Treating Catatonia in Autism

January 01, 2008

In May 2007, the novelist Ann Bauer went public with the tribulations of her autistic son. When catatonia developed, a diagnosis of schizophrenia was made, and antipsychotic medications were prescribed, but with little benefit. When the catatonia syndrome was recognized as independent of schizophrenia and successfully treated, her son returned to a more normal life.1,2

Catatonia is an abnormal behavioral syndrome that is a feature of many psychiatric disorders and is remarkably responsive to available treatments. In the past decade, it has been increasingly identified in children and adolescents with autism. Compulsive self-injurious behaviors, prolonged staring, posturing, rigidity, mutism, negativism, echolalia, and echopraxia are features that overwhelm family life and schooling. Treatment markedly improves the child's and the family's quality of life.3

Surveys of psychiatric emergency departments and wards in academic hospitals find catatonia in 10% to 15% of patients. The syndrome is identified through structured examinations using lists of catatonic signs in behavior rating scales.4 Although catatonia is intimately tied to schizophrenia in psychiatric classifications, it is more often identified in patients with mania and depression and those in toxic states.4

The syndrome of catatonia was described by Karl Kahlbaum in 1874. A decade later, Emil Kraepelin incorporated it into his concept of dementia praecox. In the ensuing decades, this association of catatonia with schizophrenia was repeatedly endorsed, first by Eugen Bleuler, and then in successive iterations of the DSM, including those in 1980 and 1994.

The successful treatment of catatonia with amobarbital (Amytal) was first reported in 1930 and was quickly followed in 1934 by the demonstration that chemical-induced seizures also resolved the syndrome.5,6 The benzodiazepines lorazepam (Ativan) and diazepam (Valium) have since replaced barbiturates, and seizures are now routinely induced with electricity.

Both treatments efficiently reduce catatonia, usually within a few days of treatment. Neither treatment, however, is effective in classic schizophrenia and the treatments now offered for schizophrenia do not resolve catatonia, suggesting that the pathophysiology of catatonia is independent of schizophrenia.4

A systematic examination in 2003 brought together the forms of catatonia described as "retarded Kahlbaum syndrome," "delirious and excited mania," and "lethal febrile catatonia" (including neuroleptic malignant and toxic serotonin syndromes).4 The authors argued that the present classification of catatonia as a subtype of schizophrenia was in error and recommended that catatonia should be given a home of its own in the next classification, much like delirium and dementia.7

At a 2001 London conference on catatonia, the incidence of the syndrome in adolescents with autism spectrum disorders was reported to be as high as 17%.8 Case reports described effective relief with lorazepam and electroshock. The connection between catatonia and autism was strengthened by the compilation of experiences in Catatonia in Autism Spectrum Disorders by Dirk Dhossche of the University of Mississippi and other editors.3 In that collection, and in a 2006 article in Psychiatric Times,9 Dhossche offered guidelines to the clinical recognition of the syndrome and described favorable outcomes with specific treatments.

Autism spectrum disorders are abnormal behaviors that become apparent early in childhood. More severe forms disrupt education, maturation, and family life. No effective treatment is known, and children and families must learn to live with the disability, assisted by special education, family counseling, and symptomatic medications.

When catatonia is recognized, however, more specific relief can be offered. Catatonia that is grafted onto autism responds rapidly to treatment, with useful improvements in the child's and family's lives.3,4 The lesson of the experiences described here is that every child with autism should be carefully examined for the presence of catatonia, using a catatonia rating scale as a guide.4 A presumptive diagnosis is verified by a reduction in symptoms within ten minutes after the
intravenous administration of lorazepam. A positive test encourages definitive treatment.\textsuperscript{4,10} Catatonia

A common form of catatonia, dominated by mutism, refusal of food, and slowing of movements in an adolescent with autism and its resolution with electroshock was described by Ghaziuddin and coworkers.\textsuperscript{11}

\textbf{Case Vignette}

A 17-year-old boy with a history of autism, recurrent depressive illness, and mild intellectual disability was hospitalized with progressive worsening of mood and slowing of movements. Autism had been diagnosed at age 4, and he received educational, speech, and social-skill interventions throughout his school years.

A severe decline marked by mutism and reduced food intake began at age 14, when he became progressively slower in movement and had to be prompted to initiate activities of dressing and eating. He would stand or sit alone for prolonged periods, undertaking no spontaneous activities. Much time was spent in the bathroom washing his hands or looking in the mirror. Abnormal movements of blinking, eye rolling, and jerking of his neck were repeatedly observed.

When the patient returned from a few weeks at summer camp, he had lost weight, his speech had become sparse, and his movements and facial expressions were further slowed. Over the next four months, he spoke progressively less and exhibited frequent motor tics, rapid blinking, and head turning. Laboratory investigations did not find abnormalities of a general medical or neurological illness.

A diagnosis of obsessive-compulsive disorder and depressive mood disorder led to the prescription of citalopram (Celexa), risperidone (Risperdal), and ziprasidone (Geodon), with little benefit. The boy’s movements slowed further and were described by his parents as “watching a movie in slow motion.”

Catatonia was diagnosed, and while in the hospital he received a course of bilateral electroconvulsive therapy (ECT) with dramatic improvement in his movements, speech, appetite, and interpersonal interactions. He became more verbal, his movements were more spontaneous, and he was able to care for his activities of daily living.

Antidepressants were prescribed and improvement was maintained. His family considered his response to ECT lifesaving.

\textbf{Self-injury}

Repetitive compulsive acts that are self-injurious are frequent in young children with autism, as is shown in the following case report.\textsuperscript{12}

\textbf{Case Vignette}

DP, a 14-year-old boy with retardation, was admitted to a university inpatient facility for persistent head banging, which required him to wear a protective helmet and to be restrained most of the day. He also wore protective gloves and was sedated to control high-pitched wailing and prolonged screaming.

His mental age was measured at 4.3 years and he communicated by inarticulate guttural sounds. Head banging and skin scratching began when he was 10 years old and had persisted despite extensive treatment with medication and both positive and negative reinforcements. After four years of failed treatments, a trial of ECT was recommended.

On hospital admission, DP was receiving carbamazepine (Carbatrol, Tegretol, others), lithium (Eskalith, Lithane, Lithobid), and thiothixene (Navane). The lithium was discontinued, and carbamazepine and thiothixene were continued during a course of twice-weekly bilateral ECT. After the sixth treatment, screaming, scratching, and head banging were reduced. By the 10th treatment, DP no longer needed the helmet, gloves, or restraints. Treatments were reduced to once weekly, and after 16 treatments he was returned to his residence. For another two months, treatments were given once every two weeks until the psychiatrists were satisfied with his progress. Anticonvulsants and an atypical neuroleptic were prescribed and no life-threatening behavior was evident for two years.

The decrease in head-banging behavior in this patient with mental retardation is consistent with the improvement effected by ECT in other abnormal repetitive behaviors. Obsessive thoughts and compulsive acts, the hand wringing of agitated depression, the stereotyped motor acts of catatonia, and the self-inflicted injuries of persons with mental retardation all improve with ECT.\textsuperscript{3,11} These positive responses, however, were usually short-lived because treatment courses were curtailed as soon as the first signs of relief were recorded. The improvement was sustained in the above patient with continuation of ECT over four months.

\textbf{Conclusion}

Catatonia is a treatable syndrome that is increasingly recognized as being grafted onto autism. The
recognition of catatonia in autistic patients offers a treatable option to reduce the severity of the psychopathology and, in some cases, to bring sufficient resolution to markedly change the quality of patient and family life. It seems reasonable that every child with autism, especially those requiring inpatient care, be examined for catatonia. A positive diagnosis offers effective treatment. Additional case material can be found in reports by Bailine and Petraviciute, Dhossche and colleagues, Ghaziuddin and colleagues, Thuppal and Fink, and Wachtel and colleagues. 

(Please see Catatonia in Autism, page )


Source URL: [http://www.physicianspractice.com/articles/treating-catatonia-autism](http://www.physicianspractice.com/articles/treating-catatonia-autism)