THE IMPORTANCE OF CATATONIA AND STEREOTYPIES IN AUTISTIC SPECTRUM DISORDERS

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Motor disturbances are often observed in individuals with autistic spectrum disorders (ASDs) and recognized as diagnostic features of these disorders. The movement disorders characteristically associated with autism include stereotypies and self-injurious behavior. Yet, individuals with ASD may also be at the risk for catatonia. Although not as frequent as stereotypies, up to 17% of older adolescents and adults with autistic disorder may have severe catatonic-like symptoms. Catatonia may be a comorbid risk factor of autism that warrants further empirical and clinical evaluations. Clinicians may need to be attentive to more subtle signs of catatonic-like symptoms in individuals diagnosed with ASDs, especially as they enter adolescence and young adulthood. Stress has been implicated as a possible precursor for symptoms; however, its role has not been empirically proven as a potential risk factor. Clinicians might also need to assess for signs of significant declines in motor movements, as this appears to be a useful diagnostic indicator of catatonic-like symptoms. The literature on stereotypies and autism is more extensive than for catatonia and ASDs, probably because of the higher rate of stereotypies with autism. Explanations for the

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occurrence of stereotypies range from genetic to behavioral contingencies, with evidence for a multifactor explanation. Assessment measures often include items that assess for stereotypies to aid with diagnosing these symptoms in individuals with autism. Treatment for stereotypies is largely behavioral at the present time and requires consistent reinforcement of treatment gains to manage the symptoms successfully. An important area of future research in autism is the relation among different types of motor abnormalities, including stereotypies and catatonia.

I. Introduction

Motor disturbances are commonly linked to physical conditions, such as Parkinson's disease and Huntington's chorea, but can also occur with psychiatric conditions, including schizophrenia and autistic spectrum disorders (ASDs). Schizophrenia, for example, can be accompanied by catatonia and waxy flexibility that are diagnostic features of the subtype, catatonic schizophrenia. Catatonia also has been observed among individuals with autistic disorder. Research suggests that when present with ASDs, catatonia may represent a different clinical phenomenon that is referred to as "autistic catatonia." Although still in its infancy, research on this concept offers important implications for conceptualizing and treating individuals with autistic disorder. Hence, the literature on catatonia and autistic disorder is reviewed in this chapter. While the prevailing clinical data are mainly in the form of case reports, these can provide useful information for conceptualizing and treating individuals with autistic disorder who present with catatonia. Other movement disorders, specifically stereotypies, are also discussed because they are diagnostic features of ASDs and overlap in symptomatology with catatonia. Reviews of the clinical presentations for each type of motor disturbance will reveal similarities and differences that may aid with differential diagnosis.

II. Catatonia and ASD

Catatonia is a life-threatening condition marked by a paucity of motor movements, negativism, and mutism. In its severe form, catatonic symptoms can be expressed as episodes of excitement and restlessness superimposed on pervasive mutism, akinesia, catalepsy, and waxy flexibility. Wing and Shah (2000) described a minority of cases with autistic disorder and "catatonia-like deterioration" that includes marked catatonia-like and parkinsonian-like features. These

symptoms usually appear gradually and tend to eventually lead to significant deterioration in movement and self-care behaviors but rarely to a classic catatonic stupor. Wing and Shah (2000) observed that most of the 30 individuals with ASDs and catatonia exhibited slowness and difficulty initiating movements unless prompted, odd gait, odd stiff posture, freezing during actions, difficulty in crossing lines or cracks on the pavement, and an inability to cease actions. All of the individuals also showed a significant reduction in speech production or else mutism. Less pronounced and subtle symptoms included aberrations of posture, movement, speech, and behavior. These less severe symptoms are described as "catatonic in nature" because they do not necessarily match the classic symptoms of catatonia and, therefore, may represent a different condition. The diverse manifestations of the catatonic-like symptoms likely contribute to the general lack of agreement on the definition of this condition and its cardinal symptoms (Ungvari and Carroll, 2004). Although not classified as a separate diagnostic category in the Diagnostic and Statistical Manual of Mental Disorders, 4th ed., Text Revision (DSM-IV) (APA, 2000), catatonia does present with some unique features that have led to five features that are considered representative of catatonic disorder. These five symptoms include—motoric immobility, excessive motor activity, extreme negativism or mutism, peculiarities of voluntary movement, and echolalia or echopraxia. The features are based on clinical observations of adults and not of younger age groups. Although there are no standardized criteria for children or adolescents, Wing and Shah (2000) described features of catatonia that are characteristic of children and adolescents who may present with this disorder. These symptoms include—increased slowness affecting movement and verbal responses, difficulty initiating and completing actions, increased reliance on physical or verbal prompting by others, increased passivity and apparent lack of motivation, reversal of day and night, parkinsonian features, excitement and agitation, and increased repetitive, ritualistic behavior.

A. Epidemiology

Historically, catatonia had been observed in patients with schizophrenia. Consequently, most of the research conducted to date has been with populations diagnosed with psychotic conditions. Although epidemiological data reveal a wide range of prevalence rates for catatonic symptoms in individuals diagnosed with schizophrenia (4–67%), lower rates (7–17%) are reported for individuals hospitalized with more acute onsets of psychotic symptoms (Caroff *et al.*, 2004). Higher rates (up to 67%) have been observed in individuals diagnosed with other psychiatric conditions including depression, bipolar disorder, personality disorders, or organic brain disorder (Caroff *et al.*, 2004; Fein and McGrath, 1990).

The prevalence rate for severe catatonic-like deterioration appears to be rare, with 6% of 506 adolescents and adults with ASDs reportedly found to exhibit such symptoms across 7 years (Wing and Shah, 2000). These individuals comprised 17% of the patients over the age of 15 who participated in the study. The 6% prevalence rate for deteriorating symptoms is similar to the 7–10% prevalence rate reported for acutely ill psychiatric patients (Ghaziuddin *et al.*, 2005; van der Heijden *et al.*, 2005) but higher than the 1.3% rate reported for a cohort of psychiatric patients tested between 1990 and 2001 (van der Heijden *et al.*, 2005). Overall, it does not appear that individuals with autism are necessarily at a greater risk for catatonia than other psychiatric populations. However, catatonia may be observed more often in children with ASDs than in children with other developmental delays such as learning and language-disordered children (see Chapter 2 by Wing and Shah). Given that catatonia occurs in other psychiatric conditions and reaction to medications (Northoff, 2002; Takaoka and Takata, 2003), biological underpinnings that are not necessarily specific to autism may help explain symptom expression.

Observations of catatonic-like symptoms among individuals with ASDs are in sharp contrast to the excessive stereotypic behaviors that are considered diagnostic features of autism. However, some of the items reflecting catatonic-like features in Wing and Shah's Chapter 2 of pre- and school-aged children included stereotypic behaviors (e.g., runs in circles, body rocking, toe-walking, and spins objects), raising some questions about the overlap of catatonic and stereotypic symptoms in assessments. There tends to be a general bias to diagnose catatonia in its severe form rather than in more subtle manifestations, thereby increasing the risk of overlooking less salient symptoms. Finally, catatonia has historically been conceptualized as a type of schizophrenia in psychiatry and not investigated as systematically in individuals with ASDs as with other psychiatric disorders (Taylor, 2004; Ungvari *et al.*, 2005; van der Heijden *et al.*, 2005).

B. Characteristic Symptoms

To date, research on "catatonic autism" has largely consisted of case reports. These case descriptions share in common similar symptomatology including manifestations of immobility, extreme negativism, mutism, and peculiarities of voluntary movements co-occurring with episodes of echolalia and bursts of hyperactivity (Dhossche, 2004; Ghaziuddin *et al.*, 2005). In Wing and Shah's (2000) assessment of 30 individuals having more severe features of catatonia, a combination of catatonic-like and parkinsonian type of symptoms were revealed. All showed significant paucity of speech or complete mutism, and most exhibited slowness and difficulty initiating movements unless prompted, as well as odd gait

and stiff posture. Other symptoms included freezing during actions, difficulty crossing lines on the pavement, inability to cease actions, and impulsive and bizarre behavior. Less than half showed sleeping during the day, while staying awake at night, incontinence, and excited phases, and none of the individuals exhibited waxy flexibility. Many of these symptoms match Wing and Shah's list of essential features described earlier for diagnosing catatonia in children and adolescents. Hare and Malone (2004) have proposed three essential symptoms for "autistic catatonia" that are similar to Wing and Shah's (2000) description of symptoms, but that are restricted to a paucity of movement. These symptoms include freezing when carrying out actions and being resistant to prompting, very slow voluntary movements, and stopping in the course of movement and requiring prompting to complete actions.

C. Risk Factors

Risk factors for the development of catatonia-like symptoms in ASDs include a significant decline in motor movement (Ghaziuddin *et al.*, 2005). Age also appears to be a risk factor. All of the individuals with ASDs in Wing and Shah's (2000) study who manifested catatonia-like deterioration were either adolescents or adults; none were children. Case descriptions of individuals with autism who developed catatonia also manifested symptoms during adolescence or young adulthood (Dhossche, 1998; Ghaziuddin *et al.*, 2005; Realmuto and August, 1991; Zaw *et al.*, 1999).

The role of stressful events prior to the onset of catatonic symptoms is still open to debate. Parents of the individuals in Wing and Shah's (2000) study who exhibited catatonic-like deterioration reported a variety of precipitating events, ranging from school examinations to bereavement. All of these events were considered stressful for the individual; however, as noted by the researchers, a comparison group of adolescents and adults with autism had experienced similar events without developing catatonic-like symptoms. Wing and Shah (2000) also noted that there were no significant differences in the age, gender, IQ, history of seizure disorder, or in the diagnostic subgroup for autism noted between the individuals showing symptoms and the comparison control group. The most common risk factor identified in this study was passivity in social situations and impaired expressive language skills that predated the onset of catatonic symptoms. Case reports of adolescents with autism and catatonia also describe passivity with symptoms of depression, obsessive compulsive, and nonspecific psychotic symptoms preceding the onset of catatonia (Dhossche, 1998; Zaw *et al.*, 1999). Realmuto and August (1991) have suggested in their case description that these psychiatric conditions might account for the increased risk of catatonia among individuals with autism.

Depression is often noted as a cause of catatonic symptoms in general, yet it is unclear if this association can be generalized to individuals with ASDs. In their case report of an adolescent male with autism and catatonia, Ghaziuddin *et al.* (2005) suggest that obsessive slowing may be a precursor to catatonia in people with autism and that regressive behavior may result from the emergence of catatonic symptoms. While informative, further research is warranted before drawing definitive conclusions about possible risk factors for catatonia in individuals with autism.

D. Etiology

Etiological explanations for catatonia include a genetic predisposition, especially for periodic catatonia. At the molecular level, chromosome 15q15 has been strongly linked to this specific subtype. Research appears to support a single gene model and an autosomal dominant mode of inheritance with reduced penetrance (Stöber, 2004). However, family constellations as well as other psychosocial and environmental factors can modify the penetrance of the disorder.

Other hypothesized causes include structures of the brain including the frontal lobe (Taylor, 1990) and the basal ganglia (Rogers, 1991). Fricchione (2004) has implicated a network of systems that includes the basal ganglia–thalamo (limbic)–cortical circuits. Fricchione (2004) elaborated further on his hypothesis and suggested that a disruption in the gamma-aminobutyric acid (GABA) and dopamine (DA) balance likely contributes to the development of catatonia. Dhossche *et al.* (2002) also has implicated a dysregulation of GABA as a shared risk factor for both autism and catatonia. Hare and Malone (2004) argue, however, that catatonic symptoms are expressions of autism and should not necessarily be conceptualized as comorbid symptoms. Hence, they suggest that sensory, perceptual, and neurocognitive systems underlying the cause of autism might explain catatonia in ASDs. Any hypothesis is mere speculation at this point, pending further empirical evaluations.

E. Assessment

Although not specific to catatonia, measures of autism including the Autism Diagnostic Interview-Revised (ADI-R) (Lord *et al.*, 1994) and the Childhood Autism Rating Scale (CARS) (Schopler *et al.*, 1988) include questions about psychomotor movements that could be diagnostic of catatonia. It is important to note that these items were neither developed specifically for this purpose, nor were they intended to be used to diagnose catatonia. Some of the items, characteristic of catatonia, are also characteristic of autism (e.g., posturing), thereby increasing the risk for misdiagnoses. To help with differential diagnosis,

Ghaziuddin *et al.* (2005) suggest that the key issue in diagnosing catatonia in autism is the emergence of "new" symptoms or a "change" in the type and pattern of premorbid functioning.

One measure of ASDs, the Diagnostic Interview for Social and Communication Disorders (DISCO), includes 28 items that specifically address catatonic-like features. Caregivers describe each type of behavior listed and give examples to aid the interviewer with the assessment of symptoms. Information from the interview, direct observation, and data from other available sources are considered to rate the presence and severity of the behavior. Wing and Shah (see Chapter 2 by Wing and Shah in this volume) found that children with autistic disorders were rated with more marked and moderate problems on the 28 items than children with learning disabilities or a specific language disorder and a normally developing group of children. There was no significant difference between the groups of children with autistic disorder where IQ scores were either above or below 70.

Over the past two decades there has been a growing interest in developing standardized measures specifically for diagnosing catatonia (Mortimer, 2004). While many of these measures have been used to assess for catatonia in patients with a diagnosis of schizophrenia or a mood disorder, the psychometric properties of these measures have not been adequately assessed with other psychiatric populations. The Modified Rogers Scale (Rogers *et al.*, 1991) is one such measure that has been found to distinguish reliably between depressed individuals with and without catatonia, as well as between depressed individuals with catatonia and individuals with Parkinson's disease. Items with the best specificity from the scale have been extracted and were renamed the Rogers Catatonia Scale (Starkstein *et al.*, 1996). Factor analysis of this scale has revealed two primary factors, hypokinetic and hyperkinetic, that account for approximately 64% of the variance (McKenna *et al.*, 1991).

A second measure of catatonia, the Bush–Francis Catatonia Rating Scale (BFCRS) (Bush *et al.*, 1996) also appears to be a reliable and moderately valid measure of catatonia. While some measures of catatonia assess the presence or absence of symptoms, the BFCRS measures the severity of 23 signs of catatonia (e.g., mutism, grimacing, echolalia, impulsivity, and combativeness). A truncated 14-item version is available as a quick screener. However, this measure has not been evaluated for use with children or with individuals with autism, thereby limiting its clinical utility with certain populations.

F. Treatment

Research on treatment is largely limited to case reports and there have been no empirically based treatment outcome studies that would aid in the development of evidence-based practices for the treatment of catatonia in autism.

Consequently, pharmacological agents are often utilized to treat accompanying depressive, regressive, and psychotic symptoms, but these are not always successful for addressing the catatonic symptoms, especially in severe cases (Dhossche, 1998; Ghaziuddin *et al.*, 2005; Realmuto and August, 1991). Electroconvulsive therapy (ECT) has been used with some success, but this option is reserved for the most severe and life-threatening cases (Ghaziuddin *et al.*, 2005; Zaw *et al.*, 1999). For an in-depth discussion of treatment issues, refer to the Treatment Section (Section III) in this book.

III. Stereotypic Movement Disorder

Stereotypic movement disorder (SMD) is characterized by repetitive nonfunctional motor or vocal responses that are severe enough to interfere with psychosocial functioning or to cause physical injury (APA, 2000; LaGrow and Repp, 1984). Although recognized as a separate diagnostic category, stereotypic movements can occur with pervasive developmental disorders (PDDs), obsessive-compulsive disorder (OCD), hair pulling, and tic disorders. If occurring with these disorders, then SMD is not diagnosed.

A. Epidemiology

Stereotypies are most often seen in individuals with mental retardation (25%) and autism (85%) (Volkmar *et al.*, 1986); however 2–3% of the general population of children and adolescents (APA, 2000; Rojahn *et al.*, 1998) and up to 15–20% of pediatric populations (Matthews *et al.*, 2001) exhibit stereotypic behaviors. Some of the more common stereotypies observed among individuals with autism include rocking (65%), toe-walking (57%), arm, hand, or finger flapping (52%), and twirling (50%) (Volkmar *et al.*, 1986). These behaviors are often the focus of clinical attention because they can interfere with learning and the application of learned skills (Morrison and Rosales-Ruiz, 1997). Early intervention is strongly recommended as they tend to be prognostic of more severe self-injurious behaviors (Guess and Carr, 1991; Schroeder *et al.*, 1990).

The age of onset for stereotypic behaviors is typically toddlerhood. Symptoms generally remit by age 5 in normally developing children but persist in children with developmental delays, reaching a peak in adolescence. Stereotypies tend to decline gradually thereafter with the exception of adults with severe or profound mental retardation who can show symptoms for years. In its most severe form, SMD can result in physical injuries, as in the case of self-biting, head banging, scratching, and hair pulling, if untreated.

B. Etiology

Since stereotypic behaviors are closely intertwined with autism, biochemical abnormalities implicated in autism may be pertinent for understanding the biological basis for stereotypical behaviors. As with many other psychiatric conditions, serotonin (5-HT) and the 5-HT system have been linked to the development of autism. Serotonin is involved in many aspects of human behavior including sleep, pain, motor function, appetite, and others (Volkmar and Anderson, 1989). Research has consistently revealed that up to 50% of individuals with autism are hyperserotonemic (Geller *et al.*, 1982). The mechanism through which 5-HT influences the symptoms of autism, however, is still unknown. Findings specifically related to repetitive and stereotypic behaviors suggest that decreases in 5-HT levels are related to *decreased* stereotypies. Consistent with this hypothesis, Curzon (1990) found that rats administered with agonists for 5-HT activity engaged in behaviors that closely resemble stereotypies. However, contrary to this finding, others have reported a decrease in stereotypies when 5-HT is inhibited from being reabsorbed by the presynaptic neuron (Powell *et al.*, 1997). Similarly, McDougle *et al.* (1996) observed an exacerbation of stereotypic behavior among a sample of adults with autism after 5-HT was reduced through the depletion of its precursor, tryptophan. This may be explained by the effects of central versus peripheral 5-HT that may differentially influence the manifestation of stereotypic behaviors.

Opioids have also been implicated in the development of self-injurious and stereotyped behaviors. According to the opioid hypothesis, when individuals engage in self-injurious behavior (SIB), the brain releases neurochemicals, such as endorphins, that block pain and produce mild euphoria. Although this may seem paradoxical, it is believed that continued self-injury actually blocks the painful stimulation that it would ordinarily produce, contributing to the maintenance of this behavior. Individuals who engage in SIB and other stereotypies may be motivated by sensations of euphoria and escape or avoidance from painful stimulation. Using animal models, researchers have observed higher rates of SIB and stereotypies after administrations of opiate agonists and lower rates after administrations of opiate antagonists (Dantzer, 1986; Iwamoto and Way, 1977). Both Campbell *et al.* (1993) and Rojahn *et al.* (1998) reported similar observations with humans, however, the declines in stereotypies observed by Campbell *et al.* (1993) after administering naltrexone, an opiate antagonist, did not reach statistical significance.

Research has examined the interaction between opiates, 5-HT, and the DA system to explain autism, and more specifically stereotypies and SIB. Stereotypical behaviors observed among animals, head weaving, for example, are noticeably less frequent following lesions to the nigrostriatal and mesolimbic DA pathways and after the administration of DA antagonists (Lewis and Bodfish,

1998). Terminal fields that receive substantial amounts of DA innervation also contain large amounts of opioid peptides and receptors, suggesting an interaction effect between the opioid and DA systems (Angulo and McEwen, 1994). Another hypothesis, the DA supersensitivity hypothesis, proposes that repetitive behaviors and SIB result from low levels of DA in postsynaptic cells of the basal ganglia, resulting in the supersensitivity of the postsynaptic receptors. The presence of small amounts of DA subsequently produces activation. Support for this hypothesis includes animal models of self-injurious and stereotypic behaviors in which these behaviors are induced following the administration of DA agonists such as L-dopa (Lewis and Baumeister, 1982). Depriving animals of sensory stimulation and restricting their interactions with the environment in controlled laboratory experiments has been found to prevent DA innervation and subsequently produces spontaneous stereotypies (Martin *et al.*, 1991). Suomi and Harlow (1971) observed similar behaviors in nonhuman primates who had experienced early social deprivation. It appears that early deprivation or restricted environmental interaction results in a loss of DA innervation of important brain regions that results in DA receptor supersensitivity (Lewis and Bodfish, 1998).

Research linking structural parts of the brain to stereotypies have revealed some abnormalities in the cerebellum and the neuronal systems that are directly influenced by the cerebellum, including those that regulate attention, sensory modulation, autonomic activity, and behavior initiation (Courchesne *et al.*, 1988). Further research using functional magnetic resonance imaging may prove useful for specifying the underlying mechanisms involved.

Nonbiological theories for explaining stereotypies include behavioral theories in which the repetitive behaviors are reinforced by contingencies. These contingencies may be: (1) positive internal reinforcement such as sensory stimulation, (2) positive external reinforcement such as social attention, or (3) negative reinforcement—the removal of aversive stimuli. According to the sensory stimulation hypothesis, the stereotypy is maintained by access to reinforcing sensory and perceptual stimulation that may be a by-product of the stereotypic behavior itself (Lovaas *et al.*, 1987). Repetitive rocking or twirling, for example, may be maintained by vestibular stimulation or eye poking may be reinforced by visual sensations. Iwata *et al.* (1994) refer to the maintenance of these behaviors as *automatic reinforcement* because they are not socially mediated.

The positive reinforcement hypothesis focuses on the social consequences maintaining the stereotypic response. Attention or tangible rewards, such as toys or food, contingent upon these behaviors are hypothesized to reinforce the stereotypic response. The consequences need not necessarily include the receipt of desired rewards but can also involve the removal of aversive stimuli (Iwata *et al.*, 1994), in which case the respondent is reinforced negatively for manifesting stereotypic behaviors. Support for socially mediated consequences include observations of higher rates of rocking and hand-flapping among children with

autism and other PDDs when confronted with a difficult task. These behaviors remit or are reduced upon the removal of the task. Rather than arguing for either the sensory stimulation or the behavioral hypothesis as causal explanations, both theories are supported empirically. Dawson *et al.* (1998) observed individuals with autism tended to engage in stereotypies for the purpose of sensory stimulation, as well as to remove aversive work or social stimuli.

A third etiological hypothesis for stereotypic behavior is the communication-based theory. Similar to behavioral hypotheses, stereotypic responses serve as a means to communicate one's needs, including eliciting a desired response from another, acquiring a desired object, or removal of an aversive environmental stimulus. This explanation may be more specific to children with communicative disorders that preclude them from using language productively. Such children have learned to use stereotypic behaviors as a means of communication to compensate for their language impairment. Support for this hypothesis includes the successful application of treatments designed to teach children sign language to communicate in lieu of engaging in stereotypic behaviors (Kennedy *et al.*, 2000). Others have suggested, on the other hand, that stereotypies are associated with cognitive impairment (Volkmar and Lord, 1998). However, Matson *et al.* (1996) found a significantly lower percentage of adults with severe or profound retardation (7%) exhibited stereotypies compared to adults with the dual diagnoses of severe or profound mental retardation and autism (75%). In addition, children with more severe manifestations of autism have been found to show a tendency to exhibit more severe stereotypical behaviors (Campbell *et al.*, 1990).

C. Assessment

Many of the assessment measures for autism include items pertaining specifically to stereotypic behavior. The ADI-R, for example, is a structured interview that assesses functioning in three domains mirroring the diagnostic criteria for autism, including the "restricted, repetitive, and stereotyped behaviors and interests" domain (Lord *et al.*, 1994). Items in this domain include repetitive motor movements and SIB. The CARS is another measure of autism that incorporates historical interview information from the parent and direct observation by a professional who rates the child's behavior in 15 domains (Schopler *et al.*, 1988). An item pertaining to "body use" measures the severity of stereotyped behaviors such as repetitive movements, rocking, spinning, and self-injury. An item pertaining to "object use" addresses inappropriate use of objects that may include stereotyped use. A third measure, the Autism Behavior Checklist (ABC) (Krug *et al.*, 1980, 1993), is a 57-item parent- or teacher-rating scale that is included as part of the Autism Screening Instrument for Educational Planning-2. One of the five subscales, "Body and Object Use," contains several items

measuring stereotypic motor movements, such as whirling, rocking, spinning, and flapping, as well as stereotyped use of objects (e.g., spinning or banging objects).

Clinicians may need to be cautious when using these measures with younger children because of potential problems with overidentifying children with mental retardation as exhibiting autism (Rutter and Schopler, 1987; Wing and Gould, 1979). Both groups exhibit language delays, social impairments, and some evidence of restricted, repetitive, and stereotyped behaviors that can complicate the process of making differential diagnoses (Vig and Jedrysek, 1999; Wing and Gould, 1979). The problem with overidentification is further complicated by the fact that mental retardation can co-occur in 70–80% of children with autism. Some of the autism assessment measures, including the CARS and ABC, have also been criticized for failing to accurately diagnose children under the age of 3 (New York State Department of Health, 1999). Using the ADI-R and CARS, for example, has led to the overidentification of very young (under 2 years) and mentally retarded children (mental age < 18 months) as being autistic (DiLavore *et al.*, 1995).

The Diagnostic Assessment for the Severely Handicapped II (DASH-II) is not a measure of autism specifically, but it does include two scales for movement disorders—stereotypies and SIB. Matson *et al.* (1997) evaluated the measure with 289 individuals with severe and profound mental retardation. The stereotypies and SIB scales correctly identified 32% of individuals with stereotypies, 94% with SIB, 75% of individuals exhibiting both, and 100% of controls, as based on DSM-IV criteria. The overall classification rate was 83%. In a second study of over 1000 individuals with severe or profound mental retardation, certain items were more characteristic of individuals with SMD and others for SIB. Repetitive body movements, limited sets of preferred activities, and repetitive words or sounds were most likely characteristic of SMD, whereas self-biting and picks at wounds differentiated the SIB group from the SMD and control groups. Although diagnostic, two to three items are not generally considered sufficient for diagnosing a syndrome per se.

D. Treatment

Treatments for stereotypies are generally behavior specific and based on behavioral functional analyses used to identify and remove or prevent reinforcing contingencies. Repeated episodes of the stereotypic response without contingent responses are necessary before the stereotypy is extinguished. Individuals who bite their arms, for example, might be outfitted with arm braces or casts to prevent them from garnering any self-stimulation from biting themselves. Aversive conditioning, including the application of contingent noxious smells and

tastes (e.g., ammonia, lemon juice), have been used successfully to reduce stereotypies and SIB in children who are unresponsive to less restrictive contingent management approaches (Stoppelbein and Greening, 2005a,b). Treatment gains tend to be situation specific and, therefore, require that contingencies be applied across settings and caregivers. Less restrictive approaches should be exhausted before resorting to aversive therapies because of ethical concerns about using noxious stimuli in treatments.

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