capabilities nor low premorbid IQ. The degree to which expected gains are not realised is likely to be a function of both severity and age at onset of the disease. Deficits could result from either an impairment of the capacity to benefit from learning circumstances, or a reduction in the occurrence of those circumstances, such as through institutionalisation, or both. Strictly speaking, a reduced rate of skill acquisition could be said to come under the umbrella of impairment by disease onset but there are important empirical and theoretical distinctions between this model and notions such as 'resistance' or 'holding-up' of performance. There are also differences between the two formulations when considering useful indicators of premorbid intelligence. Clearly, the contemporary skills assessed must be resistant to pathology but should also reach an early developmental plateau in the general population.

The importance of considering the age at onset of disorders under study is highlighted by the implications of Crawford's conclusion that in psychiatric/psychological research, schizophrenic cases should be matched to controls on the basis of premorbid IQ. If, as many believe, some schizophrenic psychoses are an age-dependent manifestation of a pathological process occurring during early brain development (Jones & Murray, 1991) such a general strategy would leave schizophrenia researchers with neither the bath water nor the baby, although it might be useful in disorders with unequivocal late onset. At present, reading tests such as the NART provide a more accurate assessment of premorbid intellectual function in schizophrenia than can be obtained by many alternative tests. However, we believe that it is unwise to ignore the difficulties associated with its use, either for individuals or for groups of patients, and premature to apply the term premorbid IQ to adjusted NART scores as has occurred in recent publications (Nelson *et al*, 1990; Frith *et al*, 1991).

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Catatonia and not neuroleptic malignant syndrome

SIR: The simplest explanation for the case reported by White (*Journal*, October 1992, **161**, 558–560) is not that the patient experienced separate episodes of catatonia and neuroleptic malignant syndrome (NMS) as claimed, but that the patient had repeated episodes of catatonia only and never experienced NMS. The case description suggests this in three separate ways: (a) the sufficiency of endogenous catatonia and neuroleptic-induced dystonia to explain all findings, (b) the extremely beneficial response to parenteral benzodiazepines, and (c) the much lower exposure to neuroleptics than in cases of NMS.

This 33-year-old woman received only 25 mg/day of chlorpromazine. In comparison, the mean dose of neuroleptic associated with NMS is equivalent to over 1000 mg/day of chlorpromazine, and causative doses as low as 300 mg/day are unusual (Pope *et al*, 1986; Shalev *et al*, 1989). We can attribute the withdrawal, fearfulness, agitation, stupor, autonomic instability, leukocythemia, and elevated muscle tone and enzyme levels before the first admission to catatonic psychosis. The observed dramatic clinical response to intravenous diazepam distinctively demonstrates that the patient suffered from catatonia and not NMS, because only the former (Greenfield *et al*, 1987) is dramatically sensitive to parenteral benzodiazepines. Indeed, response to parenteral lorazepam or intravenous amobarbital or diazepam should distinguish catatonia from NMS, although false negatives are possible.

The presentation of the second episode was neuroleptic-free, and the patient was therefore clearly purely catatonic. While the fever, tachypnoea, tachycardia, diaphoresis, creatine phosphokinase (CPK) elevation, and muscular rigidity that followed the injection of haloperidol (10 mg) appears to be drug-related, it also required the presence of catatonia, as proven by the immediate resolution

with 20 mg of intravenous diazepam. Because catatonia was required for these symptoms, there was no NMS; rather, there was a combination of definite catatonia and neuroleptic effects. In order to demonstrate NMS the authors would have had to observe drug-induced symptoms in the absence of catatonia, which they did not; their assertion of separate episodes of both catatonia and NMS is not only unproven, it is untested. The care of catatonic patients has always been risky, and the clinical state of this patient may have been as unstable as NMS, but that does not make it NMS.

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'Mabi bark tea' consumption and psychosis?

SIR: Hassiotis *et al* (*Journal*, September 1992, **161**, 404–407) suggest that the use of mabi bark may be the precipitating cause of a psychotic illness in a 23-year-old West Indian woman. They cite the temporal link between ingestion of the mabi bark drink and the onset of her psychosis, the lack of a previous personal or family history of mental illness, an identical twin not developing a similar illness, and a biochemical basis that mabi bark causes central dopamine release.

We find the hypothesis untenable. Firstly, our observations in Trinidad and Tobago are not consistent with this. Mabi bark drink (mauby) is very widely used by the Trinidad and Tobago population and has not been found to contribute to the onset of psychotic illness. In a recent analysis of 634 schizophrenic patients, the period prevalence rate was found to be 5.0/1000 population with the disorder being three times more common in African Trinidadians than Indian Trinidadians. The use of mauby was investigated and was not found to be a contributory factor in a single case. Mauby has been drunk here for over a century and continues to be a very popular drink. Their patient's consumption of mauby was described as high but two-thirds of a pint daily for a week can hardly be described as excessive. In addition, for many years it has been served as a cold drink on the psychiatric wards and to date, no reports of its association with psychosis have been made.

In considering the above, it is clear that the association between mabi bark and this girl's psychotic illness is a spurious one and more than likely to be a chance association. There are too many uncontrolled confounding variables. Why implicate mauby? Why not coffee or tea?

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Simultaneous kidney disease and manic–depressive psychosis

SIR: We would like to report the first recorded case of simultaneous transmission of autosomal dominant polycystic kidney disease (ADPKD) and manic–depressive psychosis (MDP).

Case reports. Mrs F (aged 51) had her first episode of brief mental disorder at the age of 25 after the birth of her first child. After the birth of her second child five years later she had a further depressive illness which was treated with medication and electroconvulsive therapy (ECT). At the same time she was diagnosed as suffering from chronic renal failure due to ADPKD. In 1976 she had a very prolonged episode of treatment-resistant depressive illness. She was very depressed in mood, psychotic, and almost stuporose. However, after a long period of illness she recovered completely. She has had several further episodes of a prolonged psychotic depressive illness, particularly in 1989 when she was admitted to hospital for over six months – receiving antidepressants, *L*-tryptophan, lithium, and 24 applications of ECT. She has had lithium augmentation for treatment of her depressive illness since 1978. Since 1985 she has been on peritoneal dialysis. Her maternal grandmother had some kind of kidney disease but died in her 70s of an unknown cause. Her husband is well. They have two children.

Her elder son has had an episode of severe depression of similar magnitude to that experienced by his mother. He first presented at the age of 25 with a six-week history of lethargy and depressed mood. He was treated with fluvoxamine without response. He then became suicidal, taking several overdoses and cutting his wrists. On the day of admission he had been found by his flatmate trying to electrocute himself using wires from an electric lamp. He improved slowly with antidepressants and was transferred to Day Care, without regaining his premorbid state. He relapsed, was readmitted and responded very well to ECT.