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## Obstetric complications in schizophrenia

SIR: Günther-Genta *et al* (*BJP*, February 1994, **164**, 165–170) found an excess of obstetric complications (OCs) in schizophrenic patients when compared with siblings, normal controls, or other patients. As in most such studies (Lewis, 1989) their findings reveal differences in the main at a low level of statistical significance.

As they point out, their sampling of schizophrenic in-patients leads to a selection bias towards chronicity, and they suggest that the only way of avoiding such bias would be a community study. In our community study (McCreadie *et al*, 1992) we failed to find a difference between schizophrenic patients and their siblings in their history of OCs. Our community of schizophrenic patients contains some who have had fewer admissions and probably a better prognosis.

Günther-Genta *et al* question the validity of studies which rely on maternal recall as the source of information on OCs, but O'Callaghan *et al* (1990) have shown that maternal recall is reliable, and using maternal recall found a rate of definite OCs in their schizophrenic population of 33%, which is comparable to the 45% found in Günther-Genta *et al*'s studies and which is close to the 35% that we found. Günther-Genta *et al* draw attention to the low rates of definite OCs found in Lewis *et al*'s study (1989) (with 'definite' complications in 17% of schizophrenics and 8% of controls); they suggest this reflects a low sensitivity of maternal recall. In fact these figures were drawn from information obtained solely from psychiatric records, which will clearly underestimate the proportion of patients with complications and only indirectly reflect the accuracy or otherwise of maternal recall.

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## Early responses to electroconvulsive therapy

SIR: Rodger *et al* (*BJP*, January 1994, **164**, 106–109) draw attention to the important question of the speed of response to electroconvulsive therapy (ECT). This prompted me to review data from a previous study of ECT and pterin metabolism (Anderson *et al*, 1992). The original protocol required all subjects to be assessed after two ECT applications, although these data were not reported.

Subjects met DSM–III criteria for major depression with melancholia or psychosis (American Psychiatric Association, 1980). ECT was administered twice weekly using bilateral electrode placement and an Ectron 2 Series ECT device. Severity of depression was measured by the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1969) and the Montgomery–Asberg Depression Rating Scale (MADRS; Montgomery & Asberg, 1979), but only

MADRS scores are reported here, as they are more sensitive to change. Response to ECT is defined as MADRS score under 11 at completion of the course.

There were 9 men and 14 women, with mean age of 58.9 years (range 24–76). Nine had a psychosis, and 14 melancholia. There were 13 ECT responders and 10 non-responders. They were a severely ill group with a mean MADRS score on entry of 43.2 (range 28–52). For nine patients, the MADRS score more than halved after two applications of ECT. The reduction of score for the whole sample after two applications was highly significant (paired $t$-test: $t=4.406$, $P<0.001$).

Of the 13 responders, 8 had their score at least halved after two applications of ECT, as did five (56%) with psychosis, but only four (29%) with melancholia and one (10%) non-responder. The change of MADRS score after two ECT applications was significant for each group except melancholics (paired $t$-test: responders, $t=3.509$, $P<0.005$; non-responders, $t=2.67$; $P<0.025$; psychotics, $t=2.98$, $P<0.01$). Of the total reduction in MADRS scores at completion of ECT, 46% occurred after two treatments (45% responders, 46% non-responders, 56% psychosis, 36% melancholia). The mean length of ECT course was 8.7 treatments (range 5 –16).

These results are similar to those of Rodger *et al*, and support their contention that a substantial benefit from ECT is realised after early treatments. This sample was more depressed (mean HRSD score before ECT=33, s.e.m.=1.6), assessments were performed after two, not three treatments, and subjects were not drug free. These factors may contribute to the larger effects found by Rodger *et al*.

Why do some and not others respond early? Does response time distinguish different subtypes of disorder? What are the implications for the biology of depression? As the most potent and consistent antidepressant treatment for severe depression, understanding the mechanism of action and cerebral effects of ECT is of great significance. Clearly, we have much to learn about ECT, but the discrete, definable nature of this treatment lends itself to study. Its secrets may teach us much that we need to know about affective disorders.

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## Electroconvulsive therapy and brain damage

SIR: Some critics still believe that electroconvulsive therapy (ECT) inevitably leads to brain damage and intellectual impairment (Breggin, 1993), although prospective quantitative magnetic resonance imaging has not found any structural changes in the cerebral hemispheres after two or three courses of ECT (Scott *et al*, 1991). There has never been an adequate study of the long-term intellectual effects of ECT because prospective studies have been limited to the assessment of patients before and after only one course of ECT (maximum 12 treatments). We report a prospective assessment of the intellectual effects of 125 treatments given to a patient over seven years; a preliminary report has already appeared in the *BJP* (December 1991, **159**, 867–870).

*Case report*. A 67-year-old catering assistant suffered a recurrence of a depressive illness, and required admission. She was agitated, anxious and depressed, expressing delusions of guilt, and had tried to kill herself. She had suffered five previous episodes of depressive illness that had not responded to antidepressant drugs, but had to ECT. The index episode responded preferentially to bilateral ECT, but relapse was not prevented by antidepressant with neuroleptic drugs in doses she would tolerate. Two years later she agreed to fortnightly bilateral ECT as continuation treatment. This was successful, and she was discharged to a private nursing home. She continued to receive ECT as an out-patient and remained well; the frequency of ECT was reduced to once every three weeks two and a half years later. The baseline intellectual assessments were carried out by an independent clinical psychologist a year before the patient's admission, when she was well and had not received ECT in seven years. The later assessments were carried out by a psychiatrist (AGH) who knew the patient was being treated by ECT, but was unaware of treatment details. The patient was assessed 10 days after the last ECT and was free from depressive symptoms at each visit. Psychotropic drug treatment was standard (10 mg trifluoperazine).

At the baseline assessment she was fully orientated and her mental abilities according to the Clifton Assessment Procedure for the Elderly (CAPE; Pattie & Guillard, 1979) placed her in the category of 'independent elderly' (Table 1). This did not change after 125 ECT treatments. Her