planned reductions of 15 647 beds by 1996. Day hospital places are expected to rise by 6499 over the same period to a total of 34 231. Wide Regional differences in day hospital services are expected to persist: there is a five-fold difference between the 828 places planned by the Northern Region and the 4116 in the North Western Region.

The results of this survey demonstrate a continuation of the long-standing decrease in the number of psychiatric in-patient beds. No acceleration in the rate of discharge of patients in England is expected over the next decade: it will remain about 2300 patients per year. The present total number of both in-patient and day-patient places is 89 126. These results suggest an estimated 78 400 total places after the planned closures: a shortfall of approximately 10 000 places from the current level of service provided by the Regions. The estimated 54 140 in-patient places after closures remains over 6000 places short of the long-standing government target of 47 900 (HMSO, 1984). The Audit Commission (1986) found that Health Authorities have been more successful in planning hospital closures than in implementing successor services. These figures suggest that this will continue to hold throughout the next 5–10 years. Given this, local government authorities may be expected to play an increasingly active role in providing for deinstitutionalised patients (Griffiths, 1988).

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Therapy-Resistant Depression

SIR: We read Professor Leonard's article (*Journal*, April 1988, **152**, 453–459) on the biochemistry of resistant depression with interest. We would like to ask him how his serotonergic hypothesis of resistant depression explains certain experimental findings that are at variance. Most antidepressants enhance electrophysiological responsiveness of cells to iontophoretically applied 5HT (de Montigny & Aghajanian, 1978), yet this is in conflict with receptor binding and behavioural evidence for down-regulation of 5HT function following antidepressant therapy (Peroutka & Snyder, 1980; Goodwin *et al*, 1984). Neither is it explained why ECT would appear to have to have the opposite effect to antidepressants by increasing 5HT mediated behaviour and $5HT_2$ receptor binding (Green *et al*, 1983). It would thus appear that, as yet, no one hypothesis can link together the various mechanisms of actions of the antidepressant therapies on 5HT function.

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SIR: While I agree entirely with the views of Drs O'Shea and Mathews that no one hypothesis can link the various mechanisms of action of antidepressants to changes in 5HT function, I feel that their letter ignores the fact that the healthy, genetically pure laboratory rat differs from a depressed patient. The apparent differences between the biochemical and electrophysiological changes initiated by antidepressants and ECT in rat brain would not appear to apply to the depressed patient. In my annotation, I commented on the similarity of action of antidepressants and ECT on platelet 5HT transport in depressed patients. Thus all antidepressants so far examined normalised the decreased $5HT_2$ receptor function (as shown by reduced platelet aggregation) in those patients responding to treatment; qualitatively similar changes occur in ^{3}H-5HT uptake into platelets from these patients. Such findings suggest that there is a 5HT sub-normality in depression