LETTERS TO THE EDITOR

Being positive about the smear

At several points in the recently published Code of Practice 2000,[1] dealing with the control and prevention of tuberculosis in the UK, a number of important actions and decisions turn on the results of sputum microscopy for acid fast bacilli (the "smear"). This simple, cheap, and rapid test is used to assess sputum infectivity and prompts decisions on isolation, initiation of treatment, and the need for and extent of contact tracing. It is also the major criterion used to assess specimen priority for advanced testing, including polymerase chain reaction (PCR) and automated liquid culture.[2]

However, this procedure is unstandardised and smear positivity relative to culture varies from 60% to over 80%. Ziehl-Neelsen staining continues as a primary screen despite good evidence that auramine-based methods are more sensitive and quicker.[3] Processing of sputum before staining may or may not involve digestion and/or concentration by sedimentation or centrifugation, despite advice and evidence that both improve the results.[4] Quality control schemes assess the ability to stain and microscopically examine suspensions of mycobacteria, but not the critical issue of specimen processing before staining.

Less sensitive smear techniques may cause delays in the recognition and management of the index case, including the use of isolation facilities, and an unjustified view of low infectivity which will persist even after the culture is positive. Casual, particularly occupational, contacts of such patients will be at a significant disadvantage. Suboptimal smear techniques will also mean that some specimens meriting examination by enhanced methods will not be sent for such examination. The potential benefits of the *Mycobacterium tuberculosis* PCR will be unnecessarily compromised.

It is now nearly 120 years since Ehrlich first described the acid fastness of some organisms, including mycobacteria. We think it is long overdue that all mycobacteriologists accurately, optimally and, above all, consistently exploit his discovery.

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Churg-Strauss syndrome with montelukast

The case report by Tuggey and Hosker[1] is similar to many of the cases of Churg-Strauss syndrome (CSS) which we have reported in association with zafirlukast,[2] montelukast, and fluticasone/salmeterol.[3] It also shares many similarities with the reports of CSS with inhaled steroid monotherapy.[4 5]

While a temporal relationship with the use of leukotriene modifiers is being reported with increased frequency in association with CSS, a review of this case and others in the literature does not suggest a direct causal relationship with the leukotriene modifiers. Rather, two probable mechanisms seem to predominate. In the first, CSS develops following steroid withdrawal as a result of, or concomitant with, leukotriene modifier use in patients who probably had what was perceived to be severe asthma but was likely to have been CSS masked by steroids (forme fruste CSS).[6] A second mechanism is typified by patients not tapered from systemic steroids. These patients, of which the patient in this case report seems to be an example, have worsening underlying asthma symptoms as the heralding sign of incipient CSS. While, in the past, systemic steroids would have been used to treat this worsening prodromal allergic asthma phase, the recent availability of high dose inhaled steroid therapy and leukotriene modifiers has led to a decrease or delay in systemic steroid prescription for these patients. While inhaled steroids may temporarily initially mask the syndrome due to adequate systemic absorption or local airway effects, as the disease progresses they may not have the potency to combat this systemic vasculitis—even systemic steroids are often not enough and other cytotoxic agents are required. Similarly, leukotriene modifiers may initially be added in lieu of steroids for treatment of signs of airway obstruction as the disease is not recognised as CSS. To date, all cases in the literature of CSS in association with asthma treatment fulfil one of these scenarios. The coincidental institution of these treatments near the time of worsening of the syndrome does not imply causality; rather, these new drugs seem to be unmasking the syndrome and showing that this disease is not as rare as was once perceived. While not all severe asthma is CSS, physicians should recognise worsening asthma in the setting of increased steroid therapy as a potential heralding sign of CSS and look for other vasculitic sequelae.

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AUTHORS' REPLY We agree with Dr Wechsler that it is possible that our patient's asthma was deteriorating as a sign of incipient Churg-Strauss syndrome (CSS). He is right to remind physicians that CSS is one of several causes of worsening asthma. However, we believe that it is equally important to question whether the development of CSS is causally related to the recent prescription of a relatively new class of drug. Whatever the mechanism, physicians should be aware of the possible risk of CSS associated with the introduction of anti-leukotrienes and other therapies and should report all suspected cases to their national drug surveillance authority.

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Malignant mesothelioma

We wish to suggest a minor correction to the otherwise excellent editorial by Drs Steele and Rudd in your recent issue.[1] The status of the forthcoming British Thoracic Society study of the management of malignant mesothelioma is that this study is being supported by the British Thoracic Society and the pilot study is being assisted by the Clinical Trials Unit of the Medical Research Council. Funding for the work of the Medical Research Council Staff has been obtained through the BTS Scientific Committee and from two independent mesothelioma charities—The June Hancock Mesothelioma Research Fund and the Anthony Farmer Mesothelioma Research Fund.

We have approached the Medical Research Council with a bid for full funding of the final study but the outcome of the application is not yet known.

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1 Steele JPC, Rudd RM. Malignant mesothelioma: predictors of prognosis and clinical trials. *Thorax* 2000;**55**:725–6.

Assisted discharge for patients with exacerbations of COPD

We read with interest the recent papers[1 2] which report the findings of randomised controlled trials of early supported discharge for patients with exacerbations of chronic obstructive pulmonary disease (COPD). Both

found that a proportion of such patients presenting to hospital could be safely cared for at home with respiratory nurse support, without adversely affecting mortality or readmission rates.

A similar service to those described operated for the first time in Sheffield over the winter of 1997/8, supported by government money to ease the demand for beds during the winter. Although this did not involve randomisation, our findings were essentially similar. Unselected patients with exacerbations referred by general practitioners for admission to hospital were reviewed and those fulfilling the British Thoracic Society guidelines[3] were offered home treatment. Over a 4 month period 29 of 118 patients (25%) referred were found to be suitable for supported discharge, and we successfully treated the 22 patients who consented to participate in the scheme. Although this was only a small number of patients, there were no readmissions and no home deaths. The remaining 89 patients required admission because of respiratory complications (21 acidotic, 16 pneumonia, seven both) or coexisting medical conditions (17 cardiovascular, 28 other).

We also found that a proportion of suitable patients (seven of 29) did not want to participate in our home treatment scheme. Some of these simply wanted the reassurance of being in hospital, but two patients declined as they would have lost insurance scheme benefits paid for inpatient treatment.

In summary, our experience supports the findings of Skwarska *et al*[2] that plans for future assisted discharge schemes should be based on an estimated discharge rate of 20–25% of the unselected COPD hospital referrals. We too have found that such patients can be safely treated at home, and that this is acceptable to most patients. We would welcome correspondence from the authors as to whether they encountered problems with non-participation due to insurance schemes and, if so, how they addressed them.

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1 Cotton MM, Bucknall CE, Dagg KD, *et al*. Early discharge for patients with exacerbations of chronic obstructive pulmonary disease: a randomised controlled trial. *Thorax* 2000;**55**:902–6.
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AUTHORS' REPLY We note that Dr Barber and colleagues have used an Acute Respiratory Assessment Service (ARAS) with winter bed money and have had similar results to ours and those of Skwarska *et al* in Edinburgh. We would like to comment on the proportion of patients referred for admission who are likely to be eligible for this model of care. Dr Barber's group found that 25% were suitable for supported discharge. In the Edinburgh study 29% were initially considered suitable for home care and in the study recently published from Liverpool 33% were eligible.[1] However, in our study in Glasgow 42% were considered eligible for early supported discharge and this difference may reflect the time when the patients are assessed. In Edinburgh and Liverpool assessment was done on the same day as the patients were referred, whereas in our study the patients were assessed on the day after admission. Clinical improvement and increase in forced expiratory volume in one second (FEV_1) are maximal in the first 24 hours after admission[2] and this may account for the higher FEV_1 values and also for the greater eligibility for home care in the Glasgow patients. We now believe that the ARAS model is best employed by assessing patients after 24 hours in hospital. This uses nurses' time more effectively and increases the number of patients suitable for home care.

We were interested that two patients in Sheffield did not want to participate in the home treatment scheme because they would have lost insurance scheme benefits. This is not one of the many problems with which we have to cope in the East End of Glasgow.

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1 Davies L, Wilkinson M, Bonner S, *et al*. "Hospital at home" versus hospital care in patients with exacerbations of chronic obstructive pulmonary disease: prospective randomised controlled trial. *BMJ* 2000;**321**:1265–8.
2 Davies L, Angus RM, Calverley PMA. Oral corticosteroids in patients admitted to hospital with exacerbations of chronic obstructive pulmonary disease: a prospective randomised controlled trial. *Lancet* 1999;**354**:456–60.

AUTHOR'S REPLY I am glad to see that the experience from Sheffield on supported discharge is similar to that from other parts of the country, with the exception that the high readmission rate was not apparent in this small number of patients. In our study 2% of patients did not wish to take part in the study for various reasons—often, as with the patients in Sheffield, because they wished the reassurance of being in hospital. However, we had no patient who did not participate because of problems with insurance schemes. This perhaps reflects a difference in health care funding between Scotland and England.

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BOOK REVIEW

Imaging of Diseases of the Chest. 3rd Edition. Peter Armstrong, Alan G Wilson, Paul Dee, David M Hansell. (Pp 1039, hardback; £150.00). UK: Mosby, 2000. ISBN 0 7234 3166 3

This popular radiology text is now in its third edition. The challenge for the authors to contain so much information within just over 1000 pages has been successfully met.

The early chapters on plain radiographs are excellent building blocks for any trainee. The growing dependence on high resolution computed tomography (CT) is reflected in several chapters, but this is not at the expense of the plain radiograph. The value of positron emission tomography and magnetic resonance in problem solving are evaluated. CT pulmonary angiography is presented and compared with radionuclide imaging and invasive angiography.

Chapters are written from the viewpoints of both pathological location and aetiology. Where the less usual pathologies are discussed—for example, immunologically mediated, drug induced, and transplant related problems—or the more esoteric diseases are described, additional valuable clinical comments are given.

This book can be read from cover to cover; the clarity of the writing and the good illustrations help the pages to fly by. A major strength is that it can be used to help solve those perplexing cases. The index is written with a clear problem orientated approach, but do remember that the spellings are American.

This multi-modality, multi-subspecialty imaging reference text has been updated. Intended not just for radiologists, our medical, surgical, and allied professional colleagues would be wise to sequester it in their libraries. The illustrations are beautiful, the text is clear, and the references are weighty.—KP