changes associated with particular lesion lateralisation or location. However, any hypothesis of the mechanisms of mood disturbance following focal brain injury must account for the apparent association of left anterior lesions with depression and of right-sided lesions with euphoria. It is to be hoped that as our understanding of the subcortical substrates of behaviour increases, we will be able to better understand the pathogenesis of cases such as that reported (McGilchrist *et al*, 1993).

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Parental bonding

SIR: Morris' letter (*BJP*, July 1993, **163**, 127) criticising my paper (*BJP*, March 1993, **162**, 335–344) requires a reply. Bowlby's definition of 'a poor early relationship' in the passage I cited was too general to be incorporated into a testable hypothesis. Since Bowlby went on to refer to the individual's "capacity to make affectional bonds", it seemed that the most precise hypothesis I could test was that there was a relationship between recalling having good affectional bonds in childhood and the ability to make good affectional bonds in adult life. Maternal care was the experience which came closest to having good affectional bonds. In the two samples I used, this was assessed by interview and, in the younger sample, it was also assessed by the care scale of the mother version of Parker's Parental Bonding Instrument (PBI). In an earlier paper (*BJP*, December 1988, **153**, 758–769) I reported upon findings using scales of both the mother and the father versions of the PBI. Focusing specifically upon the care scale is a perfectly legitimate exercise. To be consistent, I also focused specifically upon the affection-given scale of the Marital Patterns Test.

It seems that what Morris is suggesting is that I should extend the study to testing the additional hypothesis that being subjected to parental overprotection in childhood (the other scale of the PBI) predisposes the individual to either seek or avoid overprotection in later relationships, and that I should use the directiveness scale of the self-rating and partner-rating questionnaires (*BJP*, May 1991, **158**, 648–657) as a measure of such overprotection. This is indeed a reasonable suggestion since Parker quite often (and more correctly I think) refers to the overprotection scale as the control scale, and control and directiveness are comparable constructs. Since, however, in the 1988 paper there was shown to be a highly significant association between recalled maternal overprotection and depression, the intervening variable of depression is going to complicate matters as it did with the maternal care hypothesis—an issue which Morris studiously avoids.

Morris refers to "the total parental relationship" as getting closer to Bowlby's hypothesis. In the passage I cited, Bowlby used the term "an individual's experiences with his parents". This is a vague term which can hardly be preferable to defining the recollected relating behaviour of one or other parent in certain specific ways. I end my paper by maintaining that further progress in this field will depend upon expanding the theoretical base, increasing the range of parameters of relating to be considered, and developing new instruments which are based upon this broader perspective. In my recent book (Birtchnell, 1993) this is what I have tried to do.

BIRTCHNELL, J. (1993) *How Humans Relate: A New Interpersonal Theory*. London: Praeger.

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Neural network model of amnesia

SIR: Neural network modelling is a rapidly advancing area which, if appropriately applied, may be useful in psychiatry. The article by Carrie (*BJP*, August 1993, **163**, 217–222) is therefore welcomed with enthusiasm. Several comments are, however, important.

A simple associative network is used and the author chooses to represent diffuse cerebral pathology by random deletion of units. It is important to realise that units are substrates for the representation of patterns (during presentation and retrieval), and the memories for the patterns are stored in the synaptic weights between the units (which change before and after learning). Removal of units will naturally affect 'retrieval' due to the lack of a proper platform for the

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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ROLLS, E. T. (1989) Functions of neuronal networks in the hippocampus and neocortex in memory. In *Neural Models of Plasticity: Experimental and Theoretical Approaches* (eds J. H. Byrne & W. O. Berry), pp. 240–265. San Diego: Academic Press.

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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