Self-poisoning

SIR: I take the point raised by Halasz (*Journal,* August 1987, **151,** 267) when he mentions my use of the word 'epidemic' in describing a rise in incidence of deliberate self-poisoning in the 1960s and early 1970s (*Journal,* May 1987, **150,** 609–614). I would, however, like to reduce any confusion that might arise from his letter by reminding readers that I was referring not to a suicide epidemic, but to a rise in the incidence of deliberate self-poisoning, the vast majority of instances of which are parasuicides, i.e. non-fatal acts.

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Late Paraphrenia

SIR: Grahame's letter about late paraphrenia (*Journal,* August 1987, **151,** 268) raises important issues which are dealt with rather too casually. Holden may, no doubt, wish to answer criticisms directed at his paper, but it is worth pointing out that he could not possibly have interviewed his patients using the Geriatric Mental State (GMS) since attentive reading of his text will show that the study was a retrospective one based on case notes.

Grahame appears unshakeable in his belief that late paraphrenia is merely a form of schizophrenia in old age because of the high proportion of cases with first rank symptoms in his sample. We would suggest that both the history of the concept and the results of more recent, systematic prospective studies using the Geriatric Mental State (Naguib & Levy, 1987) should lead to a more cautious approach to this perennial problem. As we have pointed out elsewhere (Levy & Naguib, 1985), the proportion of patients with first rank symptoms in our own larger and unselected group of patients studied prospectively was much lower (16 out of 43, compared with Grahame's 14 out of 25). Thus, even if one accepts the debatable proposition that first rank symptoms necessarily indicate schizophrenia, clinical evidence does not indicate a simple equivalence between late paraphrenia and schizophrenia of late onset. Furthermore, the evidence from HLA sub-typing points in the opposite direction (Naguib *et al,* 1987).

The role of organic factors, both as reflected by cognitive tests and by CT scan measures (Naguib & Levy, 1987), is also rather more complex than Grahame is prepared to concede. We have found minor cognitive changes and ventricular enlargement in a group of late paraphrenics, and follow-up 3.7 years later did not suggest that this was a harbinger of 'organic brain syndrome', as Grahame seems to believe, nor did it bear any obvious relationship to the clinical picture in the survivors (Hymas *et al,* in preparation).

The concept of late paraphrenia or persistant persecutory state in the elderly is one which is peculiarly robust. It is unfortunate that the draft of ICD–10 (World Health Organization, 1987), currently undergoing field trials, will, if accepted, mean that such patients will be distributed among at least four different sub-categories, thus making them somewhat difficult to identify. Nevertheless, identification should be possible, and we believe that the application of a variety of new techniques to the investigation of the problem should begin to clarify the nosological status of this entity or entities. In the meantime, it would be wise to keep an open mind about the question rather than to adopt unduly dogmatic attitudes towards it.

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Comparative Trial of a New Antidepressant

SIR: I strongly agree with Dunn's comments (*Journal,* August 1987, **151,** 269) on the study by Levine *et al* (Journal, May 1987, **150,** 653–655), comparing fluoxetine and imipramine.

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There can surely be no doubt that new putative antidepressant compounds require testing against placebo in properly controlled double-blind studies before their efficacy can be regarded as established. Comparative trials against other 'established' antidepressants can never prove the efficacy of a new compound (although studies against the standard may have a place later in a compound's development). The main problem with trials against a standard antidepressant is that the investigators predict no difference between the two groups of patients. However, there is no way of testing the absence of a difference between groups. In contrast, testing a compound against a placebo leads to the prediction of a difference which can be tested by ordinary statistical techniques. Indeed, it is extremely easy to obtain a result of no difference between two treatment groups. One simply enters too few patients for statistical significance or makes any of the other design errors which beset even the best psychopharmacology trialists.

To take an example from the Levine *et al* study, patients in the imipramine comparison group were treated with 75–150 mg/day. In the event seven patients were treated at the 75 mg dose. This is well accepted to be inadequate, and a recent study (Thompson & Thompson, in preparation) confirms the inefficacy of 75 mg of a tricyclic antidepressant. Thus, one-third of the standard treatment group received a treatment which was no better than placebo, and a fluoxetine-treated group faired no better in terms of outcome than the imipramine-treated group. I would suggest that this study does not provide evidence of the effectiveness of fluoxetine as an antidepressant, and that only studies against placebo can do this.

There is one point on which I would disagree with Dunn. The use of atropine as a comparison treatment in antidepressant studies is in doubt, as it induces not only the side-effects of the older antidepressants but also may have some intrinsic antidepressant activity. The more appropriate "active placebo" would be methscopolamine, which has peripheral anticholinergic effects but does not pass the blood–brain barrier. Unfortunately, this compound is difficult to obtain and is thus not frequently used. However, in view of the relative lack of anticholinergic effects in the newer generation of antidepressants which are currently being tested, this may not be such a major flaw in current antidepressant trial design.

If psychopharmacologists are shying away from placebo-controlled antidepressant studies for ethical reasons, or if ethical committees are refusing to pass placebo-controlled studies, then they should consider the ethical implications of allowing a potentially ineffective antidepressant or any other compound onto an already overcrowded market.

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The Symbolic Significance of Munchausen's Syndrome

SIR: Cremona-Barbaro's paper on the symbolic significance of Munchausen's syndrome (*Journal*, July 1987, **151,** 76–79) prompts comment. Fanciful, dramatic and grandiose presentation is the hallmark of the Munchausen patient, and there may be some relationship between feigned bereavement and the syndrome, but it is by no means "the most common story" (Scoggin, 1983). There has been no previous mention of preoccupation with opera and operatic themes in the literature. Certainly none of our patients exhibited any such interest in the arts (O'Shea, 1987: personal communication).

The aetiology of Munchausen's syndrome would seem to be multifactorial, but there is growing evidence to support a transgenerational learning theory of maladaptive communication patterns with roots in early childhood (O'Shea, 1982; Meadow, 1982).

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SIR: It is difficult to know exactly what is "the most common story" given by Munchausen patients presenting with psychosocial symptomatology. Feigned bereavement is commonly accepted to be a very frequent presentation (Simpson, 1978; Snowdon *et al,* 1978), and will be familiar to psychiatrists and to those working in A & E departments. It is overwhelmingly the commonest story given in my own small series of Munchausen patients presenting with psychiatric symptoms.

I would agree that there has been no previous mention of a preoccupation with the opera in the

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