agents, e.g. dopamine agonists such as bromocriptine or dantrolene, may be necessary initially.

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SIR: Wilcox & Nasrallah (*Journal*, December 1986, **149**, 782–784) suggest that the reason why catatonia has become something of a rarity is that brain injury in childhood, which the authors postulate as predisposing to catatonia, has declined in frequency.

I work in the field of mental handicap, in which brain damage is considered, in many instances, to be at the root of the handicap, particularly in profoundly affected patients. I have seen hardly any mentally handicapped individuals suffering from catatonia, although only recently I have been treating someone with this condition. If my experience is shared by other psychiatrists practising mental handicap, this would be an argument against the hypothesis that an organic brain condition is a forerunner of catatonia, unless one were to say that in the mentally handicapped catatonia, for reasons to do with the level of intelligence, is not a feature.

A survey of the incidence of catatonia in the mentally handicapped would throw light on the attractive theory put forward by Wilcox & Nasrallah.

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## A Case of Resistant Schizophrenia

SIR: I wish to comment on the treatment of this unfortunate young man (Mr A) (*Journal*, December 1986, **149**, 789–793). The record of therapeutic failure includes a trial of electroconvulsive therapy: "He was treated with six ECT for a possible affective component to his disorder, but with little benefit." Such a course of electroconvulsive therapy was probably inadequate.

Prior to the introduction of neuroleptic drugs, ECT was commonly used in the treatment of patients with schizophrenia. The comparative studies of the 1960s did not demonstrate a failure of ECT; rather, they demonstrated the equivalence of group results between those patients treated with ECT and those with neuroleptic drugs. The ease of administration, lesser expense, and assumed greater safety of psychotropic drugs, however, led to their replacement of ECT. Recent concerns about tardive dyskinesia, neuroleptic malignant syndrome, and other hazards of neuroleptic drug use (as well as therapeutic failures) have led some clinicians to re-examine the application of ECT in the treatment of schizophrenia.

Friedel (1986) recently reported the successful use of the combination of thiothixene and ECT in eight of nine patients who were non-responders to extended courses of neuroleptic drugs. We have treated nine schizophrenic patients who were neuroleptic and multi-drug treatment failures with the combination of ECT and fluphenazine. Of these, seven have been functioning well in the community for at least one year, and we are encouraged enough to undertake a random assignment trial. However, these patients required an average of 15 ECT in their treatment course, a number greater than is ordinarily given to our depressed patients.

The report by Brandon *et al* (1985) of the results of schizophrenic patients treated in the Leicestershire study and that of Taylor & Fleminger (1980) also encourage the use of ECT in schizophrenic patients. These findings were recently endorsed by the NIH Consensus Conference (1985) and by van Valkenberg & Clayton (1985).

If Mr A is still psychotic, a repeat trial of ECT should be considered with a minimum of 12 induced seizures, preferably with bilateral electrode placement, and with minimum durations of 25 s for each peripheral seizure (30 s central seizure). These treatments should be given while the usual dosages of neuroleptic drug therapy are continued.

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