11 cases, initial Gram stain of the dialysate identified a fungus, indicating the value of this test for diagnosis. This finding is of further importance because it takes two to three days for a fungus to grow on culture. Nine of the 11 patients were treated with continuous peritoneal lavage with dialysate containing either amphotericin B (5 mg/l), or 5-fluorocytosine (50-1000 mg/l), or miconazole (20 mg/l). In seven of these nine patients this treatment proved unsuccessful and the infection was controlled only after the removal of the permanent peritoneal catheter. It is of interest that three of these patients (one with *Candida* and two with *Fusarium* peritonitis) were not given any systemic antifungal agent after the catheter had been removed. Five patients were able to continue or recommence chronic peritoneal dialysis following re-implantation of a new catheter after a period of two to eight weeks, during which time they were maintained on haemodialysis. Four patients were transferred to haemodialysis and one subsequently received a successful kidney transplant. Two patients who had bowel perforation—that is, simultaneous infection with a fungus and another enteric organism such as *Bacteroides*, *Pseudomonas*, or *Proteus*—died.

Our experience indicates that removal of the permanent peritoneal catheter and temporary discontinuation of peritoneal dialysis are very important steps in the management of fungal peritonitis. Its prognosis is favourable so long as it is not a manifestation of faecal peritonitis.

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SIR,—In your leading article "Treating fungal infections" (8 March, p 668) the comment was made that the imidazoles require further systemic evaluation and that the efficacy of miconazole given parenterally is still unclear.

With regard to the latter point, the results of series of patients treated with miconazole intravenous solution for coccidioidomycosis,¹ oesophageal candidiasis,² chronic mucocutaneous candidiasis,³ and fungal meningitis⁴ indicate both the efficacy and the relatively low toxicity of this agent.⁵ A recent publication⁶ which evaluated miconazole intravenous solution in systemic candidiasis arising in 37 patients receiving therapy for advanced malignancy suggested that eradication of the fungus is achieved at a rate comparable to that observed with amphotericin, and overall the drug was well tolerated.

Janssen Pharmaceutical Ltd has recently completed a one-year period of postmarketing surveillance designed to monitor the efficacy and safety of miconazole intravenous solution in routine use. A total of 89 case record forms have been received and the scheme is being continued. Although patients were often seriously ill with severe underlying disease, the fungus was eliminated in over half of the cases of proved systemic candidiasis, in agreement with the findings of other workers.⁶ Patients whose infection did not respond were often treated late in the course of their disease, underlining the point made about problems in diagnosis, or did not show objective evidence of systemic fungal infection before treatment

was begun. As with other reports on this compound, no renal or hepatic toxicity was observed and the drug was reported to be "well tolerated" in most patients.

Although we agree that intravenous miconazole is a relatively new agent, we believe that a considerable body of evidence of its efficacy has accumulated that is sufficient to recommend its use in many instances where the toxicity associated with amphotericin precludes its use. This is particularly important in the less specialised hospital unit where sophisticated renal and hepatic monitoring may be less easily achieved.

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Fasting blood glucose concentrations and treatment of maturity-onset diabetes

SIR,—Dr R B Paisey and colleagues (1 March, p 596) report close correlations between the glucose concentrations in fasting capillary blood samples of maturity-onset diabetics collected at home and seven blood glucose concentrations taken during the day. They comment that it might be reasonable to monitor diabetes control by measurement of either fasting or mid-morning blood glucose values. Because of the feasibility of treating maturity-onset diabetics with sulphonylureas aiming to lower the fasting plasma glucose to normal,¹ and the relevance of this index of control,² we have studied the precision of measurement of fasting blood glucose concentrations of maturity-onset diabetics at home and in general practice clinics.³

The mean fasting plasma glucose of all 84 maturity-onset diabetics from three general practices was 8.1 mmol/l, and the day-to-day repeatability of home samples (either with stored Reflotest strips in desiccant bottles or blood samples in vacuum collector bottles) was similar to that of venous samples measured in the clinic ($\pm 1SD$, 0.8 mmol/l (14.4 mg/100 ml)).³ This small variability suggested that it would be feasible to aim to lower the fasting blood glucose to under 6 mmol/l (108 mg/100 ml). This was attempted in 71 of the patients studied over six months, and whereas initially 29 (41%) patients had a fasting plasma glucose of under 6 mmol/l, by increasing the chlorpropamide dose and by dietary advice the fasting plasma glucose was reduced to under 6 mmol/l in 54 (76%) patients.⁴ No episodes of hypoglycaemia were induced by increasing the sulphonylurea dose (and in the six-month follow-up only two patients had hypoglycaemic episodes—both induced by losing over 8 kg weight, because of strict dieting and disseminated cancer respectively). Forty-one of the 54 patients whose fasting plasma glucose fell below 6 mmol/l maintained this degree of control, as determined by repeat fasting blood glucose concentrations in the general practice clinic three and six months later. There are now nine general practices in Oxford monitoring the diabetes of their maturity-onset patients by the means of the two criteria of fasting blood glucose concentration (measured with Reflotest on a Refomat meter) and body weight.

Although Dr Paisey and his colleagues reported that the haemoglobin A₁ concentrations in their

patients were normal if the fasting blood glucose was <7 mmol/l (126 mg/100 ml), we found raised concentrations in many maturity-onset diabetic patients who achieved a fasting blood glucose of under 6 mmol/l (mean haemoglobin A₁ concentrations 8.9%, normal range 6-8%, Bio-Rad columns). This would be apposite in relation to these patients still having a raised fasting blood glucose (mean 5.7 mmol/l (103 mg/100 ml), normal range 3.5-5.5 mmol/l (63-90 mg/100 ml)), as well as sulphonylurea-treated maturity-onset diabetic patients still having raised postprandial glucose concentrations even with entirely normal fasting blood glucose concentrations.¹

Nevertheless, the simplicity of assessing control of maturity-onset diabetic patients by means of the easily understood criterion of the fasting blood glucose concentration provides, together with the body weight, an effective and acceptable mode of monitoring therapy.

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Lithium and the kidney

SIR,—Recent information prompts us to add a postscript to our paper "Renal function after long-term treatment with lithium" (2 June 1979, p 1457) and to emphasise again the importance of keeping plasma lithium concentrations as low as possible.

Possible long-term kidney damage following lithium treatment is invariably discussed in relation to polyuria, considered by some to be inevitable in many lithium patients. Widespread changes in renal function are not seen in our population of 120 patients, who have received lithium for up to 12 years.^{1,2} The incidence of so-called polyuria (<3½ litres/day) was 5% (six patients); in some centres the incidence is up to 40%. Many patients with renal histopathological changes³ had suffered previous episodes of acute lithium toxicity, which is virtually unknown in our patients.

There is no strong evidence confirming that polyuria and renal histopathology are related by common mechanisms. Polyuria may be due to many factors. Early fears of toxic effects of lithium led physicians to insist on increased fluid intake during lithium therapy. The habit so formed, or lack of counter-instruction, would result in resetting of thirst mechanisms³ and to permanent overhydration. Similarly the taste of lithium, secreted in saliva, might be a stimulus to drink in order to remove the taste. The histological changes reported during long-term lithium therapy might be due to acute nephrotoxicity during discrete episodes of lithium intoxication. Alternatively, histological change might result from long-term polypharmacy, common in patients with chronic psychiatric conditions.

We have stressed¹ that the lithium concentrations used in many biochemical studies in vitro were very high (compared with a normal plasma lithium concentration of 1 mmol/l (0.7 mg/100 ml)). However, our Scandinavian colleagues have shown that peak urinary lithium concentrations exceeding 65 mmol/l (45 mg/100 ml) occur in their patients during