cross the blood-brain barrier and may be given by inhalation.[5,6]

In the few asthmatic patients whose symptoms are worsened by non-steroidal anti-inflammatory drugs (those sensitive to aspirin) we recommend using paracetamol for mild analgesia; for more severe analgesia opiates are not contraindicated provided that asthma is adequately controlled.

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SIR,—Professor Peter J Barnes and Dr K Fan Chung are to be congratulated on their acceptance of allergy as the cause of most cases of difficult asthma but not on their description of the common allergens. This is an appreciable weakness as the most important therapeutic action is discovering the causative allergens and removing them if possible, as it often is. In our experience allergens in bedding are of major importance among the inhalant allergens, and these receive no mention apart from a brief reference to the house dust mite. Sensitivities to feathers, flock, kapok, rubber, wool, and, occasionally, man made fibres all need consideration. After all, patients spend from a quarter to a third of their lives in close proximity to these potential allergens.

Among foods the common allergens are certainly not "nuts, shellfish, and strawberries." We have seen patients with asthma caused by nuts but never, in many years of practice, asthma caused by shellfish or strawberries. We do not deny that they may occasionally offend, but they cannot be an important contribution to difficult asthma. In our experience eggs, milk, cheese, chocolate, and wheatflour are the foods most commonly implicated in asthma and may account for some cases of difficult asthma.

The differences between Professor Barnes's and Dr Chung's observations and our own cannot depend on differences in the patients observed—the disease could not be so different in patients in London from that in patients in Birmingham and Liverpool—but depend on the way in which food allergens are identified. Ingelfinger *et al* state that the requirements for diagnosing food allergy are a triad of (1) consistent reproduction of symptoms on blind provocation; (2) functional or structural changes in the target organ; and (3) showing an immunological mechanism in the pathogenesis. These criteria, they rightly state, are rarely met. They conclude, therefore, that food allergy is rare. This conclusion is contrary to our own experience. We believe that the Ingelfinger triad gives false results. It is theoretically and deductively based, and we think that the correct criteria are clinically and inductively based. This was established in the 1920s by Rowe, who found that diets based on dermal testing were inadequate and that diets empirically based and excluding common foods, especially wheat and other grains, usually eaten daily and unsuspected by the patients, produced more satisfactory results.[3] And as for being uncommon, the offending foods are perceived as more common the more assiduously and the more correctly they are sought.

Admittedly in some patients identifying the offending foods can be tedious and time consuming. But many cases can be relieved by simple means—for example, when the adolescent girl whose asthma is due to her eating 0·25 kg of chocolate daily stops eating chocolate her asthma disappears. This may not advance theories about the physiological and pathological processes concerned, but the outcome is of profound benefit to the patient, and that is the real object of the practice of medicine. Wilfred Trotter spoke of the importance of the practical art of medicine as well as of the applied science.[4] The practical art of treating asthma seems to have been lost in the search for a scientfic precision that still eludes our grasp.

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Oil fired boilers and body odours

SIR,—I believe that the presence of traces of vanadium in emissions from an oil fired boiler may contribute to body odour, besides those mentioned by Dr A B Shrank as adversely affecting natural secretions.[1] Subclinical poisoning with vanadium may impart a garlicky odour to sweat and saliva similar to that caused by thallium and some other transition elements.[2] Vanadium forms up to 10% of the blood cell pigment in sea squirts and sea cucumbers, the fossilised remains of which account for the presence of vanadium in petroleum products. The percentage of vanadium in the ash from burning crude oils may be as high as 45%.[3]

Vanadium is readily absorbed through the respiratory tract. Many of the reported cases of industrial poisoning by vanadium occur in workers handling petroleum ash and cleaning oil fired boilers.[4] Vanadium poisoning leads to other more serious symptoms including anorexia, diarrhoea, albuminuria, and persistent cough, together with the unforgettable sign of a green tongue.

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Rape and subsequent seroconversion

SIR,—Dr S Murphy and colleagues raise the issue of seroconversion to HIV after rape and of its possible prevention by zidovudine.[1] Of equal importance is the possibility of seroconversion to hepatitis B.

Transmission of hepatitis B after a single sexual exposure to a partner positive for e antigen has been described.[2] Effective prophylaxis exists against hepatitis B and should be seriously considered for certain victims of rape.

Our policy is to offer accelerated vaccination[3] against hepatitis B to all seronegative rape victims when the aggressor is known to be positive for hepatitis B antigen or belongs to an ethnosocial group with a high prevalence of hepatitis B. If the woman is seen within 15 days of the attack she is also offered hepatitis B immune globulin, a strategy known to prevent up to 75% of seroconversion after sexual contact.[4]

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Use of medical record linkage to study readmission rates

SIR,—Mr J Henderson and colleagues suggest that comparable data from other parts of the country would be useful to determine whether there is a geographical variation in readmission rates and in trends over time.[1]

In 1986 we reported readmission rates in Leicestershire Health Authority (acute district catchment 780 000).[2] We have also analysed data from 1985 for patients readmitted within 30 days of discharge (table). Patients were identified by name, date of birth, post code, and gender.

Readmission rates within 30 days of discharge and mortality in readmitted patients in Leicestershire

	No of readmissions per 100 patients (Oxford)	Deaths per 100 readmitted patients
General medicine	6·5 (7·2)	14·3
Geriatric medicine	5·4 (9·7)	26·5
General surgery	4·5 (5·3)	7·1
Gynaecology	4·0 (5·6)	0·8
Urology	5·2	5·1
Cardiology	5·2	5·7

These data can be compared with the readmission rate within 28 days in the Oxford districts. With the exception of geriatric medicine, the rates are similar. We agree that readmission rates are one of the few potential measures of outcome available from routine data and suggest that considering the mortality in readmitted patients is also of interest.

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SIR,—Dr J Henderson and colleagues suggest that data on readmission rates from other parts of the country would be of interest for comparative purposes.[1]

In Newham a study has recently been completed on the possible use of readmission rates as a performance indicator using routinely collected Körner data from three districts in North East