pattern to be retrieved, rather than a loss of the memory trace itself. Perhaps this distinction between the memory trace and the neural platform on which it could be retrieved is an important one highlighted by the current model and merits further neurobiological and neuropsychological elaboration.

It has been argued that changes in the connectivity and plasticity of synapses (connections between units) might be more relevant to memory impairment than loss of neurons (loss of units) (e.g. deToledo-Morrel *et al*, 1988). Models of memory impairment following random synaptic deletion have been well studied in the neural network literature and they demonstrated graceful degradation, that is relative preservation of function as synapses are deleted until a critical point following which function declines (e.g. Amit, 1989).

Temporal gradient for retrograde amnesia is not demonstrated in the current model; this is hardly surprising as none of the parameters in the model are time-sensitive and no attempt has been made to provide the model with a sequence of patterns to learn. To be able to describe this, various authors have designed networks with time-dependent parameters (e.g. McClelland & Rumelhart, 1986).

One further issue is that while medial temporal and diencephalic systems are essential for the laying down of memory, the actual memory trace is stored diffusely elsewhere in the cortex. Neurobiological models of amnesia will have to take into consideration different roles of the medial temporal system and the rest of the cortex (e.g. Rolls, 1989). It is unclear whether the author intends the current model to address the hippocampal system or another part of the cortex where memory is distributively stored.

Neural networks are emerging as a group of powerful but complex models. Their application in psychiatry may yield important insights. However, the level of modelling (e.g. psychological, brain systems, local cortical circuits) as well as the limitation of a particular model must be adequately addressed. The choice of model and the parameters is crucial and requires very careful consideration.

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Suicide prevention

SIR: We are in agreement with Dr MacDonald (*BJP*, August 1993, **163**, 260) that suicide prevention in severe psychiatric disorders should not be compromised by transfer of resources into new community-based initiatives, the effectiveness of which may as yet be uncertain. In our original letter (Hawton & Morgan, *BJP*, March 1993, **162**, 422) we did, however, emphasise that the assessment and management of suicide risk concerns the whole spectrum of day-to-day clinical psychiatric practice, the review of which should not demand redistribution of resources as Dr MacDonald fears. There is much to be done in defining new styles of clinical practice as its emphasis moves increasingly towards the community, and in establishing close links between general practitioners and community mental health teams, particularly with regards to the management of acute suicide risk. In our experience, general practitioners are eager to begin this debate.

Dr MacDonald dismissed our point concerning the comparable incidence of suicides and of certain common chronic organic diseases. Surely the psychological distress leading up to an act of suicide is a real challenge to all clinicians, demanding closer attention to ways of increasing consultation rates and improving our skills in recognising short-term suicide risk.

The Gotland study (Rutz *et al*, 1992) is of course certainly not free of methodological problems but the authors faced these fully in presenting their findings. It is true that the change in the suicide rate immediately after the educational programme might represent a chance finding, but it is nevertheless impressive that following the educational programme, as the prescribing of antidepressants increased, so both the number of days of work lost because of depression and the psychiatric hospital admission rate for depressive disorders decreased. The papers on the Gotland study ought to be reviewed in the original by those who wish to judge their findings for themselves, but in the meantime this study should at least stimulate debate and replication. Education, not shifting of valuable resources, seems to be the most important implication of both

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.

RUTZ, W., VON KNORRING, L. & WÄLINDER, J. (1992) Long-term effects of an educational program for general practitioners given by the Swedish Committee for the Prevention and Treatment of Depression. *Acta Psychiatrica Scandinavica*, **85**, 83–88.

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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,