shorter with propofol and an assumption has been made, drawing on studies of seizure duration, that this will inevitably lead to reduced efficacy.

Twenty patients fulfilling DSM–III–R criteria for major depressive disorder were rated using the 17-item Hamilton Rating Scale for Depression (HRSD; Hamilton, 1967) and the Beck Depression Inventory (BDI; Beck, 1961). Mean length of fits (17.5 seconds) and mean total length of fits (118.9 seconds) for the propofol group were significantly different than for the methohexitone group (25.5 and 175.5 seconds respectively; $P<0.05$). We found a significant improvement on both HRSD and BDI ratings in both groups but no difference in the number of treatments needed to give a 60% improvement in ratings. Indeed, there were more treatment failures, defined as those patients who failed to show a 60% improvement over the course of ECT, in the methohexitone group than the propofol group.

ECT studies, when measuring outcome, are fraught with difficulties. It is, nevertheless, crucial that we understand the effects of changes in anaesthetic practice on the treatment we prescribe. It is not sufficient to concentrate on fit duration and current as a measure of the success of ECT. The standard by which any treatment is measured ought to be as close to the desired clinical effect as possible.

We believe our study, which is still being analysed, is the first to compare methohexitone and propofol prospectively in terms of clinical response. There is a continuing need for more research into ECT, using large samples, if we are to shed light on the mechanisms involved.

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Suicide prevention by general practitioners

SIR: Dr MacDonald ends his letter (*Journal*, October 1992, **161**, 574) with a plea: "So please, can we now have a moratorium on this idea that practitioners can prevent suicide?" Such a view cannot be allowed to go unchallenged.

Glancing through the *Oxford Textbook of Medicine* we found the following incidence rates of some common organic disorders: Crohn's disease 7.1, ulcerative colitis 10–12, multiple sclerosis 0.5–9.5 (all per 100 000 population). In 1989 the overall suicide rate throughout England and Wales was 7.4. So why the defeatist attitude about suicide prevention, particularly when we know that the majority of persons who end their lives seek help in the final week of their lives? We have never heard complaints that early detection and treatment of the organic diseases cited above is not feasible because they are so rare.

We accept that prevention of suicidal behaviour will partly depend on social changes which are more the responsibility of politicians that clinicians. There is no doubt, however, that clinicians should have a major role. This includes general practitioners (GPs) as well as members of psychiatric services. The potential preventative role of GPs is supported by the finding of a significantly decreased suicide rate on the Swedish island of Gotland following an educational programme for GPs on the assessment and management of depression and suicidal potential (Rutz *et al*, 1989).

By focusing on suicide prevention we open up crucial aspects of clinical care. The assessment and management of severe mental illness (especially depressive disorder), and of suicide risk itself (both in hospital as well as in the community), are perhaps the most important of these. Effective prevention of suicide is a goal which surely will be achieved as the end result of improvements in clinical techniques relevant to the whole of psychiatric experience. Why not accept that these are in urgent need of attention?

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Psychosis and multiple sclerosis

SIR: The application of sophisticated neuro-imaging technology to the detailed evaluation of patients such as those described by Feinstein *et al* (*Journal*, November 1992, **161**, 680–685) may yield potentially important clues to the aetiology of 'functional' psychoses, and we have recently employed this strategy in the study of schizophrenia (Buckley *et al*,

1993). The putative aetiopathological association of psychosis and multiple sclerosis (MS) is, however, difficult to discern from the multiple confounding factors: the variability of neurological presentation, the frequent existence of substantial cognitive impairment even at the early stages of MS, the use of steroids in treatment, and the marked psychological distress and psychosocial dysfunction which accompany MS.

We have studied the relationship of bipolar affective disorder to MS, and have described seven patients in whom mania appeared as the initial presentation of demyelination (Hutchinson *et al*, 1993). The mean age at onset of psychosis was 29.8 years (range 21–52), which is earlier than that of the patients described by Dr Feinstein *et al*. Affective symptoms antedated the emergence of neurological findings by just under two years in two patients and five years or more in the remaining patients. Subsequent psychotic episodes were unrelated temporally to neurological exacerbations or steroid treatment. A family history of affective illness was noted in only one patient. In contrast to the findings of Feinstein *et al*, we were unable to discern any distinct pattern of white matter lesions evident on magnetic resonance imaging (MRI). We agree with the assertion of Dr Feinstein *et al*, supported by earlier clinical and epidemiological research (Minden & Schiffer, 1990), that an aetiological association exists between the pathological process of MS and psychosis. Some recent findings may suggest a genetic basis to this association (Schiffer *et al*, 1988). The nature of relationship between psychosis and MS merits closer scrutiny both from a nosological perspective and with regard to the effective management of MS patients who exhibit psychotic symptoms.

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Adaptive behaviour scale in Down's syndrome

SIR: We are interested in two particular points in Collacott's article (*Journal*, November 1992, **161**, 675–679): the significant decline in Adaptive Behaviour Scale scores with advancing age in Down's syndrome, and the increased variance of the scores in the older age group.

We have been looking for some time at a possible association between raised mean cell volume (MCV) and cognitive decline in Down's syndrome, previously noted by Hewitt *et al* (1985), and have conducted a small local study to examine this idea further. Sixty-three Down's syndrome subjects from three hospitals were selected from an original group of 113, by excluding patients with known possible causes of macrocytosis (hypothyroidism, anaemia, B12/folate deficiency, and treatment with anticonvulsants). By means of a carer interview all patients were rated on a simple scale (available from the authors) designed to measure functional disability: ability to wash, dress, feed and toilet themselves, etc. The average disability score was then compared between those with normal MCV (less than 96 fl), and those with high MCV (greater than 97 fl). Within the two groups, scores were further divided into those less than 50 years, and those older.

Although the results did not reach significance, there was a trend towards higher MCV subjects being more disabled, this being most pronounced in the older group. The variance of disability scores was also greater for the older group, in keeping with Collacott's findings, and at least partially accounted for by the high/low MCV split. We have speculated in an article soon to be published in the *Journal* that the raised MCV found in Down's syndrome, and the premature appearance of dementia, may follow free radical stress, a process which is probably accelerated in Down's syndrome and which may also be relevant to ageing. It seems possible that from an early age some of these individuals are less able to handle the additional oxidative stress, and it is this group that show earliest intellectual deterioration. The extent of macrocytosis may help to identify these subjects before any measurable cognitive decline has occurred.

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