a general practice population, and our paper is an estimate of that prevalence. In previous papers we have reported fully the incidence of *Trichomonas vaginalis* and *Candida albicans* infections^{1,2} and summarised the result of three years' work on *Chlamydia trachomatis*. Given that background it seemed appropriate to confine our study to the stated aim.

There is an unfortunate error in our paper. "Anaerobes were cultured with gardnerella in twice as many patients as gardnerella alone" should have read "Anaerobes were cultured with gardnerella in half as many patients as gardnerella alone." The figures are correct in the table but the text was altered while being transferred from our manuscript to the pages of the *BMJ*.

Laboratory doctors have a vested interest in highlighting the inadequacies of side room tests, and Dr Kerr and colleagues are no exceptions. There is evidence that microscopy for "clue cells" on a wet preparation is as reliable as laboratory culture.⁴ The drawback is that many doctors have lost this skill because of the centralisation of even simple laboratory techniques. Doctors can hardly be blamed for wanting to rely on side room tests when the proper laboratory is less than enthusiastic about a genuine clinical problem. We are well on our way to providing a simple, rapid bedside test for the diagnosis of gardnerella infections. We have shown that gardnerella is present in a variety of people, ranging from the completely asymptomatic to patients with severe vaginitis.¹ In addition, we have shown that if gardnerella is present with other pathogens it adds to the pathogenicity. The onus is now on those people who think that gardnerella is not a pathogen to prove that it is a commensal.

Infertility tends to dominate clinical research on genitourinary infections as a measure of severity and outcome. If men had a profuse foul smelling discharge about which little could be done it is likely that the scale of severity would be adjusted. We have no evidence that patients' assessment of vaginal discharge is unreliable, but we do have evidence that women underreport symptoms about which experience has taught them that little can be done. It seems olympian to state that gardnerella is unproved as a pathogen when microbiologists are less than keen to take on the, admittedly laborious, task of looking for it. Our patients will not thank us for an academic dispute about its pathogenicity.

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Fetal intracardiac transfusions

SIR,—Dr Magnus Westgren and others discuss fetal intracardiac transfusions in patients with severe rhesus isoimmunisation (26 March, p 885). While applauding the establishment of this new technique, I was concerned that there was no mention in the paper about what the fetus experienced while having its heart punctured.

The fetuses were at a gestational age when they could feel pain, and I wonder whether 10 mg of morphine and 25 mg of promethazine given intramuscularly to the mother an hour before the transfusion would have been sufficient analgesia. Would there not be a case for giving a general anaesthetic to the mother, and hence to the fetus, before this procedure?

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AUTHOR'S REPLY.—We agree with Dr Heyse-Moore that morphine and promethazine did not result in sufficient analgesia for the fetus during the procedure. Several of the less severely affected fetuses moved when the skin was punctured. In some cases fetal movements have caused the needle tip to become dislocated, which is a potentially dangerous complication during an intracardiac transfusion.

General anaesthesia might be one way to solve this problem, but we think that giving pancuronium bromide to the fetus to produce muscular paralysis might be a better way to solve this problem.^{1,2} What the fetus experiences mentally during the procedure is beyond our knowledge, but the puncture of the heart will most probably not differ from any other puncture of the fetal body in respect to pain sensation.

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The sickening of medical research

SIR,—Dr Richard Smith bemoans the state of medical research in Britain (16 April, p 1079). In an earlier paper (6 February, p 409) he stated that the United States far outstripped any other country in the number of citations that articles published there received in other journals. In addition to the sheer volume of scientific output in the United States there is another very irritating factor that we think should be mentioned. This is the intensely parochial attitude exhibited by many American authors, who seem neither to read nor to quote European papers, even when these have been original articles or large clinical series.

A typical example appears this year in *Radiology*.¹ Two American authors describe a sheathed needle technique for liver biopsy in high risk patients. They report using the method between 1984 and 1987 in 22 patients but make no mention of a publication in the *Lancet* in which six British authors had previously described the same technique in a similar number of patients.²

This is not an isolated example and this practice results in a high citation rate for the United States compared with other countries. If American authors continue to tend to cite only papers by other American authors appearing in American journals while British (and European) authors cite good papers regardless of their country of origin or the journal in which they are published it seems inevitable that the reported discrepancy between the citation performances will continue to grow.

Another frustrating practice experienced by

many British authors as well as ourselves is that one can submit an apparently sound article to an American journal, have it rejected, and then see, relatively soon after, an article in the same journal on an identical subject which (often) refers to a smaller number of patients or experiments, comes to the same conclusion as one's own rejected paper, is (to the subjective eye) less well constructed and written, and, of course, comes from an institution in the United States.

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Penicillamine nephropathy

SIR,—Dr C L Hall and others (16 April, p 1083) mentioned the incomplete association between the risk of toxicity with penicillamine and the possession of the histocompatibility antigens HLA-B8 and HLA-DR3 but made no reference to a second recognised risk factor.

In a study of 66 patients with rheumatoid arthritis we showed that those who had an impaired sulphoxidation ability (measured by examining urine for metabolites after oral dosing with a probe drug) had a significantly increased risk of toxicity, including proteinuria, after treatment with penicillamine.¹ There was a 25-fold increased risk of toxicity for patients possessing either HLA-DR3 or poor sulphoxidation compared with those possessing neither.

The importance of impaired sulphoxidation has been confirmed by others, and it is also important in patients with rheumatoid arthritis who develop toxicity after treatment with gold.² The mechanism for the association remains speculative but may relate to altered metabolism of the thiol moiety in these drugs. The ability to predict, and hence avoid, side effects, especially if the tests can be simplified, offers a rare opportunity for patients in whom the risk of toxicity is high.

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Psychiatric illness among British Afro-Caribbeans

SIR,—Drs Roland Littlewood and Maurice Lipsedge (2 April, p 950) go beyond the available evidence in suggesting that British Afro-Caribbeans are at greater risk of schizophrenia than those born in the Caribbean. Neither of the studies they cite addresses this question. Both studies show that younger Afro-Caribbeans, most of whom are British born, seem to be at higher risk. My own study relating solely to Caribbean born migrants shows that the younger members of this

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.

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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.

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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.

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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.

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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.