

reduction in ECT-induced seizure duration with this agent (Dwyer *et al.*, 1988) necessarily means that propofol causes a consequent reduction in the efficacy of this treatment. Although this rationale is consistent with widespread clinical wisdom, there has in fact been no scientific substantiation of a minimum necessary seizure length. If anything, recent studies indicate that seizure length is clearly not related to efficacy. For example, Sackeim *et al.* (1987), in a study comparing seizure threshold doses of bilateral and unilateral ECT, found no difference in the mean individual seizure duration despite a significantly better response with the bilateral treatment.

In view of the lack of any data concerning this question, we recently reported a retrospective study reviewing the experience with propofol in a specialised mood disorders unit at Prince Henry Hospital, Sydney (Mitchell *et al.*, 1991). Over a 30 month period, 66 patients with primary depression were treated with ECT. The study period spanned 15 months before and after the introduction of propofol into this hospital. Choice of anaesthetic induction agent was determined by the individual preference of the anaesthetist and not by diagnosis, age, or physical status of the patient. Either thiopentone or propofol was used. The 21-item Hamilton Rating Scale for Depression (HRSD) had been administered prospectively before and 1-2 weeks after ECT to 37 of these patients who were involved in other research projects. These ratings were made without knowledge of the anaesthetic agent used.

The ratio of the mean mg/kg doses of thiopentone and propofol actually administered (2.01) was close to the published equipotency ratio of 1.61. We replicated the previous reports of a reduced seizure duration with propofol, finding a mean of 18.1 with this agent compared with 24.7 with thiopentone ($P < 0.01$). Despite this reduction, we found a highly significant improvement in HRSD ratings in those patients given propofol (mean reduction from 28.2 to 7.5, $P < 0.01$), which was not significantly different to that observed with thiopentone as the anaesthetic agent. We did, however, observe a weak trend suggesting that patients receiving propofol required more treatments per course (13.0 v. 10.8; $P = 0.18$).

Although we are fully aware of the limitations of a retrospective study, our findings suggest that propofol may not, in fact, impair the efficiency of ECT. Prospective studies are necessary to answer the question definitively. As with many issues related to ECT, the discussion about propofol requires more data and less unsubstantiated polemic.

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Clozapine

SIR: We were somewhat surprised by the recent letter concerning the prescribing of clozapine for treatment of psychosis in the community (Phelan *et al.*, *Journal*, September 1992, **161**, 425-426). Firstly, clozapine has *only* been used *outside* research settings in the UK. This is the commonest misconception that bedevils clozapine; that it is a new research drug. It is a licensed drug with known effectiveness which was demonstrated in ordinary psychiatric practice in the 1960s and more recently by one of the most rigorous multicentre, placebo-controlled drug trials performed in the last decade (Kane *et al.*, 1988). Indeed, trials of clozapine have all been performed on clinical samples, not specialised research cohorts. In particular, its efficacy has been documented in subgroups of seriously ill, long-stay in-patients. These patients, resistant to typical antipsychotics, are a massive burden on hospital services and a perpetual risk to themselves and others.

Secondly, the service implications of an 'almost full-time' nurse to monitor blood sampling, and liaise with patients, their families, doctors, and the laboratory would seem extremely cost-effective in comparison with the prolonged and frequent hospital admissions otherwise required, and should not such ill patients have high levels of input in any case? The use of clozapine in our catchment area has allowed at least one patient to be brought out of a private long-stay facility costing £70,000 per year. Of the 2000 patients on clozapine in the UK, 500 are maintained in the community without problems. The monitoring system is specifically designed to prevent fatalities, and the smooth running of clozapine prescribing is the precise function of the

Clozaril Monitoring Service. The suggestion that there is a need to be particularly aware of physical illness is a moot point. Except for haematological problems (which are reversible on drug withdrawal), clozapine has fewer contraindications than other antipsychotics.

There are many situations in which such liaison and (initially) intensive monitoring is commonplace: for example, lithium treatment of bipolar disorder, or physical treatments such as gold injections for severe rheumatoid arthritis.

There are now examples of efficient community/out-patient services which are giving clozapine to large numbers of patients without the need for incarceration in hospital. Two models essentially operate in the UK: a clozapine clinic where patients all attend on a single morning for blood sampling and prescription; or community psychiatric nurses (CPNs) trained to take blood. A single CPN suffices for a large number of patients, and in practice the clinic nurse works 1–2 sessions a week (Launer, 1991).

It is an inescapable fact that the reintroduction of clozapine is one of the most dramatic advances in psychopharmacology since the introduction of phenothiazines in 1957. It would be a pity if overstated economic fears conspired to deny extremely sick patients a chance for recovery which they previously may never have had, and shortsightedly deny catchments the opportunity for making real savings in patient care.

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Multiple personality disorder

SIR: Recent correspondence (Correspondence, *Journal*, September 1992, **161**, 415–420) continues to perpetuate the erroneous notion that multiple

personality disorder (MPD) either does not occur in the UK or is a misdiagnosis of some other condition.

My first encounter with MPD in a clinical setting in the UK occurred without warning some ten years ago in a working class, uneducated, psychologically unsophisticated patient without prior knowledge of the condition. The transformation was so all-encompassing that it transiently made me doubt my own sanity.

Since then I have either personally interviewed, treated, or been consulted about many other cases, both in urban Surrey and in Aberdeen. Why, in that case, does the literature continue to insist that MPD is a peculiarly North American phenomenon?

I believe the answer lies in the uncomfortable relationship between psychotherapy and psychiatry in this country. Many MPD patients have told me that they feared to reveal their condition to psychiatrists, sensing that they would be misunderstood and thought to be schizophrenic. Such is the scepticism of the psychiatric establishment regarding this condition that the fear was perhaps not entirely misplaced. Psychotherapists, whose attitude is, we hope, less judgemental, seem from my observations to be often quite familiar with clinical cases of MPD, through either personal experience or supervision. Professional ridicule and accusations of gullibility await those who are foolish enough to declare an interest in public, or seek to study this fascinating condition.

The much greater integration of psychotherapy into psychiatry in the USA may explain the greater rate of diagnosis, as a non-judgemental 'therapeutic' attitude is a prerequisite for detection of MPD, which can be effectively concealed from external observers for decades.

I suspect that the same judgemental scepticism pervades the review committees of our journals. I have not as yet managed to publish on this topic except through the medical columns of women's magazines whose motives are far from altruistic. I believe that this condition has much to teach us on the structure of personality. At the very least it deserves a fair hearing.

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SIR: Professor Merskey's opinion (*Journal*, March 1992, **160**, 327–340) that the diagnosis of MPD is the very cause of the disorder and does not prove its existence leads to the classical double-bind state: "You're damned if you do and you're damned if