

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.

M G PEARSON
B D W HARRISON
Fazakerley Hospital,
Liverpool L9 9AL,
UK

M FALGUERA
A NOGUES
A RUIZ-GONZALEZ
Hospital Arnau de Vilanova,
25006 Lleida,
Spain

- 1 Pearson MG, Ryland I, Harrison BDW. A national audit of acute severe asthma in adults admitted to hospital. *Quality in Health Care* 1995;4:24-30.
- 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.
- 3 Pearson MG, Littler J, Davies PDO. An analysis of medical workload by speciality and diagnosis in Mersey: evidence of patient to specialist mismatch. *J R Coll Phys* 1994;28:230-4.

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.

- 1 Hyman CL, Roblin PM, Gaydos CA, Quinn TC, Schachter J, Hammerschlag MR. Prevalence of asymptomatic nasopharyngeal carriage of *Chlamydia pneumoniae* in subjectively healthy adults: assessment by polymerase chain reaction-enzyme immunoassay and culture. *Clin Infect Dis* 1995;20:1174-8.
- 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-