(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 non-specific 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

anorexics might show clinical improvement following mirror confrontation.

In contrast, preferred treatment for dysmorphophobia might involve the reverse behavioural approach. Support for this hypothesis comes from clinical accounts of patients who report themselves much improved after cosmetic surgery when objective ratings of their appearance indicate no significant change.

The patients who do well have a supportive confidant, an optimistic attitude, and have been prevented from looking at the particular body part, which is shrouded in dressings after surgery. This arguably may allow the dissolution of the distorted body image, which in part may be a manifestation of heightened cognitive awareness for, and the preferential visual scanning of, the body part in question. A similar cognitive-behavioural approach could be envisaged for over-focused dysmorphophobic patients.

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Organic Factors in Catatonia

SIR: Though Wilcox & Nasrallah (*Journal*, December 1986, **149**, 782–784) note that catatonia has been reported in non-schizophrenic individuals, they fail to mention that in a study of 55 consecutively admitted catatonic patients Abrams & Taylor (1976) found a preponderance of cases with manic (34) and depressive (5) disorders but only four with schizophrenia. Similarly, of the 25 cases studied by Barnes *et al* (1986), ten were idiopathic and nine had a depressive illness. There was a single case of schizophrenia. Both the idiopathic and the affective groups had a high incidence of recurrent episodes and a family history of catatonia. These findings confirm Kahlbaum's original description of catatonia as a non-specific syndrome, rather than a disease entity, with a wide range of causes, of which affective disorders appear to be the most common.

The undue emphasis on the association of catatonia with schizophrenia is also pertinent to the tragic patient reported by Ainsworth (*Journal*, January 1987, **150**, 110–112). In view of the striking affective changes in her patient's mental state (as well as the family history of depression and mutism), an affective disorder, possibly mania, seems a more appropriate diagnosis than catatonic schizophrenia. Though reported as a case of Stauder's lethal catatonia, no evidence is presented of its characteristic features, i.e. mounting fever and extreme hyperactivity progressing to stuporous exhaustion and death. Clinically it resembles the more familiar benign variety of Kahlbaum (Mann *et al*, 1986).

Ainsworth raises several questions which have an important bearing on the treatment of such patients. Though amylobarbitone sodium interview is reportedly helpful in distinguishing catatonia of psychiatric origin from that with a toxic-metabolic or neurological basis, its value in the lethal variety is uncertain. Whether a putative affective disorder developed subsequent to a sub-clinical viral encephalitis or predisposed her patient to increased infection is a moot point (Wilson, 1976), but the brief return to normality after i.v. diazepam would argue against the usefulness of such techniques rather than render a diagnosis of encephalitis less probable in light of the clinical and pathological findings. Orthostatic hypotension due to the high doses of chlorpromazine could have caused the patient to fall, and while the cause of death seems unrelated to the catatonia, a viral encephalitis may well have compromised her gag reflex, resulting in fatal aspiration. The outcome for patients with benign catatonia, whether of psychiatric or other origins, is described as excellent and most recover spontaneously or rapidly after ECT, although a high rate of recurrence has been reported, particularly among those with a family history of catatonia (Barnes *et al*, 1986). In patients with lethal catatonia ECT may also be dramatically effective, and perhaps lifesaving (Mann *et al*, 1986).

Finally, several authors have commented on the similarities between lethal catatonia and the neuroleptic malignant syndrome, which may well be a drug-induced variety of the former. Because of the dangers posed by neuroleptic treatment in iatrogenic and other forms of lethal catatonia, as well as evidence that some patients develop stupor and fever only after the initiation of such treatment, a prudent approach would be to discontinue its use in benign catatonia too. In view of its efficacy, ECT would appear to be the treatment of choice in catatonia, although in some cases of the lethal variety other