depot injections, and 10 patients were on a variety of other compounds including tricyclic antidepressants, anxiolytics and fenfluramine. Twelve patients were receiving antiparkinsonian drugs and 8 patients were receiving sedatives at night. The majority of the patients were receiving the drugs three times daily.

We decided to maintain all the patients on a single neuroleptic, haloperidol. This was prescribed in oral doses equivalent to their previous medication calculated on a basis reported by Howard (1976). All patients receiving antiparkinsonian drugs were switched to procyclidine. No other drug was allowed apart from nitrazepam at night which was given if required only. The drug administration was reduced to once daily in 1/3 of the patients and twice daily in the other 2/3.

Prior to the change of medication the patients' psychiatric morbidity was assessed on a Brief Psychiatric Rating Scale (BPRS). The ward sister, who had known the patients for many years, was asked to rate their behaviour on Wing's ward behaviour scale. Extrapyramidal side-effects were rated on a modified extrapyramidal rating scale (Okasha and Hirsch, unpublished). Each patient was asked to complete a standardized side-effects checklist, with the help of nursing staff if necessary. All the patients continued with the usual activities including occupational and industrial therapy.

Six months later all the clinical ratings were repeated.

The results are shown in the table (see pp. 224). There was no significant difference in the BPRS nor on the extrapyramidal symptoms (EPS). Two patients were withdrawn from the antiparkinsonian drug during the trial but this was added in 3 patients. The nurses' rating scores showed a significant decrease, as did the patients' complaints of side-effects.

Caution must always be used in interpreting the results of an open study. However, although the trial was initially viewed with some anxiety by the nursing staff, it was soon welcomed as it was found that patients could be maintained at the same level or better on a single medication given once or twice daily. It was felt that there was an increase in drug compliance by patients and also an increase in the amount of time the nurses could spend with the patients in other activities. Our experience supports the report (Dimitriou *et al*, 1977) of beneficial results of single dose regime. The 50 per cent decrease in side-effects reported in patients is highly significant. This indicates that limiting the treatment to one drug alone renders the therapeutic regime more acceptable to patients. We therefore believe that it is usually of little value to give patients more than one type of neuroleptic. Probably the most important variable is the dosage of the neuroleptic which should be carefully adjusted.

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SODIUM VALPROATE AND TARDIVE DYSKINESIA

Dear Sir,

Since A. C. Gibson (*Journal*, July 1978, **133**, 82) has been unable to replicate our encouraging findings concerning the efficacy of sodium valproate in the treatment of tardive dyskinesia, we would like to point out a few salient points in our experimental design:

1. A half of our subjects were over 65 years of age. This combined with the high dose of sodium valproate used by us led to relatively high blood levels of the drug.
2. The offending and 'high potency' neuroleptics were avoided and our patients were treated with 'low potency' neuroleptics, such as chlorpromazine, which are known to have effects on many other transmitter systems in addition to dopamine. At the pharmacokinetic level these drugs are known to increase blood levels of many other therapeutic agents by inhibiting their metabolism. When used in combination with 'high potency' neuroleptics sodium valproate seems to be ineffective in the treatment of tardive dyskinesia. We have recently documented that sodium valproate does not enhance the efficacy of pimozide in the treatment of tardive dyskinesia.

We think that differences in experimental design between Gibson's and our work explain the discrepant findings.

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Table to Barton and Snaith letter on p. 220

TABLE

Results of active treatment following conclusion of the two-week trial period

Real ECT			Simulated ECT		
Outcome at end of 2-week trial	Number of further ECT	Outcome after further ECT	Outcome at end of 2-week trial	Number of ECT	Outcome after active ECT
+++	3	+++	++	4	+++
+	7	+++	+	7	+++
+	5	+++	0	7	++
+	6	++	0	5	+++
+	5	+	0	5	+++
0	5	+++			

Outcomes on Hamilton ratings:
+++ Excellent ++ Good + Poor 0 None

Table to Rao and Coppen letter on p. 223

TABLE

Clinical state and unwanted side-effects before and after stabilization on haloperidol

		Baseline (on multipharmacy)		Haloperidol only	
Rating scale	n	mean	S.E.	mean	S.E.
Brief psychiatric rating scale	27	7.25	1.26	9.66	1.72
Wing's ward behaviour scale	30	6.2	0.67	4.93*	0.71
Extrapyramidal symptoms rating scale	28	9.6	1.38	10.5	1.61
Side-effects checklist	27	6.37	0.78	3.07*	0.49

* Less than baseline, $p < 0.001$

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