I agree that confidence intervals are valuable indicators of the precision of a sample estimate and if I were writing the paper today I would include these as a matter of routine. Undoubtedly, a larger sample of patients would have led to narrower confidence intervals and allowed us to test other hypotheses about traditional ECT. As we explained in the paper, this would also have exposed more depressed patients to a treatment known to be associated with more severe confusion, anterograde amnesia and, to a lesser extent, a greater risk of the loss of personal memories.

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Daily living programme

SIR: Muijen *et al*'s preliminary account of an intensive community support scheme as an alternative to prolonged or revolving-door hospital admissions made interesting reading (Muijen *et al*, *Journal*, March 1992, **160**, 379–384). They rightly draw attention to the worrying finding that the study group in the Daily Living Programme included two patients who later killed themselves and another man charged with murder. In assessing such treatment programmes we must, of course, bear in mind that other such controlled studies have not attracted such tragedies and so it would be unwise to draw general conclusions about the worthiness or desirability of such a programme.

However, it would be valuable to have further information regarding these three violent episodes. One wonders about their diagnoses, severity of illness and although contact was 'regular', was it frequent and of what kind was it? How early in the treatment programme did the events occur and if early on, were mean figures added in (to cover for the rest of the treatment phase as if the suicides were still included) on the assumption that the suicides were a tragic, but random event?

While such programmes are in an early phase and before they are generally introduced we need to ensure that such unfortunate events were indeed random (and not dependent upon whether a patient is assigned to a hospital or community treatment group). Any cost-savings of such a community treatment package (if as well run as the Maudsley's) seem to be slight, and the justification may, therefore, rest on qualitative rather than quantitative factors.

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AUTHOR'S REPLY: Dr Green asks about the tragedies in our study. Among our 187 Daily Living Programme (DLP) patients and controls seen for 18 months after trial entry, the DLP had three, and the control group two, deaths from self-harm. The deaths were 4, 5 and 14 months after trial entry; all had had brief in-patient stays. The control deaths were 7 and 12 months after trial entry following admissions of 4 and 3 months' duration. The difference of three versus two deaths does not begin to approach statistical significance. (Further details will appear in our final report.)

Many studies have noted a high suicide rate in psychotic patients, and there is no easy way to reduce this. The risk remains high despite intensive in-patient and/or community care. When monitored, suicides occur surprisingly often even in patients on section and on 24-hour 'special' observation by nurses. There is no evidence as yet that standard in-patient care actually reduces suicide rates in the long-term. Evaluation has to be even-handed between innovative and standard models of care. It rarely is.

The DLP patient's homicide took place more than seven weeks after admission for self-cutting. During five days of in-patient observation he was wary but coherent and rational. Repeated examination found no signs of depression or psychosis. With the ward team's agreement he was discharged into intensive community care, and was seen repeatedly at his home and the DLP base despite his evasiveness. He managed well, and was found a job after having been unemployed for several years. He had not been sectionable at any time. Two days after last being seen he suddenly killed a child. In custody he revealed previously concealed paranoid delusions.

Had the above patient remained an in-patient well beyond the national mean duration for such cases he could have discharged himself, been lost to out-patient follow-up, and become an ex-patient for whom district services could disclaim responsibility. Community care teams like the DLP which rightly do assertive outreach, however, continue their responsibility and thus their vulnerability to blame for adverse events. With whom does the responsibility

https://doi.org/10.1192/S0007125000124699 Published online by Cambridge University Press

lie for tragedies where there has been no outreach, and previous carers may not even know about them?

Conclusions from the three-year study await our final report this year. They will be based on the clinical and social outcomes, users' (patients and relatives) satisfaction, risk assessment, and an economic analysis.

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Enhancement of recovery from psychiatric illness by methylfolate

SIR: We are grateful to Dr Procter (*Journal*, August 1991, **159**, 271–272), Dr Anderson *et al* (*Journal*, January 1992, **160**, 130) and Drs Wing & Lee (*Journal*, May 1992, **160**, 714–715) for their interest in our study reporting enhancement of recovery from psychiatric illness by methylfolate (Godfrey *et al*, 1990). Procter is sufficiently impressed by this and earlier literature to doubt the need for further controlled trials of treatment of folate deficiency in psychiatric patients. Anderson *et al* and Wing & Lee have some reservations about some aspects of the design of our trial, while at the same time stressing the importance of the folate–mental disorder connection and drawing attention to their own interesting contributions to the subject.

To deal first with the design features of our methylfolate trial, we do not agree that it was inappropriate to use the Hamilton Rating Scale for Depression (HRSD) in schizophrenic patients. The HRSD is as valid for depression as a symptom in other psychiatric disorders as it is in depressive illness. Nor did we find the inconsistencies between the HRSD and Beck scores surprising. Many of our patients were psychotic, and self-report in such patients, many of whom are without insight, is bound to be unreliable. The object of our study was to evaluate long-term (six months) clinical outcome, and the use of clinical outcome scales in such a context is well established in psychiatric research. We do not agree that it is difficult to distinguish between residual symptoms and pronounced impairment, or between full social recovery and no or partial social recovery. The absence of baseline scores was dictated by the fact that the point of entry to the trial was the finding of a low or borderline red cell folate level, at which point many of the patients were already on standard medication. We were studying in a placebo-controlled manner the outcome of an adjunct to treatment and we had no wish, nor would it have been practical, to influence the normal treatment that these patients were otherwise receiving. We cannot exclude the possibility of baseline differences occurring due to chance, but nor have we any reason to believe that such differences exist.

We agree with Procter and have stressed for many years (Carney, 1967; Reynolds, 1967) that our observations are relevant to other psychiatric diagnostic categories. The influence of folate treatment in different mental disorders may be mediated by an effect on mood, drive, initiative and sociability (ultimately cognition), as may also be the case for S-adenosylmethionine, a metabolite closely related to folate in methylation processes within the nervous system (Reynolds *et al*, 1984).

Procter and Anderson *et al* discuss possible connections with monoamine and biopterin metabolism, which we have emphasised since our earliest studies of folate (Reynolds *et al*, 1970, 1984). In a recent study of 34 patients with a DSM–III diagnosis of depression (Bottiglieri *et al*, 1992) we found significant positive correlations between red cell folate and cerebrospinal fluid 5-hydroxyindoleacetic acid (CSF 5-HIAA), homovanillic acid (HVA), and tetrahydrobiopterin (BH4). As expected, there were also significant correlations between the latter three CSF metabolites. These observations further support the links between folate and monoamine theories of affective disorder. This and other recent studies (Carney *et al*, 1990) also emphasise the importance of studying the more stable red cell folate in psychiatric disorders and we would encourage Anderson *et al* and Wing & Lee to do so in their own studies.

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https://doi.org/10.1192/S0007125000124699 Published online by Cambridge University Press