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 dessicant bottles or blood samples in vacuum collector bottles) was similar to that of venous samples measured in the clinic (±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 Reflomat meter) and body weight.

Although Dr Paisey and his colleagues reported that the haemoglobin A_1 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 A_1 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 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 changes[2] 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 mechanisms[3] 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 stressed[1] 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