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 (Morris et al, 1987; Sahakian et al, 1987) we found that patients with relatively early Alzheimer disease only reacted as rapidly as controls when an immediate response was required (i.e. with a zero second delay). The data show that when the test stimulus is delayed the Alzheimer patients respond a good deal more slowly than the age-matched controls, and that in this respect they perform far worse than recently diagnosed patients with Parkinson's disease who showed much smaller differences from their own controls on this task.

We suggest that psychomotor speed is a complex variable, and that contrary to belief it is not a useful way of differentiating between cortical and subcortical dementia.

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# Dementia in Parkinson's Disease

SIR: Oyebode et al (Journal, December 1986, 149, 720–725) present data about dementia in 43 patients with Parkinson's disease, and draw conclusions concerning the prevalence of this. Their population was drawn from patients attending an illustrious neurological centre. Perhaps the area served contains patients with Parkinson's disease who were not referred to the centre, but were looked after by general practitioners, geriatricians, or psychiatrists.

There might have been a tendency to fail to refer the bedridden, the incontinent, and the demented.

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# Anorexia Nervosa or Dysmorphophobia?

SIR: An alternative diagnostic formulation is possible (Journal, December 1986, 149, 780-782). As Hay (1970) points out, dysmorphophobia is a nonspecific symptom often indicative of a 'sensitive personality development', or occasionally early schizophrenia. The phenomenological distinction between the overvalued idea and the delusion depends in part on external evidence of physical abnormality and adverse environmental experience, often in the form of teasing in childhood. This girl was clearly obese at the age of 12, was instructed to lose weight by two professionals, and had previously been teased by family members. The positive family history of a transient psychotic episode in her brother with no further deterioration would support the formulation of a constitutionally-based sensitive personality prone to neurotic elaboration. While the patient meets the criteria for both agoraphobia and social phobia, she apparently never lost enough weight or persisted long enough in her dieting to qualify as a case of anorexia nervosa by DSM-III criteria, but would be more appropriately labelled as having atypical eating disorder.

These diagnostic distinctions may have significant clinical relevance for treatment. Slade and others have documented the variability in body size estimations of anorexics who, as a group, have a tendency to over-estimate certain body part dimensions. Unfortunately, no body image measurements are reported in this case. In contrast, body image measurements have been described in dysmorphophobic patients which might help differentiate them from the anorexic group. Jerome (1980) described a group of patients with dysmorphophobia requesting cosmetic rhinoplasty who were more accurate than normal controls in estimating their own nose size, and spent more time looking at this particular feature of their appearance in mirrors. The data suggested that dysmorphophobics may be utilising a 'perceptual defence' of over-focusing on specific aspects of their body image. This contrasts with the clinical findings in anorexia nervosa, which suggest an avoidance of observation of their own bodies with parallel mirror avoidance and a relative over-estimation of specific body parts in comparison with controls. Furthermore, a recent article by Norris (1984) suggests that