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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More cases of paroxetine withdrawal syndrome

SIR: We wish to add five more cases of paroxetine withdrawal syndrome to those reported by other authors (Barr *et al*, 1994; Pyke, 1995).

All of our cases occurred in young women (aged 26–39 years), without concurrent organic illness, diagnosed with major depression. Paroxetine was started at a dose of 10 mg/day in the first week and increased to 20 mg for the rest of the treatment period (12–14 months). The drug was well tolerated and laboratory tests were always normal throughout the period. A benzodiazepine was used during the first 2–3 months as an adjunct to manage anxiety but paroxetine was, thereafter, the only maintenance treatment.

In three cases paroxetine was discontinued by alternating 20 mg one day and 10 mg the other day during a week. After that, 10 mg/day was maintained for 15 days, then patients were prescribed 10 mg every other day for one more week, before stopping medication. In two patients the tapering was done directly from 20 to 10 mg and after 2 weeks on this dose they stopped the medication.

All patients complained of vertigo, lightheadedness or gait instability during withdrawal. Three patients referred to the symptoms in their next planned out-patient consultation, but two demanded urgent treatment and were prescribed lorazepam 1 mg/day for one week. In all five cases, the symptoms persisted for approximately 7 days.

Very similar withdrawal syndromes have been described with other serotonin selective reuptake inhibitors including fluvoxamine, fluoxetine and sertraline, and muscarinic and serotonergic factors have been implicated in the production of these symptoms but what is striking from a clinical point of view is the fact that a conservative tapering regime was unable to prevent symptoms appearing. As a result of this experience we are now applying a dosage reduction of 5 mg per week in an attempt to avoid this withdrawal syndrome.

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Yohimbine and sinusitis

SIR: There is clinical evidence that yohimbine has an effect in restoring erectile capacity in men with erectile dysfunction (ED) but may be accompanied by side-effects.

Case report

A 59-year-old man described a 3 year history of erectile dysfunction. After discussing treatment options he chose oral medication. A trial of yohimbine 5.4 mg tid. was initiated. Three days after starting medication he developed pain and discomfort above both eyes which he described as like 'a thick head cold'. The area was tender to touch but there were no other symptoms to suggest sinus problems, influenza or an anxiety state. There was no change in his mental state. He stopped medication and symptoms resolved within 24 hours. A week later he re-exposed himself to the tablets and symptoms returned after three days, and again resolved within 24 hours of stopping. During each treatment period, no success was noted with sexual function. The patient refused a third trial of yohimbine with phenylephrine cover.

Yohimbine is an alpha-2-adrenergic antagonist. Sinus congestion is treated with a decongestant agent like phenylephrine, a direct acting alpha agonist which causes peripheral vasoconstriction. It is possible that the described symptoms are a consequence of alpha-2-adrenergic antagonism affecting the sinus mucosa. This may explain the delay in onset of symptoms similar to the latent clinical response of the drug. Side-effects would be expected to occur rapidly given the short plasma half life of 35 minutes (Owen *et al*, 1987). There are no reports in the literature associating yohimbine and sinusitis,