

mas is extremely successful⁵ and its role in the management of these patients should be re-evaluated.

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*.*The authors reply below.—Ed, *BMJ*.

SIR,—We are grateful for the opportunity offered by the letter from Professor Hall and his colleagues to elaborate on some of the points that we alluded to in our review article on prolactinomas. We also feel that Professor Hall and colleagues may have misconstrued some of our treatment stratagems. We argued not that surgery, radiotherapy, and medical treatment were mutually exclusive forms of therapy but that they were usually complementary approaches to the patient with a prolactinoma. We have argued that external radiotherapy, delivered by a linear accelerator, has been effective in lowering serum prolactin concentrations in all patients with prolactinomas but we currently reserve its use for patients with microadenomas.¹ Although to date a normal serum prolactin concentration has been obtained in only one third of patients, the fall has been progressive in all and the number with a normal prolactin value will progressively increase. In most series the cure rate for surgery in macroadenomas is 50% or less. However, we did emphasise in our review that an alternative approach to the patient with a large prolactinoma is to administer bromocriptine to induce pregnancy and then to reintroduce bromocriptine during pregnancy should there be evidence of tumour expansion.

For the patient with a prolactin secreting microadenoma our current advice is to try dopamine agonist therapy, such as bromocriptine, in the first instance. Only in patients who are either intolerant of, or resistant to, dopamine agonist therapy is surgery then advised. Our enthusiasm for surgery has certainly been tempered by the results from Hardy and his colleagues showing that their high initial success rate is followed by a 50% recurrence rate by five years.² We indeed await with interest other largescale long term studies of recurrence rates after transphenoidal removal of microprolactinomas. In the most recent abstract published by Professor Hall and his colleagues (presentation 36 at British Endocrine Societies' 4th Joint Meeting, March 1985, Oxford), out of 29 patients the number of surgically cured patients followed up for more than two years was two. We do not therefore feel that, on present data, transphenoidal surgery should be the first line treatment of choice for small prolactinomas. Nevertheless, for the optimum treatment of all patients with

prolactinomas we believe that all modalities of treatment should be available.

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Convulsions associated with cyclosporin A in transplant recipients

SIR,—Like Dr M L P Gross and others (16 February, p 555) we feel that cyclosporin A alone may not have been the major causative factor in the patients of Dr M Beaman and others (12 January, p 139), who suffered convulsions while having high serum cyclosporin A concentrations. Six patients aged 14-55 years (5 women) have had convulsions after orthotopic live transplant operations in the Cambridge/King's College Hospital series since July 1982 (four have been reported on previously).¹

These occurred one to four months after transplantation while the patients were receiving cyclosporin A. In four patients the convulsions were preceded by a recent change in immunosuppression, although this was for rejection in only one patient. The whole blood cyclosporin A values in all these patients were within the acceptable range of 250-750 mg/l. One patient had a history of convulsions associated with peritonitis and on this occasion her convulsions occurred within three days of the development of a Gram negative septicemia. Likewise, only one patient had a history of renal impairment or hypertension. She had had a mild mesangiocapillary glomerulonephritis before liver transplantation and at the time of the convulsions her serum creatinine was 128 μmol/l (1.5 mg/100 ml) and her blood pressure was 170/100 mm Hg. Her convulsions were initially controlled with phenytoin, but two weeks later she developed chickenpox and was treated with intravenous acyclovir. Shortly afterwards her convulsions recurred and progressed to status epilepticus, her blood pressure at this time being 160/120 mm Hg. Examination of the cerebrospinal fluid showed no evidence of an infective cause. The convulsions were eventually controlled with intravenous diazepam and phenytoin, and she made an uneventful neurological recovery.

There was thus little to support the concept of "rejection encephalopathy" in our patients, with only one patient having evidence of graft rejection. Although renal impairment and hypertension were important features in young patients having convulsions after bone marrow transplantation and treatment with cyclosporin A,² only one of our patients had evidence of this. Unlike the patients reported by Dr Beaman and colleagues, all our patients had cyclosporin A blood concentrations within the acceptable range at the time of their convulsions, and on the few occasions when their drug concentrations were high they did not have further fits. Hypomagnesaemia has been reported to be associated with cyclosporin neurotoxicity,³ but there was nothing to suggest that this was present in our patients at the time of their convulsions, and none subsequently required magnesium supplementation. Of possible importance is the fact that five of our six patients were being treated with corticosteroids concurrently and these could interact with cyclosporin A in several ways. Both drugs cause fluid retention, which may

increase the risks of epilepsy. In addition, tissue concentrations of cyclosporin A might be increased by competitive inhibition of hepatic cyclosporin A metabolism⁴ and possible displacement of cyclosporin A from binding sites within the central nervous system by corticosteroids.

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*.*This correspondence is now closed.—Ed, *BMJ*.

Misuse of published reports in propaganda

SIR,—It seems that some members of parliament do not feel obliged to read published articles in full or feel justified in abstracting only those conclusions which can be bent to make a particular case, disregarding those that contradict it. I refer to the use made of a recent article by myself and my colleagues entitled "Prescribing: the power to set limits" (9 February, p 450) by the Conservative member for Hazel Grove, Mr Tom Arnold. In copious correspondence with his constituents Mr Arnold has quoted our article as confirmation of his own support for the government's proposed limited drug list. Mr Arnold also put the following question to the House (20 February): "To ask the Secretary of State for Social Services, if he will make a statement on the conclusions of the article 'Prescribing: the power to set limits,' by J M Harding and others in the *British Medical Journal* of 9 February," a copy of which he had received. In a written answer Mr Kenneth Clarke replied: "The article referred to by my Hon Friend concludes that: generic prescribing and a limited list of drugs may improve the quality of prescribing and be the only way to curb prescribing costs. We fully agree with these conclusions." (25 February.)

What do Mr Clarke and Mr Arnold think of our conclusions set in context? "Generic prescribing and a limited list for all drugs (not just the categories dealt with in the DHSS proposals) may improve the quality of prescribing and be the only way to curb prescription costs, which are inflated by some drug companies. But a restricted list must be flexible and responsive to patients' needs, and there should be a mechanism for consumer feedback. It should apply to all prescribing, as a division between private and NHS prescriptions furthers a two class system of health care in which the NHS may be seen as second class. Certain investigations and treatments are already influenced by ability to pay. If certain drugs are not suitable for prescribing on the NHS perhaps they are not suitable for all."

It should be clear to everyone reading our paper in full that we oppose the government's initial limited and subsequent expanded list of drugs on the grounds that they will "limit the