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