It is true that age and age of onset may no longer be regarded as reliable predictive factors in view of the somewhat contradictory results reported by different authors (Huff *et al*, 1987), but the sinister prognostic significance of visuo-spatial dysfunction must be considered as one of the most robust findings in the clinical literature on Alzheimer's disease. Its relationship to dysphasia and other focal features on the one hand and to overall severity on the other is a complex one which my group is trying to unravel in the course of a prospective study.

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Gluten Sensitivity in Schizophrenia

SIR: Singh & Kay (*Journal*, January 1987, **150**, 130–131) make telling points in their criticism of our study of gluten sensitivity in schizophrenia, most of which we have already made and accept. We hardly perceived ours as the critical experiment; such experiments are difficult to perform in any science and very difficult in clinical psychiatry. They are usually misreported simplifications of the history of science. Indeed, we experienced, as others will, considerable problems in getting psychotic people to co-operate convincingly in accepting dietary controls, and so the attempt, which is what we report, was of short duration and in a special hospital. We certainly don't wish to deter others from performing more adequate work, but in our limited study the major changes occurred in the patients before they had a gluten-free period. We were pleased that, fortuitously perhaps, the *Journal* published Singh & Kay's letter after one by Wing suggesting "very impaired patients" were "not impervious to social stimulation", as seemed obvious in our study.

In addition, though, we are blamed for not considering the heterogeneity of the syndrome, while in fact we discussed it and concluded that gluten-free diets may be of value to some schizophrenics. We could go on at length over details of interpretations, but perhaps it would be wisest to concede that Singh and Kay have done an immense amount of work in this field, which we took seriously enough to examine and to try to confirm. Hence we are sorry if our inability to support them in our short-term and necessarily imperfect study can be read as wishing to dismiss their efforts and their approach completely. Far from it – we want, with them, to see what other studies can demonstrate, but we needed to report what we found.

We note, though, that Singh & Kay have written several similar letters criticising other peoples' failures to confirm their hypothesis (Singh, 1979; Singh & Kay, 1983).

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Sub-cortical Dementia

SIR: In his review of sub-cortical dementia (*Journal*, December 1986, **149**, 682–697) Cummings makes the statement: "Sub-cortical dementias are characterised by psychomotor retardation, whereas the cortical dementias" (among which he includes Alzheimer's disease) "manifest a normal psychomotor speed through most of the clinical course", and supports this with reference to Cummings & Benson (1986). Although it is true that this view has become something of an orthodoxy in neurological texts, it flies in the face of much evidence from other sources. For instance, it is clear that on the Digit Copying Test (DCT) component of the Kendrick Battery (Kendrick *et al*, 1979) groups of patients with dementia predominantly of the Alzheimer type were significantly slower than non-demented subjects. Evidence of slowing also comes from other sources using quite different techniques. Thus slowing is also found in Alzheimer patients when they are asked to identify pictures presented tachistoscopically (Neville & Folstein, 1979) or when they carry out tasks using a peg-board (Miller, 1977). This issue is discussed by Woods (1982), who suggests that a critical point may be the degree of cognitive load in terms of choices available. Another important variable which may be seen as a form of cognitive load is the delay involved before the subject is expected to respond. In a study using a computerised visual matching to sample task with variable delay