Letters to the Editor

Hepatic abscess and cystic fibrosis

Sir,

In a recent issue of the *Journal*¹ we described two male siblings with cystic fibrosis, whose course was complicated by the development of liver abscesses. At the time, the cause of infection in these patients was unclear, as culture of the material aspirated from the liver abscesses was sterile in both cases. Recently, however, one of these patients (Case 2) developed a further, large abscess in the right lobe of the liver. Culture of the material obtained on transcutaneous drainage of this lesion grew *Pseudomonas cepacia*, an organism with which the patient's sputum had been colonized for several years. In view of this, it is likely that this hepatic abscess resulted from metastatic infection from the patient's lower respiratory tract. Immunological studies, including a nitroblue tetrazolium test (NBT), were again entirely normal. The abscess resolved following a 6-week course of parenteral anti-pseudomonal antibiotics.

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Peritoneal dialysis in a patient with haemophilia and chronic renal failure

Sir,

Although haematuria is a common manifestation of haemostatic abnormality in haemophiliacs, chronic renal failure is seen infrequently.¹ The management of the chronic renal failure may require dialysis. The safety of dialysis in patients with congenital haemophilia and uraemic bleeding diathesis is not well known.

A 27 year old man with severe haemophilia (Factor VIII <1%) was found to have renal failure in March 1987, and was admitted with uraemic symptoms in October 1987. An ultrasound examination of the abdomen showed normal sized kidneys. Serology for human immunodeficiency virus (HIV) (Western blot and ELISA) was positive. Haemodialysis was begun through a subclavian Quinton Mahurkar catheter three times a week under factor VIII coverage. Two weeks later, a peritoneal dialysis (PD) catheter was introduced under general anaesthesia. The patient was transfused factor VIII pre- and post-operatively for about a week. Subsequently the patient underwent thrice weekly PD without factor VIII replacement except on two occasions when the peritoneal fluid return was blood tinged. The patient received factor VIII for a pericardial effusion,

haemarthrosis and tonsillar bleed during this period. The PD catheter had to be replaced twice because of blockage, and after four months the patient was maintained on haemodialysis.

The aetiology of renal failure in our patient is unclear. Renal failure with heavy proteinuria and hypertension has been reported in patients with positive HIV serology.² The incidence of hypertension in haemophiliac patients despite renal abnormalities is comparable to that in the general population.¹ There have been three previous reports^{3–5} on the management of chronic renal failure with haemodialysis in patients with haemophilia. They subsequently underwent renal transplantation. Our experience indicates that PD may be relatively safe; these patients do not require routine factor VIII replacement therapy prior to each procedure. PD also excludes the danger of bleeding through a vascular access site. Since a majority of haemophiliacs are serologically positive for HIV, as was our patient, the risk of exposure of medical personnel may be less in a closed system of peritoneal dialysis. The anticipated complications of local haemorrhage or haematoma formation either in the abdominal wall or intraperitoneally are uncommon with the newer sialastic catheters. Optimization of haemostasis prior to catheter placement is appropriate, but routine administration of factor concentrates before each dialysis procedure is not indicated unless bleeding complications are present.

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The drug treatment of asthma

Sir,

I very much enjoyed reading the Festschrift for Professor Margaret Turner-Warwick, *Postgraduate Medical Journal* 1988, Volume 64, Supplement 4. However I did notice that in Dr Hargreave's article, 'The drug treatment of asthma: how can it be better applied?' Dr Hargreave states that 'The

nebuliser solution of ipratropium contains the preservative, benzyonium chloride, which can trigger airway constriction in some asthmatics¹. I would point out that in the UK ipratropium bromide nebuliser solution contains no preservative or stabilising agent and comes in the form of sterile unit dose vials. This has been the case since June 1987 and on 31st March 1988 the multi-dose ipratropium bromide formulation, which did contain the preservative benzalkonium chloride and EDTA, was discontinued. The reason for this change in the solution was so that the potential risk of preservative-induced paradoxical bronchoconstriction would be eliminated.

I would be most grateful if the opportunity arises, that this distinction between the formulation currently available in Canada and that available in the UK could be made in a subsequent issue.

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Porphyria cutanea tarda and human immunodeficiency virus: two new cases

Sir,

Concerning the case presented by Ong *et al.*,¹ we report two more patients with porphyria cutanea tarda (PCT) associated with human immunodeficiency virus (HIV) infection.

Case 1

A 32 year old male with a background of intravenous drug addiction and heavy alcohol intake presented with a 2-year history of multiple blister-like lesions and small scars on the face and the back of his hands, cutaneous hyperpigmentation and facial hypertrichosis. Generalized lymphadenopathy and hepatomegaly were also observed. The most prominent laboratory results were: positive anti-HIV antibodies (ELISA); a lymphocyte count of $1.1 \times 10^9/l$, with 0.16×10^9 CD4 cells; alanine transaminase (SGPT) level of 52 IU/l (normal range: 0–35 IU/l); aspartate transaminase (SGOT) level of 65 IU/l (1–40 IU/l); gamma-glutamyl-transferase (SGGTP) of 117 IU/l (0–30 IU/l); plasma iron concentration was $19.5 \mu\text{mol/l}$ (12–26 $\mu\text{mol/l}$); transferrin, 47 $\mu\text{mol/l}$ (45–70 $\mu\text{mol/l}$); urine uroporphyrin, 343.2 nmol/day (<48 nmol/day); and coproporphyrin in urine, 1578 nmol/day (<300 nmol/day). The patient refused to continue the study.

Case 2

A 33 year old male intravenous drug addict and heavy drinker was hospitalized with fever, asthenia, weight loss, dysphagia and progressive dyspnoea of one month duration. He presented dark skin pigmentation, facial hypertrichosis and scars on face and hands, as well as oral candidiasis and

hepatomegaly. Radiography of the thorax revealed bilateral alveolar infiltrates. Laboratory results were as follows: positive anti-HIV antibodies (ELISA); lymphocyte count of $0.89 \times 10^9/l$; SGOT level of 117 IU/l; SGPT level of 49 IU/l; SGGTP of 26 IU/l; lactate dehydrogenase (LDH) was >667 IU/l (normal range: 120–240 IU/l); serum iron concentration, 2.7 $\mu\text{mol/l}$; transferrin, 27 $\mu\text{mol/l}$; urine uroporphyrin, 836.4 nmol/day; and urine coproporphyrin, 480 nmol/day. In spite of antibiotic treatment, the patient died 20 days after admission secondary to consumption coagulopathy. Necropsy was not authorized.

The association of PCT with HIV infection has been described in four patients,^{1,2} to which we now add two new cases. All six were males belonging to different risk groups (two intravenous drug addicts, three homosexuals and one who admitted to having had sexual contact with a Central African prostitute), and reported varying degrees of alcoholic intake in the past. All developed symptomatic HIV infection (one, persistent generalized lymphadenopathy, and the remaining five, diverse infections included in group IV-C of the present classification of the Center for Disease Control in Atlanta). Four died after varying periods of time following diagnosis of PCT (ranging from 3 weeks to 6 years), one remains alive after 6 months, and we have no information regarding the status of our case 1.

Although it has been suggested that HIV or some associated opportunist agent might alter the porphyrin metabolism, interfering with the function of the cytochrome P-450 dependent mixed-function oxidase system,² this has not been proved. It is also difficult to assess the exact role of alcohol in the clinical expression of PCT in these patients.

We consider that the communication of new cases of this association will help to establish its true incidence and prognostic significance, as well as to stimulate the investigation of possible pathogenic mechanisms.

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Renal and hepatic impairment in association with diclofenac administration

Sir,

We should like to report a case of renal and hepatic impairment following the administration of the non-steroidal anti-inflammatory drug diclofenac.