this study and that of Michel & Valach (*BJP*, June 1992, **160**, 757–760) which showed that knowledge about suicide and attitudes towards suicide prevention improved significantly in general practitioners who received a simple educational programme. A significant step in the right direction would be for psychiatric experience to become a required part of the training of all general practitioners.

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Seasonal affective disorder

SIR: We read with interest the paper by Murphy and colleagues (*BJP*, September 1993, **163**, 327–331) describing their findings on suppression of melatonin secretion in response to light in patients with seasonal affective disorder (SAD). Their paper makes reference to our earlier work (Thompson *et al*, 1990) and suggests that their failure to replicate our findings (that patients with SAD are supersensitive to light in winter) may be related to the fact that seven of our patients were taking benzodiazepines during the study, or because our controls differed in light sensitivity from theirs, since they found melatonin suppression values (using our method of calculation) to be 87% and 19% in our and their studies respectively.

We have recently studied the effect of daily treatment with 20 mg temazepam for seven days on melatonin secretion and light sensitivity in seven healthy male volunteers (Allen *et al*, 1993) and have found no effect on either parameter. This suggests that the small doses of benzodiazepines taken by seven of our patients were unlikely to have affected our results.

There is known to be a large variation between individuals in melatonin secretion and quite possibly in light sensitivity and this no doubt explains the differences between control values in the two studies. It would therefore be unwise to directly compare the studies or to merge the control groups. The findings of Murphy *et al* do not therefore invalidate our study.

Our study used a control night in which subjects were exposed to dim light, the light sensitivity being expressed as the difference in melatonin between control and 'bright light' nights. Murphy *et al* calculated sensitivity using measurements during a one-hour baseline period (01:00 to 02:00) and during the second hour of a two-hour period of exposure to light (03:00 to 04:00). It is possible that these differences make our test more sensitive. Furthermore, Murphy *et al* do not address the main finding of our study as they did not repeat their tests in the summer.

We demonstrated a significant seasonal variation in light sensitivity in SAD patients, with supersensitivity compared to controls in winter and a trend to subsensitivity in summer. The summer follow-up is also important in partially confirming the diagnosis of SAD. Preliminary findings from a seven-year follow-up study in our unit suggest that over 50% of DSM–III–R diagnosed winter depressives (American Psychiatric Association, 1987) remit or lose their seasonal pattern. Thus diagnostic heterogeneity of the samples may be a further cause of the differences in the findings.

We do not doubt the findings of Murphy *et al*, but their study differs in a number of ways to ours and cannot therefore be directly compared.

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Late-onset schizophrenia versus late paraphrenia

SIR: Howard *et al* (*BJP*, September 1993, **163**, 352–357) come to the conclusion that "although there are clinical similarities between cases of schizophrenia with early and late onset, there are sufficient differences to suggest that they are not phenotypically homogeneous".

The case for separation of late-onset schizophrenia, or what some of us still prefer to call late paraphrenia,

has repeatedly been made by those associated with this section within the Institute of Psychiatry. We have emphasised the aetiological (Naguib *et al*, *BJP*, January 1987, **150,** 124–127), symptomatological (Almeida *et al*, 1993), neuroradiological (Naguib & Levy, 1987; Burns *et al*, 1989), logical and pragmatic basis for doing so (Almeida *et al*, 1992). The main points of difference are those of terminology and cut-off age, both of which may appear arbitrary at first sight.

We choose the term 'late paraphrenia' partly because of its historical associations, but mainly because it is immediately identifiable as different. The vicissitudes of its appearance and disappearance from the ICD system of classification and its potentially dire effect on case identification have been described elsewhere (Quintal *et al*, 1991), as have those of 'late-onset schizophrenia' in the DSM system from which it is about to disappear as an identifiable entity (Levy & Almeida, 1993). This will make it difficult to separate these cases for more detailed studies, as they will be lumped together with schizophrenics who have grown old ('graduates').

The cut-off age has, to a certain extent, been a matter of taste, being 55 to 65 years in Europe and 45 years in the USA, but it need not be so. Some years ago, in a different context, a previous Editor of your journal (Slater, 1938) argued in favour of choosing the 'point of inversion' as that by which to separate 'involutional depression' from that occurring earlier in life. A similar point of inversion appears in age distribution by first admission for schizophrenia (Van Os *et al*, 1993): 65 years of age! There are, of course, more sophisticated mathematical techniques to clarify such issues, and we suggest that they should be applied to large series from different countries.

At a recent lively symposium on the topic at the Congress of the International Psychogeriatric Association in Berlin, several groups in Germany, Sweden, Denmark, Holland, the USA, and Canada appeared to have access to a sizeable number of cases. There was a clearly expressed wish to establish an international consortium to study some of the important and unanswered questions. Those who might be interested in joining such a consortium (minimum entry 40–50 well-documented cases) should get in touch with either of us, or with Dr Robert Howard at the address below.

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Benzodiazepine fatal poisonings

SIR: We are glad that Serfaty & Masterton (*BJP*, September 1993, **163,** 386–393) point out the important role of benzodiazepines in fatal poisonings. In 1990 we carried out a survey of drugs used in fatal self-poisonings in Switzerland (Michel *et al*, 1993), and we compared these findings (179 cases) with the drugs taken by suicide attempters over 12 months in the city and agglomeration of Berne (296 cases). We were surprised by the high number of benzodiazepines not only involved in attempted suicide (46 cases; 3% of all drugs taken, single or in combination with other drugs) but also in completed suicide (43 cases; 8%). Flurazepam was the benzodiazepine used most frequently in both groups, being named on 16 death certificates as only drug, and on 12 certificates in combination with other drugs. In some but not all of these cases alcohol was involved.

Our findings are therefore consistent with those of Serfaty & Masterton, and it is especially interesting that flurazepam which is still available in Switzerland appears to be the benzodiazepine with the greatest risk when taken in overdose. As could be expected, barbiturates were over-represented in fatal poisonings compared with non-fatal overdoses ($\chi^2 = 12.01$, $P = 0.001$). There was no statistically significant difference in the distribution of antidepressants (although there was a trend towards relatively higher numbers in fatal poisonings), benzodiazepines, neuroleptics or non-opiate analgesics between the