibrotic lung disease and lung masses. Each HRCT scan has a brief description which includes some general comments on the disease process and specific remarks about the particular radiograph. The last chapter is on pitfalls and artifacts, most of which are well known to those who see a reasonable number of HRCT scans.

The book by Webb *et al* is the second edition of a much respected formal text which has been updated to take account of the advances of the last four years. It has a good section on the technical aspects of HRCT scanning followed by a chapter on anatomy which includes several very helpful line diagrams. There is a chapter on the radiographic findings in HRCT which is then followed by the main body of the book which is divided according to appearances – for example, parenchymal opacification or nodules. This leads to some conditions appearing in several chapters although these are generally cross referenced. It concludes with sections

on the uses of HRCT and an illustrated glossary of terms. All the chapters have tables of features of particular diseases and differential diagnoses.

These books will appeal to different types of readers. Although the book by Webb *et al* costs £15 more, it has 30 more pages and they are full of information whilst a lot of the pages in the atlas are half empty. The images in the atlas are not quite so good. Both contain a list of references but these are much more comprehensive in the book by Webb. Anyone who wants to obtain a good understanding of the basis and use of HRCT scanning will be advised to buy the book by Webb, but those who are more interested in looking at a range of HRCT images without the support of technical and clinical detail will find the atlas useful to flick through for selection of examples. – AM

Non Invasive Respiratory Support. A K Simonds. (Pp 192; £19.99). London: Chapman & Hall, 1995. 0 412 56840 3.

Nasal masks are sprouting on faces over Britain and Europe faster than teenage acne. As the arguments for use of CPAP and NIPPV rage, there are few balanced reviews of the literature to educate those new to the field until now. This knowledgeable, thorough, well argued, and extremely well laid out paperback book on assisted ventilation is now available for both newcomers and “old hands”.

From the first chapter, the potted history sets the scene for the newcomer. Concise, factual, and easy to read, this is not a book to be put down in a hurry. Chapters 1 and 2 cover the background methods and pathophysiology concisely with clear tables and diagrams. Chapters 3 to 5 offer good advice on equipment and setting up patients on the different machines, but this is brief. An overview of assisted ventilation in respiratory failure (Chapter 4), followed by a series of chapters (Chapters 6 to 10) on specific conditions including intensive care units, domiciliary care, neuromuscular disease, chronic obstructive pulmonary disease (COPD), and pre-lung transplantation, give this book its real “value for money” feel. Chapter 9 argues the pros and cons of NIPPV in COPD very knowledgeably and fairly. Chapters 11 and 12 deal very neatly with the high problem/small number of patients seen by only a few specialist centres. Chapter 13 addresses obstructive sleep apnoea and CPAP treatment adequately but is not extensive. The final three chapters are informative on physiotherapy, home care, and the broader picture in Europe, and underline the thoroughness of the book. The only disappointing factor is that some of the figures and illustrations are poor (especially figures 3.5, 3.12, 3.13, and 10.1), and in fig 11.1 the top panel is mislabelled as PaCO₂ when it should be PaO₂. These are minor drawbacks and may represent cost cutting to keep the book keenly priced and thus widely available.

I suggest that any centre considering purchasing any form of non-invasive ventilation should consider buying this book before writing their business plan. For teaching material, this is a modern essential. I would recommend that all personnel involved with assisted ventilation (registrars and above, respiratory scientists and physiologists, nurses and physiotherapists) should buy or read this book soon. – BGC