that the use of ketoconazole is 'rarely necessary'. Fluconazole is a good substitute for ketoconazole as an antifungal agent but at higher concentrations ketoconazole also has widespread inhibitory effects on the cytochrome P-450 enzymes involved in adrenocortical steroidogenesis (Raven & Hinson, 1996). As these include 17α-hydroxylase and 11βhydroxylase but not 21-hydroxylase, there is no consequent increase in ACTH and resulting 'cortisol escape'. For this reason, ketoconazole is recommended as the drug of choice to decrease excess cortisol production (American Medical Association, 1993). Because of its cortisol-lowering effect, ketoconazole has also been advocated as a treatment for major depression, a disorder associated with hypercortisolaemia.

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Consent to treatment

SIR: We read with interest the recent article by Brabbins et al (1996) in which the authors make a strong argument for clinicians seeking informed consent for neuroleptic treatment in the majority of schizophrenic patients. They conclude that a pro forma could be used to record patient consent which would include a record of information given, benefits and risks discussed, measurement of capacity to consent and the absence of duress.

While agreeing with the argument for pursuing informed consent in this patient group, it is not so clear to us that the use of a formalised pro forma is the best method of recording consent. A recent American Psychiatric Association task force report (1992) recommended that informed consent for neuroleptic treatment should be documented by a progress note rather than by use of consent forms. This was felt to reflect an understanding of informed consent as a process rather than an event. Such thinking is consistent with recommended UK practice, i.e. that 'consent' is the voluntary and continuing permission of the patient to receive a particular treatment (Department of Health, 1993). In a recent survey of 81 British consultant

psychiatrists we asked whether they would consider using a standardised consent form for patients on long-term neuroleptic treatment. Only 31% responded positively despite 73% admitting to concerns in regard to future litigation from patients going on to develop tardive dyskinesia. There is clearly some resistance among British clinicians to the use of formalised consent forms. The obtaining of informed consent for treatment is an ongoing process and should be recorded in such a manner that reflects this process. A single pro forma, however detailed, may therefore be counter-productive.

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SIR: There are many issues involved in the prescribing of medication to those who may be unable to give consent. Some of these have been reviewed recently in the context of schizophrenia (Brabbins et al, 1996). However the problems arising with drug use in patients with dementia have been less well aired. Brabbins et al suggest that clinicians should document attempts to obtain informed consent, but do not answer the question, 'what should we do if this cannot be given?'. In those patients who have psychotic disorders the argument revolves around whether or not to invoke Mental Health legislation. Where patients have severe cognitive impairment informed consent is usually impossible to obtain. Current practice is to give medication without consideration of the legal status of the patient; it is administered unless the patient actively refuses to take it. Yet, there is doubt as to the efficacy of these drugs in managing behaviour related to dementia (Schneider et al, 1990). We feel quite comfortable prescribing drugs which have a definite benefit to such patients e.g. diuretics, antibiotics and cardiac drugs. However, should the use of drugs which frequently benefit the carers or other patients rather than the recipient themselves and have a high incidence of often irreversible side-effects continue in those who are

unable to give consent? Patients have not only the right to refuse treatment, but should also have the right to consent.

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Obstetric complications and schizophrenia

SIR: We read with interest the article by Kendell et al (1996) on the association between obstetric complications and development of schizophrenia later in life. Pre-eclampsia was the obstetric complication with the highest correlation with the development of schizophrenia. We would draw to the authors' attention the interesting observation that pre-eclampsia may, like schizophrenia, be represented in excess during the winter-early spring months (Boyd et al, 1986; Obed et al, 1994). The authors claim that, despite the evidence for a contribution of foetal anoxia in the aetiology of schizophrenia, improved obstetric care in the industrialised countries has not resulted in a fall of the incidence of schizophrenia. Improved obstetric care has indeed provoked a drastic fall in neonatal mortality, but not necessarily a lower incidence of babies surviving with anoxic damage. It is therefore possible that, despite improved obstetric care, the proportion of babies who survive after anoxic damage, and are therefore probably more liable to develop schizophrenia, has not changed significantly. An indirect confirmation to this hypothesis is that, apart from pre-eclampsia, the only other obstetric factor which in Kendell's study predicted a future development of schizophrenia was a longer stay in hospital for the infant.

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Excess mortality of mental illness

SIR: We suggest that the methodology used in Baxter's study (1996) has produced an underestimate of the true excess mortality in a psychiatric population. Excess mortality in mental illness is greatest in young people, early in the course of their illness. A mixed cohort therefore includes many people who have already survived the period of greatest excess mortality. The Danish study of mortality in first episode schizophrenia (Mortensen & Juel, 1993) found an aggregate excess mortality of 3.34, twice the rate of 1.59 found in the Salford study. This may also be an underestimate of the true mortality, as some people die (e.g. from suicide), without being recognised by the mental health services. The second source of error is inherent in the record linkage methodology, which misses deaths that are not recorded on the registers. The size of the error depends on factors such as the accuracy of the registers and the record linkage process, and the frequency of emigration and change of name among the study cohort. We are currently conducting a follow-up study of 360 people with schizophrenia, in which case register information has been verified by other means. We found that 4 of 66 deaths (6%) were not picked up by the initial record linkage. Two were recorded as alive on FHSA databases, and two died abroad.

These comments do not invalidate Baxter's conclusions, but confounding variables such as the demographic characteristics of the cohorts and the length of follow-up will have to be standardised before the results of different studies can be compared.

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