group have similarly raised incidences of major mental illness.¹

When misdiagnosed affective disorders and unclassifiable psychotic reactions are excluded a substantial excess (confined largely to young Afro-Caribbeans) is left of cases of a disorder which can be described only as classical schizophrenia. Attempts to attribute this disorder to social or psychodynamic causes have a long and distinguished history of failure. The contemporary notions on the cause of this disease were reviewed in 1987,² and about the only point of agreement now is that it is essentially a neurobiological disease. Viewed in this light the speculative explanation which Drs Littlewood and Lipsedge produce seems unlikely. There is no doubt that Caribbeans in this country are subjected to appalling discrimination. While it seems likely that this might give rise to an excess of neurotic and affective disorders, and possibly reactive psychoses, it seems most unlikely that it would explain a sharp increase in the incidence of schizophrenia.

Whether the apparent increase of schizophrenia in Caribbeans is confined to the United Kingdom is an open question. Two studies have attempted to quantify the incidence of the disease in Jamaica.^{3,4} Burke's study showed a sharp peak in incidence in young men, which was not observed in Royes's much more thorough investigation.

Schizophrenia is a devastating disease which is poorly understood. Changes in its frequency in population groups are not unknown.⁵ The existence of a definable group within the United Kingdom with, it appears, about a tenfold excess risk should provide the best opportunity so far to elucidate its cause. It would be tragic if this point were lost among what seems to be premature speculation, and it would do the Caribbean community no service.

GYLES GLOVER

Charing Cross and Westminster Medical School,
London SW1P 2AR

- 1 Glover GR. The pattern of psychiatric admissions of Caribbean-born immigrants in London. *Soc Psychiatry* (in press).
- 2 Murray RM, Lewis SW. Is schizophrenia a neurodevelopmental disorder? *Br Med J* 1987;295:681-2.
- 3 Burke AW. First admissions and planning in Jamaica. *Soc Psychiatry* 1974;9:39-45.
- 4 Royes K. The incidence and features of psychosis in a Caribbean community. *Proceedings of the 3rd World Congress of Psychiatry* 1962;2:1121-5.
- 5 Torrey EF. *Schizophrenia and civilisation*. New York and London: Jason Aronson, 1980.

Delays in treating bladder cancer

SIR,—Mr M J Stower emphasises that a considerable delay still exists in the treatment of bladder cancer in Bristol (30 April, p 1228). Awareness of this problem prompted a change in the way that patients presenting to their general practitioners with macroscopic haematuria were managed in Swansea.¹ Before June 1986 all such patients were seen in clinic before an intravenous urogram was obtained. Then the patient was seen again in clinic with the result before the inevitable admission for cystoscopy was arranged.

A study of 30 consecutive patients showed that the mean time from the onset of symptoms to visiting the general practitioner was 19 days and from the date of the referral letter to cystoscopy 85 days. This was considered to be excessive. From June 1986, instead of being seen initially in clinic, these patients had intravenous urography performed first. Some patients attended the clinic to receive the result while others were admitted for cystoscopy without any clinic visit.

Of the first 30 patients, 20 had a clinic visit after their intravenous urography and the time from the

date of the referral letter to the cystoscopy was reduced to a mean of 67 days. For the 10 patients who had no clinic visit the corresponding time was 52 days.

The three year survival of patients with bladder cancer falls from 60% to 25% if treatment is delayed for more than four weeks,² so even a delay of 52 days is excessive and unacceptable. We agree with Mr Stower that efforts must be directed at the earlier diagnosis and treatment of patients with bladder cancer. We did not encounter any problems as a result of abolishing clinic visits. In no patient did a detailed history or physical examination alter the choice of intravenous urography as the initial investigation. We therefore urge that clinic visits should be abolished in such patients. Our results suggest that this will save time and resources without any obvious disadvantage to the patient.

MEL JONES

Royal Liverpool Children's Hospital,
Liverpool L12 2AP

ROBERT COX

Cardiff Royal Infirmary,
Cardiff

KAREN DAVIES

Llanelli General Hospital,
Llanelli

MICHAEL ROSE

Morrison Hospital,
Swansea

- 1 Jones MW, Cox R, Davies KI, Rose MB. The value of the pre-clinic intravenous urogram in the earlier diagnosis of the cause of haematuria. *Br J Urol* (in press).
- 2 Wallace DM, Harris DL. Delay in treating bladder tumours. *Lancet* 1965;iii:332-4.

Improving outpatient clinics

SIR,—The hospital cited in the study by Mr M Duncan and others (30 April, p 1247) is in Islington district, and I must assure your readers of the serious attention accorded by the managers and staff of the health authority to the problems described.

Two important general points emerged from the study. The first is the attention drawn to the levels of pay which the National Health Service is constrained to offer to its clerical and secretarial staff and the difficulties they impose, particularly in urban areas where similar jobs with higher pay are easily available. This should not be forgotten, even in the light of the welcome conclusions of the pay review bodies for nursing and other staff.

The second, more general, point is the key role of the administrative and clerical staff in the smooth functioning of clinical services. In the cause of serving patients people from many different backgrounds are required, and, while they should all perform their roles as effectively as possible, it is wrong to dismiss any of them as being of lesser importance to the process as a whole.

E MOONMAN

Islington Health Authority,
Whittington Hospital,
London N19 5NF

Iron and infection

SIR,—The scientific case for the importance of iron in infection is established beyond reasonable doubt.^{1,4} As Professor C Hershko (5 March, p 660) and colleagues point out, however, direct clinical evidence for the involvement of iron in infection in man is often unsatisfactory in that it is relatively scarce and often lacks enough detail to be convincing. Nevertheless, the review omits so much of importance that it gives a false impression.

For example, the authors state that splenectomy increases susceptibility to infection in thalassaemia. There is, however, good evidence that splenectomy per se does not predispose to infection.⁵ What matters is the underlying disease. Eraklis and others state that splenectomy does not enhance infection if performed for trauma, idiopathic thrombocytopenic purpura, extrahepatic portal vein thrombosis, or localised tumours.⁵ In thalassaemia there is a strong tendency for an increased saturation of serum transferrin with iron. This is increased still further by splenectomy, and the transferrin is often fully saturated.⁶

Eraklis and others point out that when splenectomy accompanies hepatitis, cirrhosis, thalassaemia major, or Wiskott-Aldrich's syndrome the risk of infection is high, with a 21% death rate among 97 patients.⁵ While this evidence is circumstantial and does not prove that hyperferritaemia predisposes to bacterial infection in thalassaemia, it does at least establish a *prima facie* case for the involvement of iron, which cannot be ignored.

Similarly, the authors are vague about possible effects of iron sorbitol citrate (Jectofer) on kidney infections. No mention is made of the work of Fletcher and Goldstein, which showed convincingly that iron sorbitol citrate could stimulate the growth of *Escherichia coli* already lodged in the kidney.⁷ Since several kidney pathogens possess factors linking virulence to the acquisition of iron,^{3,4} these systems may play a crucial part in clinical infections.

With regard to oral supplementation, there is no evidence in normal people that small amounts of iron have any effect on the saturation of serum transferrin.⁴ If this did happen, we would probably all suffer from haemochromatosis. Supplementation of patients suffering from underlying disease such as kwashiorkor may, however, have undesirable results, leading to greatly increased susceptibility to infection.⁴ It is important to distinguish between these two possibilities.

Finally, we emphasise that it is the availability of iron for bacteria, and not the total amount present, that is the key to its importance.^{1,4} This is a complex issue, but it is the heart of the matter and if ignored can lead to much confusion.

J J BULLEN
C GILLON WARD

Department of Surgery,
University of Miami,
Jackson Memorial Centre,
Miami, Florida 33101, USA

- 1 Ward CG. Influence of iron on infection. *Am J Surg* 1986;151:291-5.
- 2 Payne JM, Finkelstein RA. The critical role of iron in host-bacterial interactions. *J Clin Invest* 1978;61:1428-40.
- 3 Griffiths E. Adaptation and multiplication of bacteria in host-tissues. *Phil Trans R Soc B* 1983;303:85-96.
- 4 Bullen JJ, Griffiths E, eds. *Iron and infection*. Chichester: John Wiley, 1987.
- 5 Eraklis AJ, Kevy SV, Diamond LK, Gross RE. Hazard of overwhelming infection after splenectomy in childhood. *N Engl J Med* 1967;276:225-9.
- 6 Pootrakul P, Rugkatsakul R, Wasi P. Increased transferrin iron saturation in splenectomised thalassaemic patients. *Br J Haematol* 1980;46:143-5.
- 7 Fletcher J, Goldstein E. The effect of parenteral iron preparations on experimental pyelonephritis. *Br J Exp Pathol* 1970;51:280-5.

Körner, nomenclature, and SNOMED

In relation to his letter (23 April, p 1198) Dr W Dodd has asked us to point out that, in addition to being an honorary fellow on the staff of Edinburgh University in the department of general practice, he also holds the post of managing director to Abies Informatics. His work in the department on nomenclature and classification and his interest in classification preceded this appointment by several years.