prognostic features our series may have been, a good outcome for 3 out of 21 is very poor.

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Possible causes of catatonia in autistic spectrum disorders

I read with interest the paper by Wing & Shah (2000) on catatonia in autistic spectrum disorders. The authors quite correctly make the point that catatonia, although a useful clinical concept, is a description of a number of behaviours. However, they have not attempted to investigate the aetiology of catatonia in their sample of 40 patients. Three other possible causes for their observations spring to mind.

First, the onset of catatonic symptoms in adolescence or early adulthood, in this largely male sample, could be related to the development of schizophrenia, although it may be difficult to diagnose. It has presumably been excluded as no patients had first-rank symptoms according to the accounts of relatives or carers, although in Table 3 (p. 359), the heading "bizarre/ psychotic" catatonic manifestations were found in 40% of their patients. The fact that 'Others had occasional visual hallucinations or paranoid ideas' suggests that they may qualify for an additional diagnosis of schizophrenia according to the ICD-10 (World Health Organization, 1992). The authors have not specifically stated whether the patients had been assessed for a diagnosis of schizophrenia.

Although the patients may be difficult to interview on account of communication disorders or cognitive problems, nearly half did not have impaired language and the number of mute patients is not stated. Furthermore, 70% of the patients had a level of cognitive ability within the range from mild learning disability to average

intellectual ability, not incompatible with a diagnosis of schizophrenia.

Second, the possible explanation for catatonic symptoms is the development of an affective disorder. In 13 of the 30 patients, precipitating factors included bereavement, pressure at school, lack of structure after leaving school and lack of occupation, which are more commonly associated with a depressive illness. Central to the diagnosis of catatonia are increased slowness, difficulty in initiating and completing actions and lack of motivation, among others, possibly symptoms of depression.

Third, and most importantly, catatonic symptoms may be difficult to distinguish from the extrapyramidal side-effects of antipsychotic drugs (American Psychiatric Association, 1992). In Wing & Shah's description of the criteria for catatonia, a secondary feature listed was "Parkinsonian features: tremor, eye-rolling, dystonia, odd stiff posture, freezing in postures, etc.". Although the patients are fairly young, they are also a tertiary referral group and it is likely that they would have received other, previous treatments. Recent estimates of prescriptions of psychotropic medication to adolescents and adults with developmental disabilities vary from 12 to 40% (Connor & Posever, 1998). There was no record of previous treatment and, more specifically, a history of current or prior exposure to antipsychotics is omitted.

It is helpful to know that catatonia can complicate autistic spectrum disorders and that individuals who present with catatonia may have an undiagnosed autistic spectrum disorder. However, although recognition is necessary to institute appropriate management, this paper offers only limited help in this direction. There would have been a greater clinical impact if it had addressed the possible causes of catatonia or the other associated psychopathology. The study also raises the question of whether catatonia represents the expression of other, more common mental disorders in those with limited communication skills.

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Authors' reply: Dr Chaplin notes that neither the possible causes nor the treatment of catatonia were discussed in our paper. As the *Journal* requires papers to be 3000–5000 words long, we decided to focus on the clinical picture of catatonia in autism and its prevalence. We have written and intend to publish a second paper dealing with causes and treatment and are grateful to Dr Chaplin for providing us with the opportunity to write a few more words on these subjects.

The individuals in the study had all been seen by one or more clinicians before the tertiary referral to Elliot House. During the course of the multiple assessments, possible underlying causes, including schizophrenia, depression, obsessivecompulsive disorder and identifiable brain pathology such as parkinsonism, would have been considered. These conditions, together with autistic spectrum disorders and catatonia, are defined and diagnosed only on history and clinical picture and there is overlap of clinical features among them all. In the individuals in our study, the developmental history and clinical picture, including the "bizarre/psychotic" behaviour in some people, fitted best with autistic spectrum disorders. We do not argue that psychiatric conditions, such as schizophrenia, cannot occur in association with autistic disorders. The point of our paper is that catatonia can occur as a complication of autistic spectrum disorders alone.

Twenty-one individuals in our study had received psychotropic medication for possible psychiatric conditions, and two people were treated with electroconvulsive therapy, all without useful effect on the catatonic features. The side-effects of neuroleptic medication were considered as possible causes of the catatonia. Of the 21 individuals who were medicated 10 were given drugs only after the onset of catatonia. The temporal relationships were