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DRUG SIDE-EFFECTS AND BRAIN DAMAGE

DEAR SIR,

Neuroleptic drugs induce extrapyramidal disturbances which mimic Parkinson's disease, but this is not taken to mean that they can cause Parkinson's disease: a patient whose Parkinsonism continues long after neuroleptic withdrawal is assumed to have been suffering from incipient Parkinson's disease. Involuntary movement disorders are another group of extrapyramidal disturbances and, *a priori*, it would hardly be surprising if neuroleptics induced conditions mimicking these. Such extrapyramidal disturbances would likewise be expected to be reversible on drug withdrawal; if the disorder persisted, the directly analogous inference to be drawn is that an underlying disease process was responsible.

For several years psychological (Johnstone *et al*, 1981; Owens and Johnstone, 1980) and radiological (Weinberger *et al*, 1979) evidence has been accumulating of organic brain damage in chronic schizophrenics unrelated to drug treatment, and further studies (Kleinman, 1981; Andreasen, 1981; Owens, 1981), described at the recent Annual Meeting of the Royal College of Psychiatrists, indicate an association between brain damage, irreversible movement disorder, and negative symptoms in chronic schizophrenic patients. It is thus becoming increasingly apparent that the involuntary movement disorders seen in neuroleptic-treated schizophrenic patients may be categorized as either drug-induced, if drug withdrawal is followed by their disappearance, or as the result of structural brain disease with or without superimposed drug-induced effects, if the movements persist following drug withdrawal.

The term 'tardive dyskinesia' should be reserved for those cases in whom irreversible movement disorders occur late in the course of the schizophrenic or other brain disease processes.

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SUICIDE IN FAMILIES: DRAW A LIFE-CHART

DEAR SIR,

The suicidal family described by Dr Khin-Maung-Zaw (*Journal*, July 1981, **139**, 68–69) is of considerable interest as an example of violent suicide and non-fatal deliberate self harm in several members of a family extending over more than one generation and including suicide in identical twins. Whether 'it is at least likely that there was a genetic predisposition to violent suicide in the family' does however still seem to be a matter for conjecture on the basis of the evidence presented in the paper, and the interactional effects of illness events may well have been more important than the author suggests.

To illustrate this, I have tabulated the data along the lines of a Meyer life chart which demonstrates how the suicides seem to have triggered the onset of

depression (single episode or recurrent) in other family members.

It is again a matter of conjecture whether these temporal links signify causal relationships, but the table does illustrate how useful it can be to set out complex clinical data in a way that does full justice to temporal relationships between events. Time spent in completing a Meyerian life chart is so often amply rewarded that such a technique ought to be an integral part of any psychiatric case history.

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TABLE

Preceding suicides in paternal greatgrandfather (cut throat) and maternal grandmother (cut throat)

	Twin 1	Twin 2	Father	Mother	Maternal grandmother
1970	Suicide (shot self) age 21	Depression (In-patient: ECT)		Grief Depression (out-patient: ECT)	
1971				Depression (Out-patient: ECT)	
1972				Depression (Out-patient: ECT)	
1973					
1974					Depression (In-patient) age 74
1975					
1976					
1977					
1978		Depression (Out-patient: ECT)			
1979		July: Depression (In-patient: ECT) Recovered Sept: Married Dec: Depression Suicide (shot self)	Depression (preceded son's suicide)		
1980			Depression: cut wrists and throat (In-patient: ECT)	Depression (Out-patient: antidepressants)	
1981			Continuing In-patient Treatment ↓	Continuing depression ↓	

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