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ABSTRACT The phenomenon of catatonic-like states in people with autistic spectrum disorders is discussed in the context of current knowledge about catatonia as it occurs in severe mental illness and, less frequently documented, in conjunction with developmental disorders. The existing literature on catatonic-like states in people with autistic spectrum disorders is summarized, and it is suggested that such states are not directly comparable with the existing concepts of catatonia. A concept of 'autistic catatonia' is outlined in terms of both its phenomenology and its possible aetiological and maintaining factors. A case study is presented that examines this phenomenon from a cognitive neuropsychological perspective, together with implications for everyday management. The implications of this work for both research and clinical practice are discussed.

KEYWORDS
autism; autistic catatonia; catatonia

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Introduction

The occurrence of a catatonic-like state in certain individuals with a diagnosis of an autistic spectrum disorder has been noted in a number of clinical reports, but essentially this remains an under-researched area in terms of phenomenology, epidemiology and aetiology. The aim of this article is to begin to provide a framework for investigating and understanding such states, with the ultimate goal of developing appropriate and effective clinical and social interventions and support.

Definition of catatonia

'Organic catatonic disorder' is described in ICD-10 (World Health Organization, 1992) as 'a disorder of diminished (stupor) or increased (excitement) psychomotor activity associated with catatonic symptoms' and is defined

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in conjunction with organic cerebral dysfunction. ICD-10 lists the following diagnostic criteria for 'organic catatonic disorder':

One of the following should be present:

1 Stupor, i.e. profound diminution or absence of voluntary movements and speech, and of normal responsiveness to light, noise and touch, but with normal muscle tone, static posture and breathing maintained (and often limited co-ordinated eye movements).
2 Negativism (positive resistance to passive movement of limbs or body or rigid posturing).
3 Catatonic excitement (gross hypermotility of a chaotic quality with or without a tendency to assault).
4 Rapid and unpredictable alternation of stupor and excitement.

Prevalence of catatonia

Estimates of prevalence in psychiatric populations range from 0.6 percent for adolescents (Cohen et al., 1999) to between 6.9 and 10 percent for psychiatric inpatients (Blumer, 1997; Bush et al., 1996). Bush et al. (1997), studying cases of chronic catatonia resulting in long-term hospitalization, found an association with both Parkinsonian symptomatology (24 percent of cases) and akathisia/tardive dyskinesia (10 percent of cases), with catatonia being the sole presentation in only 21 percent of the cases studied. Cohen et al. (1999), in their discussion of nine cases of catatonia in adolescents, noted that, as well as being associated with diagnoses of schizophrenia, psychotic depression, mania and schizophreniform disorder, two of the cases had a diagnosis of pervasive developmental disorder.

Given the variation in presentation of catatonia, the question of subtypes of catatonia in people with a diagnosis of schizophrenia is raised by Pfuhlmann and Stoeber (1997). Catatonic conditions are also noted as a side-effect of psychopharmacological intervention (Lauterbach, 1998); following certain metabolic disorders, e.g. vitamin B deficiencies (Catalano et al., 1998); as the sequelae of encephalitis (Johnson and Lucey, 1987) and temporal lobe epilepsy (Dubin et al., 1985); and in conjunction with severe obsessive-compulsive disorder (Hermesh et al., 1989).

Aetiology and models of catatonia

Blumer (1997) notes that, due to increased use of neuroleptic medication, catatonia is no longer as strongly associated with diagnoses of schizophrenia as previously, but has become more associated with both medical conditions and affective disorders. The search for the aetiology of catatonia has likewise changed over the past decades. From an earlier emphasis on

biochemical factors (e.g. Gjessing's 1974 model based on catecholamine metabolism disturbance affecting the sympathetic nervous system), there is now a focus on presumed neuropsychological dysfunctioning. Northoff et al. (1999) have proposed a model of catatonia implicating abnormalities in frontal-parietal cortical function. Following assessment of attention, executive, visual-spatial and working memory abilities in 13 individuals with catatonic symptoms, it was observed that performance on visual-spatial object perception was significantly poorer when compared with control groups both with and without similar psychiatric diagnoses. Northoff et al. (1999) suggest that catatonia involves visual-spatial deficits related to attentional-motor dysfunction and right frontal-parietal cortical function.

Joseph (1999), reviewing clinical and research work, including recent neuro-imaging studies, on catatonic conditions notes that catatonia and what can be termed 'disturbances of will' are indicative of medial frontal injuries. However, the linkage between localization of dysfunction and behaviour is not straightforward:

> as the frontal subdivisions are richly interconnected, and as frontal lobe abnormalities are not always discrete or well localised, a wide array of seemingly divergent waxing and waning symptoms may be manifest, sometimes simultaneously, including manic depression and what has been referred to as the 'frontal lobe personality'. (Joseph, 1999)

(See also Belfer and d'Autremont, 1971, for a discussion of catatonia-like symptomatology related to left frontal lobe dysfunction.) Interestingly, the still relatively new technology of transcranial magnetic stimulation is reported to have been successful in treating catatonia when applied over the right-frontal cortex (Grisaru et al., 1998). Models of catatonia implicating dysfunction in the basal ganglia region have also been proposed, although like other proposed models this is on the basis of a single case (Luchins et al., 1989).

A more functional approach to understanding such conditions, incorporating phenomenological and environmental factors, was taken by Perkins's (1982) multifactorial model of acute catatonia, in which the presence of severe physical illness, organic brain disease and fear are significant factors in the onset of catatonia. In this formulation, catatonic behaviour could be

> viewed as the ultimate response to fear in a person under enormous psychological and physical stress and in whom regression to a primitive form of expression has occurred. The presence of organic brain disease may be necessary to lower the threshold for the release of this form of behaviour. (Perkins, 1982)

The utility of a multifactorial model with its emphasis on the *function* of catatonia appears to have face validity with regard to existing clinical practice with people with autistic spectrum disorders (Clements and Zarkowska, 2001).

Catatonia and autistic spectrum disorders

Although discussions about catatonia have generally focused on people without developmental disorders or learning disabilities, several clinical reports can be found. Reviewing clinical reports of catatonia in children and adolescents, Dhossche and Bouman (1997a) report that in a minority of the 28 cases examined, catatonia was found in conjunction with learning disabilities and autism. Catatonia has been noted in conjunction with both learning disability of an unspecified nature (Gaind et al., 1994) and specific neurodevelopmental disorders, for example Prader–Willi syndrome (Dhossche and Bouman, 1997b). The first clearly reported association between catatonia and autistic spectrum disorders can be found in Wing's (1981) seminal case series on Asperger syndrome. Wing noted catatonic stupor in the presentation of the 17 percent of her sample of 34 cases who were considered to be psychotic, whilst Gillberg (1985) described a case of a 14-year-old boy with a diagnosis of Asperger syndrome who showed severe recurrent psychotic episodes, which included extreme passivity and lack of spontaneous actions. In the context of a follow-up study of children with autistic spectrum disorders in adulthood, Gillberg and Steffenburg (1987) reported the estimated prevalence of 'catatonia' to be 6.5 percent at the time that their population reached adulthood. Compared with other populations for which estimates of prevalence are available, this appears to be a significantly increased rate.

Realmuto and August (1991) propose that catatonia in people with autistic spectrum disorders is directly related to the autism rather than being a separate comorbid condition *per se*. However, they go on to examine the significance of bipolar mood disorders and 'organic deficits' as risk factors for catatonia and discuss three cases. Again, these cases are of young people (aged 16–21 years) with autistic spectrum disorders. Bell (1997) describes the case of 'John' who developed a catatonic-like state at the age of 21. His symptoms involved postural freezing and remaining in one room, with catatonic excitement and self-injury if attempts were made to prompt him to move. This individual had a previous history of travelling very long distances, a habit that reappeared 5 years after the onset of the catatonic-like state, and the account contains indications that there may have been a cyclical variation in his presentation.

Dhossche's (1998) single case study of an adolescent with an autistic spectrum disorder who presented with catatonia is important as the catatonic

symptoms, including stuporous episodes with mutism, waxy flexibility, posturing and episodes of agitation, match the current DSM-IV criteria for the catatonic features specified.

A recent case report by Brasic et al. (1999) of a 23-year-old man, previously diagnosed as having a pervasive developmental disorder, describes the cause of persistent catatonia (indicated by motoric immobility, mutism, and peculiarities of voluntary movement such as prominent grimacing in conjunction with mutism, catalepsy and right-side hemiparesis). In this instance, there was evident deterioration in motor functioning over a 2 year period. Subsequent neuro-imaging using positron emission tomography indicated hypometabolism in the right cerebral cerebellar areas. In this instance, Brasic et al. consider that an unspecified neuro-degenerative condition underlay the catatonic and degenerative symptomatology.

The most comprehensive investigation of the extent and phenomenology of catatonia in people with autistic spectrum disorders is that recently conducted by Wing and Shah (2000) on a sample of 506 referrals to a specialist assessment centre. Of this group, 6 percent met Wing and Shah's criteria for catatonia, which encompassed:

- increased slowness affecting movement and verbal responses
- difficulty in initiating and completing actions
- increased reliance on physical or verbal prompting by others
- increased passivity and apparent lack of motivation.

Wing and Shah also included day/night reversal, Parkinsonian features, excitement and agitation and increases in repetitive behaviours as signs of catatonia in people with autistic spectrum disorders. Despite the variations in diagnostic criteria used, the figure of 6 percent accords with the previous estimates of Gillberg and Steffenburg (1987). In their discussion, Wing and Shah note that the onset of catatonia in their sample generally occurred between 10 and 19 years. No distinct precipitating events or factors could, retrospectively, be identified for the majority of people. On the basis of their phenomenological similarities, Wing and Shah suggest that catatonia in people with autistic spectrum disorders may have a common neuropathology with conditions including obsessive-compulsive disorder, Tourette's syndrome and attention deficit disorder. They also draw attention to the *behavioural* overlap between autistic disorder and the catatonia reported in their sample, in terms of motor stereotypies, mannerisms, mutism, ritualistic behaviour and 'negativism'. Using parental self-report and less rigorous criteria (sitting and staring, 'freezing' and standing very still for long periods), a community-based audit of people with autistic spectrum disorders and learning disabilities (Bromley et al., 2002) found that catatonia thus defined was reported by 46 percent of families interviewed.

The concept of 'autistic catatonia'

A particular difficulty with studies in this area is that (with the exception of the cases described by Dhossche, 1998, and Brasic et al., 1999) what is usually described as 'catatonia' in the context of autistic spectrum disorders does not always match the wider definitions of catatonia. The study by Wing and Shah (2002), whilst comprehensive, used an idiosyncratic and broad definition of catatonia. As noted, it is difficult to determine whether the phenomenon described in the non-autistic population is the same as that being termed 'catatonia' in people with autistic spectrum disorders. However, given the paucity of information about 'catatonic' states in people with autistic spectrum disorders and the apparent heterogeneity in both presentation and putative causation, an over-rigid adherence to diagnostic criteria developed for non-autistic populations may be inappropriate. Perhaps the term 'autistic catatonia' might, at this stage of our knowledge, have greater utility.

Based on clinical reports, it is suggested that a basic clinical definition of autistic catatonia would involve the following behaviours being present over at least 1 month:

- freezing when carrying out actions and being resistant to prompting
- very slow voluntary motor movements
- stopping in the course of movement and requiring prompting to complete actions.

In addition, a resting tremor and/or noticeable jerky movements may sometimes be observed. Whilst there are anecdotal accounts of some individuals reporting feelings that time is passing too slowly or too quickly, the frequency of such experiences in this population is unclear.

Although the prevalence of and association between these signs is currently unknown, on the basis of the available clinical data, psychological and neuropsychological models for such autistic catatonia can be advanced.

It is possible that some forms of autistic catatonia might reflect extreme adaptations made by the person with autism to environmental factors, for example, escape from contingent demands (cf. Perkins, 1982). Alternatively, autistic catatonia could be conceptualized as resulting from the same psychological processes that underlie other aspects of autistic behaviour. For example, it is possible to conceptualize autistic catatonia within the framework of Frith and Happé's (1994) lack of central coherence model. In this instance, the inability to integrate discrete 'bits' of both sensory data and motor action could lead to very slow responses and impairments in voluntary behaviour by people with autistic spectrum disorders. Similarly, the need to engage in ritualistic and repetitive actions by people with autistic

conditions may be involved in the development of autistic catatonia (cf. Realmuto and August, 1991). Finally, theories of autism that emphasize the role of executive dysfunction (Russell, 1997) may provide a link with models of catatonia in non-autistic populations, in whom catatonia is conceptualized as resulting from frontal lobe dysfunction and associated impairments in motor control (Taylor, 1990).

A case study of autistic catatonia

In order to illustrate the presentation of autistic catatonia in people with autistic spectrum disorders and to examine implications for clinical practice, a detailed case study is presented, with a focus on the neuropsychological assessment of the early stage of such a condition. The subject, a young man 'A' aged 18 years, had a developmental history and current behaviour patterns that fulfilled the criteria for an autistic spectrum disorder. A number of standard neuropsychological assessments were carried out, including the Wechsler Adult Intelligence Scale–Third Edition (UK) (WAIS–III), the Visual Space and Object Perception Battery (VSOP: Warrington and James, 1991), the Rey Complex Figure Task (CFT), the Trail Making Test (TMT) and other tests of perseveration and planning (Lezak, 1995). A's cooperation and concentration were very good during the formal assessments and he performed all of the tasks requested of him. His performance IQ score on the WAIS–III was in the low 60s, indicating mild learning disability.

On the Visual Space and Object Perception Battery, A performed within the normal ranges for spatial location, but was markedly impaired on tasks involving visual recognition and decision-making. No constructional difficulties were evident from his performance on the Complex Figure Task, other than the very long time taken for completion. On a maze task, A made errors when required to change direction with his pen, but on both this test and the Complex Figure Task he generally recognized and self-corrected such errors. On the Finger Tapping Test (Luria, 1973), which is very sensitive to difficulties in switching from one task to another and in inhibiting previous responses and actions, A did not show any motor perseveration.

Although formal testing indicated no apparent problems with planning actions and movements and no significant signs of perseveration, either motor or verbal, on a day-to-day basis A's most frequent difficulties related to his ability to move and carry out activities. These included:

- extreme difficulty in initiating actions such as getting up and out of bed, sitting and standing up, moving from one room to another

- needing physical assistance to carry out actions, e.g. walking
- becoming stuck, for example when stepping off the bottom step of the stairs, going through doorways, turning round when walking
- difficulties in coordinating his limbs when getting dressed and requiring assistance
- problems in stopping his actions, for example when in the bathroom and washing his hands
- auditory hypersensitivity (hyperacusis) to loud voices, fireworks, doors slamming, dogs and thunder, resulting in an apparent panic reaction.

These difficulties had worsened over a 4 year period since A was 15 years of age and had led to the curtailment of previously established activities, such as walking to the local shops. His parents reported that he did not become 'stuck' when carrying out similar actions as a child, but that he often displayed stereotyped rocking and flapping behaviours (when excited) and that his bodily movements had been more fluid and less jerky.

A rocking side-to-side gait was observed when A was walking and he presented with a noticeable resting tremor. It was also noted that A's saccadic eye movements were larger and faster than would otherwise be expected. He had considerable difficulties in passing through doorways, but was able to cross an 'artificial' threshold on the floor (masking tape) without difficulty. It is possible that the difficulties in passing through doorways were related to the turning into the doorways and the perceived visual changes involved (e.g. differing perspectives and view, contrasts in floor materials etc.), rather than the crossing of thresholds *per se*. An important and particular instance of A becoming 'stuck' concerned his leaving the bathroom on the first floor of the family home, which could take up to half an hour to complete. When attempting to sit down or stand up from his chair, a distinct sequence of events was observed. When attempting to sit down, A stood beside the chair for a period of time and then quickly positioned himself over the chair and grasped the chair sides, during which time he appeared to be very tense. When attempting to lower himself into the chair, he invariably stopped halfway and became very tense and would then stand up. This process would be repeated several times until he was able to sit down. When sitting down, he showed no apparent distress.

Informal physical and verbal prompting did not have any noticeable effect on the speed of A's actions or on the manner in which he carried then out. Moreover, he showed a marked degree of resistance to being physically cued (limbs becoming rigid, returning to his starting position etc.). A was able to carry out many actions which did not involve whole body coordination and movement, such as eating and drinking in an unimpaired manner.

A was verbal, but did not speak spontaneously. He answered questions about his thinking and actions and stated on several occasions that he found it hard to move and that he was anxious about moving. For example, when he was in the bathroom for 25 minutes, he replied 'too difficult, I can't do it ' when asked to come out and down the stairs.

On the basis of the assessment, it appeared that A did not have problems in the planning of actions, but that his *execution* of actions was markedly impaired. He appeared to have difficulties in processing three-dimensional depth cues, whilst being over-dependent on visual cues for controlling his actions. Similar difficulties in depth perception have been reported in accounts by people with autistic spectrum disorders (e.g. Grandin, 1995). Overall, the integration of discrete actions into a smooth coherent whole was a major difficulty for A, leading to problems with transitions and changes.

Behavioural intervention

A's difficulties in ascending and descending were particularly marked at his day service provision; it took him up to 3 hours to ascend a single flight of 18 stairs. A programme of cueing and environmental changes based upon the assessment was prepared with the day service and implemented using a single case experimental approach. This comprised a baseline phase of no intervention (10 sessions, each comprising an ascent and a descent of the stairs) and an intervention phase in which both environmental and interpersonal factors were manipulated on the basis of the assessment (15 sessions). The environmental factors manipulated were ensuring that all doors were wedged open at the top and bottom of the stairs; minimizing the use of the stairs by other people during the time A was attempting to ascend/descend; and providing mirrors to enable A to see around corners at the top and bottom of the stairs. Interpersonal factors were manipulated to direct A's attention to the necessary action for moving up/down each step. An instruction was given with a countdown for each action necessary (e.g. 'A puts right foot on stair, 1, 2, 3'; 'A turns left, 1, 2, 3') and A was given 30 seconds in which to process this information. This was based on the need to strengthen A's internal action plans. A verbal confirmation of 'well done' accompanied by a smile was given if the action was successful, the instruction being repeated if it was not. There was no other verbal or physical interaction with A whilst he was ascending/descending the stairs, save that necessary for safety reasons. The time to ascend/descend each step was recorded in seconds. A statistically significant difference in the time taken to ascend was found between the baseline and intervention phases (modal time for each step at baseline = 12 seconds; modal time per step post-intervention = 1 second; Mann–Whitney $U = 31.00$, $p = 0.019$). This

was also found for the modal times for descent which reduced from 75 seconds per step at baseline to 1 second per step post-intervention (Mann–Whitney $U = 0.50$, $p < 0.001$).

A's ability to ascend and descend stairs at a 'normal' speed subsequently generalized to other settings and has been maintained 18 months after the intervention was carried out.

Conclusions

We would concur with Realmuto and August's (1991) view that such autistic catatonic states are probably best considered as a particular intrinsic expression of the underlying autistic spectrum disorder, rather than as comorbidity. This would suggest that interventions for these behaviours should be derived from an understanding of individuals' sensory, perceptual and neurocognitive functioning, rather than from a diagnostic label of catatonia.

Two related central features of the presentation of autistic catatonia that require further investigation are the apparent impairments in the integration of external and internal information within controlling internal action plans, and the impaired integration of discrete 'chunks' of action into single continuous movement, both of which are time-dependent processes (Weardon, 1994). Boucher (1999) has proposed that impaired human timing systems, so-called 'biological clocks', may be involved in autism. One implication of this theory is that if a human timing system could only cope with very small 'chunks of time' (up to a maximum of a few seconds), longer and more complex sequences of behaviour could not be properly processed, but only experienced as 'unconnected mini-events'. Whilst Boucher's proposal remains wholly speculative, this model does provide a linkage with other neuropsychiatric models of catatonia which implicate subcortical timing dysfunction (Raitiere, 1986; Taylor, 1990). Interestingly, such hypothesized timing and movement difficulties seem to concur with the processes involved in the rare occurrence of catatonia following hypnosis (Kornfeld, 1985; Reeves et al., 1998).

At this stage, it would appear that intensive structured behavioural interventions, paying close attention to environmental and cognitive neuropsychological factors, seem to be the most appropriate forms of intervention and support for people with autistic catatonia. However, it is evident that much remains to be investigated, with regard to both aetiology and clinical support and interventions, particularly given the apparently raised prevalence of catatonic states in people with autistic spectrum disorders.

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