

Brief Report: Pervasive Developmental Disorder Can Evolve into ADHD: Case Illustrations

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Despite prominent attentional symptoms in Pervasive Developmental Disorders (PDD) the relationship between PDD and Attention Deficit Hyperactivity Disorder (ADHD) has received little direct examination. In addition, outcome studies of children with PDD often focus on language, educational placement, or adaptive skills, but seldom on loss of the PDD diagnosis or change to another clinical syndrome. We present three cases in detail, and tabular data on eight more, that illustrate a clinical presentation in which prototypical cases of PDD evolve into clear-cut cases of ADHD from early to middle childhood.

KEY WORDS: ADHD; autism; loss of the PDD diagnosis; outcome; PDD.

INTRODUCTION

Pervasive Developmental Disorders (PDD) and Attention Deficit Hyperactivity Disorder (ADHD) have some cognitive and behavioral features in common, but the relationship between them has been seldom explored directly. Furthermore, outcome studies of PDD have focused on educational and vocational placements, adaptive skills, cognitive levels, and sometimes loss of diagnosis but seldom on change in diagnosis to another syndrome. We present three cases in detail, and tabular data on eight more, that illustrate a clinical presentation in which prototypical cases of PDD evolve into clear-cut cases of ADHD from early to middle childhood.

Despite some obvious commonalities between ADHD and autism spectrum disorders (Gillberg, 2003b; Kennedy, 2002), the nature of the relationship

between these sets of disorders has rarely been directly examined. Symptom overlap has been demonstrated in several studies. Hyperactivity and inattentiveness are so frequently observed in children with PDD that a diagnosis of ADHD is precluded if the hyperactivity and inattention can be attributed to a PDD disorder (APA, 1994); this overlap has been substantiated by McGrath, Joseph, Tadevosyan, Folstein, and Tager-Flusberg (2002) and Goldstein and Schwebach (2004), who found a high degree of ADHD symptomatology in a sample of children with autism. The reverse claim is more controversial: Rutter and Yule (1994) found autistic symptoms to be rare in hyperactive children, but Gillberg (1992) found mild autistic symptoms to be common in children with ADHD, and Clark, Feehan, Tinline, and Vostanis (1999) found a high rate of autistic symptoms reported by parent checklist in children with clinically diagnosed ADHD. Recently, Frazier *et al.* (2001) examined a series of children with PDD only, ADHD only, and symptoms of both ADHD and PDD. They found that the attention and hyperactivity symptoms in the PDD children were similar to those in the children with ADHD alone, and that the PDD symptoms were similar in children with and without attention problems and hyperactivity.

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They concluded that the disorders are frequently truly comorbid and that PDD should not be an exclusionary criterion for a diagnosis of ADHD.

Some individuals with autism respond well to ADHD medications, not only with benefits to attention and hyperactivity, but possibly with improvements in core features of autism, such as stereotyped behaviors and inappropriate language (Hamden, 2000; Towbin, 2003). Cognitively, executive functioning has been shown to be impaired in both clinical groups (Pennington & Ozonoff, 1996), although findings conflict about whether the nature of these deficits differs in the two groups (McGrath *et al.*, 2002; Ozonoff & Jensen, 1999) and executive dysfunction is a nonspecific finding common to many clinical groups.

Abnormalities in dopamine function have been implicated in both disorders. Genetic and animal model research on ADHD suggests dopamine abnormalities (Cabib, Puglisi-Allegra, & Ventura, 2002; Russell, 2002; Vigiano & Sadile, 2002; Wigg *et al.*, 2002). Individuals with ADHD have been found to have abnormally high levels of both dopamine metabolites (Oades, 2002) and dopamine transporters (Madras, Miller, & Fischman, 2002). Many symptoms of ADHD respond to stimulant medication, which blocks the uptake of dopamine and facilitates its release (Solanto, 2002). Neurochemical research on autism has also suggested a role for abnormalities in dopamine systems (Damasio & Maurer, 1978; Volkmar, 2001). A recent review (Green-Snyder *et al.*, 2003) concluded while there is no simple picture emerging of the role of DA in autism, there is evidence that dopamine may play a role in autism as well as in ADHD.

Kinsbourne (1991) presented one of the few theoretical models relating ADHD to autism, including a role for DA. He postulated a clinical syndrome that includes elements of both PDD and ADHD: the overfocused child, marked by a narrow focus of attention and social withdrawal. This syndrome is viewed as a set of behaviors serving to defend against an unstable arousal system. The syndrome of overfocusing shares symptoms with autism, namely, perseverative and narrowed interests, repetitive movements, and social impairment. The overfocused child, however, does not have language or cognitive impairments, and is capable of warm social attachments. Kinsbourne (1991) views these disorders as possibly lying on a continuum of arousal and stimulus-seeking involving mesolimbic and nigrostriatal DA functioning, from ADHD to normal to overfocused to PDD.

The postulated syndrome of DAMP (deficits in attention, motor control, and perception) elucidated

by Gillberg (2003a) is another clinical picture in which deficits in attention and motor control (i.e., ADHD plus Developmental Coordination Disorder) predict the presence of some autistic symptoms (Gillberg, 2003b). Therefore, although these children do not necessarily show autistic symptoms or meet criteria for any autistic spectrum disorder, and although family patterns in DAMP and ASD may be different (Gillberg, Gillberg, and Steffenburg, 1992), DAMP may represent another group of children where overlap between ADHD and ASD symptomatology can be seen.

Very few studies have examined outcome for young children with PDD in terms of diagnostic stability. Cox *et al.* (1999) found that of 9 children with PDD at 20 months, all retained the diagnosis at 42 months. Looking over a longer time span, however, McEachin, Smith, and Lovaas (1993) reported that of 19 children diagnosed with autism around 34 months and given intensive behavioral therapy, 8 achieved normal intellectual and educational functioning by age 7, and were indistinguishable from peers with no history of behavioral disturbance according to measures of intellectual performance, educational placement, adaptive and social skills, and subjective clinical impressions when assessed at ages ranging from 9 to 19. To our knowledge, no studies have reported on cases of PDD that evolved into another distinct clinical syndrome.

The purpose of the present paper is to present a series of cases demonstrating a clinical presentation that so far may be unreported: clear cases of autism or PDD-NOS that evolve into clear cases of ADHD from early to middle childhood. These cases have been drawn from the clinical practice of the first and fourth authors, a neuropsychologist and an OT, both specializing in autism. Three cases are presented in detail below and data from 8 additional cases are presented in Table I (detailed descriptions of the 8 cases are available from the first author).

The diagnosis of a PDD disorder was, in all cases, made by the first author, a pediatric neuropsychologist with more than 25 years of research and clinical specialization in autism and related disorders. The diagnoses were made on the basis of behavioral observation, neuropsychological testing, extensive parent interview, and frequently teacher interview, using DSM-IV criteria. In some cases (described in the case studies), the initial diagnosis was made by another psychologist, a neurologist, or a psychiatrist; these were all confirmed by the first author when she first evaluated the child. Diagnoses of ADHD-I,

Table I. Clinical Characteristics

Case	Gender	Age in years: Diagnoses	Regression	Early dev.	Main treatment	Medical Hx and medications	ADHD type	IQ	Family Hx of PDD or ADHD	Residual Sx of PDD
AA	Boy	2: PDD-NOS 6: ADHD-C	16-22 mos., lang., play, eye contact	NI till 16 mos.	ABA	Ear infections	C	Verbal = Average Perform = Borderline	None Disclosed	Immature peer relationships, occasional perseverative play, jumping and flapping when excited
BB	Boy	3: PDD-NOS 11: ADHD-I	18-24 mos., social, lang., play	NI till 18-24 mos.	Integrated preschool	Ear infections	I	Verbal = Average Perform = Average	Brother- possible ADHD	Immature peer relationships, perseveration on preferred activities
CC	Boy	3: AD 5: PDD-NOS 7: ADHD-I	15-18 mos., lang., eye contact, stereo, resistance to change	NI till 15-18 mos.	ABA	Vocal and motor tics	I	All subtests Average or Above Average	ADHD in several 1 st degree relatives	Mild perseverative interests
DD	Boy	3: AD 4: PDD-NOS 6: ADHD-C 9: ADHD-C	24 mos., social and play	Language delay	ABA	Ear infections, myoclonic sz; Adderall	C	Verbal and Perform = Low Average Range	None Disclosed	Some persev. play, impulsive socially, hand flapping difficulty
EE	Girl	3: AD 5: PDD-NOS 9: ADHD-I	wds lost 12 mos., imitation 2nd year	few words, lost around 1 year	ABA	food allergies, ear infections, Ritalin	I	Verbal and Perform = Average	None Disclosed	sustaining a conversation, toe walking
FF	Boy	4: PDD-NOS 5: AD 7: PDD-NOS 9: ADHD-C	Around 2 years, social, lang., behavior	nl till 2	Half day preschool with tutor age 4	Ear infections	C	Verbal and Perform = Average	None Disclosed	Mild res. to change, delayed lang. compreh.
GG	Boy	5: ASD 8: ADHD-C	No	no concerns till 2, poor social, sensory	Special ed preschool	Ear infections, Clonidine for rages	C	All subtests Above Average to Superior	None Disclosed	Difficulty with peer relationships, preoccupations with topics
HH	Girl	1: PDD-NOS 2: PDD-NOS 4: ADHD-C	13 mos., eye contact, imitation, lang.	nl till 13 mos.	ABA	Ear infections	C	All subtests Below Average to Average	None Disclosed	Mild social awkwardness

Table I. (Continued)

Case	Gender	Age in years: Diagnoses	Regression	Early dev.	Main treatment	Medical Hx and medications	ADHD type	IQ	Family Hx of PDD or ADHD	Residual Sx of PDD
JJ	Boy	3: PDD-NOS 4: PDD-NOS 5: ADHD-I	12-14 mos., mood and eye contact	lang. delayed	ABA	Ear infections, low tone	I	Verbal and Perform = Average	None Disclosed	Social inflex and poor grasp of rules
KK	Boy	2: ASD 3: ASD 4: PDD-NOS 5: ADHD-I	no	Motor OK, lang. delayed	ABA	Ear infections	I	Verbal and Perform = Average	None Disclosed	Limited comp. of social rules, problems with sustained interactions, occasional repetitive play
LL	Boy	2: PDD-NOS 5: PDD-NOS 6: PDD-NOS 8: ADHD: I	15-18 mos., eye contact, no progress in lang.	Single words 12-15 mos.	Speech, playgroup, ABA	Ear infections, asthma, hay fever	I	Verbal and Perform = Average	None Disclosed	Mild difficulty in sustaining conversation

Key for abbreviations: ABA = Applied Behavioral Analysis; AD = Autistic Disorder; ASD = Autistic Spectrum Disorder; C = combined type; I = inattentive type; nl = normal; PDD-NOS = Pervasive Developmental Disorder - Not Otherwise Specified; stereo = stereotypies; sz = seizures.

ADHD-H, and ADHD-C were also made by the first author. These were also made on the basis of testing, behavior observation, parent interview and frequently teacher interview. Diagnoses were made using the ADHD Rating Scale-IV (DuPaul, Plower, Anastopoulos, & Reid, 1998); interviews were used to confirm that the behaviors were demonstrated in more than one setting (usually school and home, but sometimes church or organization such as Brownies) and were significantly interfering with adaptive skills and social adjustment. In all cases, the symptoms qualifying the child for an early diagnosis on the PDD spectrum, and the features qualifying him or her for a later diagnosis of ADHD are described below.

Some of the children displayed mild residual symptoms of PDD at outcome; these are listed in Table I. It should be stressed that in all cases, these features were minor and only displayed occasionally; none of these children would have qualified for a diagnosis of even mild PDD-NOS.

Case 1

History

AA was born at 37 weeks gestation after a pregnancy marked by high blood pressure and rupture of membranes during an amniocentesis at 16 weeks followed by bed rest. Early motor milestones were normal. He began to use single words at 12 months, but between 16 and 22 months showed a gradual regression in communication and social skills. Eye contact decreased, play skills and imitation declined, and he lost the use of many of his words. Testing at 24 months showed cognitive skills at the 10-month level and language skills at the 7-month level. He showed a lack of interest in interactive play, hand flapping and other repetitive behaviors, staring off into space, and he was described as remote from familiar people. He would occasionally poke himself in the eye, bite his hands, or carry around an object in each hand. He received the diagnosis of PDD-NOS from a pediatric neurologist and a child psychologist, both of whom specialized in autistic disorders.

From the age of 2–7 through 5 years, AA was involved in an intensive Applied Behavior Analysis (ABA) program with additional occupational and speech/language therapy. By age 3, he was able to follow some two-step directions. By 3–5, he was using 2-word phrases and by 4 years he was speaking in full sentences and asking 'wh' questions. He began to initiate play with another child at 4 years and pretend

play emerged at 5 years. AA attended an ABA-based integrated preschool for 2 years and was included in a typical kindergarten for the following 2 years, from age 5–8 through age 7–6; he then transitioned to a regular first grade.

Medical History

AA's history was significant only for recurrent ear infections. From age 3–6 to 5, he was on a gluten and casein-free diet, which parents felt was associated with an increased rate of language development and firmer stools. One dose of secretin had no effect on his behavior. Sleep, appetite, vision, and hearing were normal. Mother reported that fevers were associated with fewer atypical behaviors and better conversational ability.

Current Presentation

An evaluation at age 6–8 that included cognitive testing showed nonverbal ability in the borderline range and verbal skills in the average range with his lowest ability in the area of Arithmetic. Vineland Communication was average but Daily Living and Socialization were low for his age. He showed age appropriate toy play, greeting, sustained eye contact with the examiner, and separation from mother. He was consistently interactive but his high activity level and impulsivity interfered with his social exchanges. He engaged in some limit testing (flopping to the floor, attempting to leave), but he responded well to limit setting, rules, and a reinforcement schedule. He was motivated by social praise and showed pride in success, but gave up easily when tasks became difficult. He needed ongoing prompts to stay on task and was highly distractible by external stimuli. Hand flapping and pacing were seen when he became agitated. Mother described immature relationships with peers; although he engaged in reciprocal pretend play with peers, his impulsivity and difficulty with perspective taking interfered with sustained play. His fantasy play was often creative and novel, but tended to focus on aggressive themes, sometimes perseveratively. When very excited, he would sometimes still jump and flap his hands. Joint attention was good, and he was attuned to others' emotions, although his responses were on some occasions prosocial and on others immature and 'silly'.

On interview, his mother endorsed eight of nine behaviors indicative of inattentiveness (failing to give close attention to details, difficulty sustaining attention, not listening when spoken to, not following

through on tasks, difficulty organizing tasks, frequently losing things, easily distracted, and forgetful). Six of the nine behaviors indicative of hyperactivity were also present (fidgeting, leaving his seat, difficulty playing quietly, often 'on the go', talking excessively, difficulty waiting his turn, and interrupting others). AA therefore met criteria for ADHD-Combined Type and no longer met criteria for any form of PDD. Mild residual PDD behaviors included poor peer relationships (although characterized by immaturity and impulsivity rather than aloofness or 'one-sidedness'), occasional perseverative play (but marked by impulsivity and aggression) and rare motor stereotypies (jumping and flapping) when excited.

Case 2

History

CC was born after a 42-week pregnancy marked by some vaginal bleeding at the end of the third trimester. Labor, delivery, and infancy were uneventful. Early motor milestones were achieved at the typical times. Toilet training was completed at age 3–6. Language development was delayed and atypical. A few single words that had been acquired before 15 months disappeared around that time. Single words were not used consistently until age 2 years and consisted only of labels; they were not used to request. Attention to language and comprehension of simple commands were poor. At the same time that language declined (15–18 months), eye contact also decreased and motor stereotypies and resistance to change appeared. An intensive ABA program was begun just before age 3. His vocabulary quickly improved and sentences emerged within a few months, although language remained echolalic for several years. Interest in peers was very limited, but typical peers were instructed to persist in their initiations to him, and reciprocal play with peers gradually emerged between ages 3 and 5. His ABA program also aggressively worked on tolerance for new situations and changes in routine. At age 3, therefore, he met criteria for Autistic Disorder, showing poor eye contact, poor joint attention, poor peer relationships, lack of emotional reciprocity, delayed language, echolalic and perseverative language, poor pretend play, and resistance to changes in routine.

An evaluation at age 5 years noted that CC frequently avoided eye contact and when prompted, eye contact was sometimes fleeting. When attention was engaged in an activity, eye contact was more

appropriate. Some phrases were scripted or echolalic, but other language was more appropriate and flexible. He was overfocused on his own activities and needed to be prompted to attend to other people. He engaged in elaborate symbolic play but did not respond to the examiner's attempts to join his play. He was compliant on testing. He was distractible but could be easily redirected. He did not persist in problem solving when tasks became difficult and was more motivated by edible reinforcements. In his preschool, he was responsive to other children's social bids and could sustain brief social interactions with them. Resistance to change, which was very aggressively worked on in the two previous years, had subsided. It was concluded at this time that CC met criteria for PDD-NOS (impaired eye contact, lack of emotional reciprocity, impaired language comprehension and a tendency to echolalia, and mildly perseverative interests). Most cognitive skills were within the average range.

Medical History

At age 2, staring spells that resembled absence seizures were noted but an EEG was normal and his neurologist did not diagnose a seizure disorder. Tics emerged between age 5 and 9, with both vocal and motor tics appearing and receding from time to time. Family history was positive for ADHD.

Current Presentation

At age 7–6, another evaluation noted good eye contact, appropriate greeting and conversation with the examiners, continuous social engagement, and good comprehension. All cognitive and adaptive skills were in the average or high average range. Despite an above average overall performance on the Wisconsin Card Sort, he showed a marked tendency to lose set. Family reported that CC's social relationships with peers were quite good; he had a best friend and was well liked by the children and teachers. He had no supports in his first grade class and the teachers were unaware of his history or prior diagnosis. In the structured environment of the testing room, CC's attention and perseverance were good. In school and at home, however, attention was problematic. Teachers reported that he had a tendency to get 'silly' when tasks became difficult in school and was considered markedly distractible, but was not seen as disruptive or destructive. His activity level was considered high and it was difficult for him to remain seated in school. He had difficulty sustaining mental effort and disliked learning tasks that

required concentration. On interview, CC's mother endorsed seven of nine features of inattention (failing to give close attention to details, difficulty sustaining attention to tasks, does not follow through on instructions, difficulty organizing tasks, avoids tasks that require mental effort, easily distracted, forgetful) and two of the nine hyperactivity behaviors (fidgets, difficulty playing quietly). He therefore met criteria for ADHD-Inattentive Type. There was still some mild residual tendency to perseverative interests and being overfocused on preferred activities, but no other signs of PDD could be seen.

Case 3

History

EE was born at full term after an uneventful pregnancy and delivery. She was a healthy infant but had many food allergies during her first year and was on a grain-free diet until 14 months and a soy-free diet until 4 years. EE met early motor milestones at the expected ages. She used a few single words at 9 months but then stopped developing additional language and stopped using her few words at about 1 year. During her second year, she seemed to lose other skills, such as clapping to music and imitating, and she developed extreme phobias, severe tantrums, and became socially aloof. At age 2 years, she had no expressive language except for labeling letters of the alphabet. She engaged in repetitive play and displayed extreme distress at changes in her routine.

At age 3 years, EE was diagnosed with Autistic Disorder by a psychologist and a pediatric neurologist. She then began intensive intervention in a home-based program using an Applied Behavioral Analysis (ABA) approach for 20–25 hours per week. EE began to use two-word phrases shortly after intervention began at age 3 years, and she used sentences at 4 years. EE began to develop pretend play at about age 3–6, and she began initiating interactions with another child at 4–6.

EE was evaluated at age 5–2. When prompted by her mother, she would make fleeting eye contact, but she did not make any spontaneously. EE was not socially related during the evaluation. She would engage with the examiner in sensory-motor play, but she was usually very aloof and self-involved. Most of her interactions with others were need-based, and she did not initiate any social interactions. EE was easily distracted from the testing session by external and internal stimulation. She was compliant with all of

the tasks, but she often put forth only minimum effort. When she did not understand what was said to her, she would echo the statement or question back to the examiner. Autistic behaviors had improved greatly since she began intervention, including resistance to change in routine, hand flapping, preoccupation with water fountains, and tantrums. Interest in peers and pretend play were just emerging. She had poor ability to sustain interactions or attention to play, or initiate or sustain conversation. EE had many fears of common objects. At this time, EE was judged to meet criteria for PDD-NOS (impaired eye contact, failure to develop age-appropriate peer relationships, prefers solitary activities, echolalia, impaired conversational ability, lack of varied and spontaneous pretend play).

Medical History

EE had food allergies and ear infections. An EEG, MRI, and hearing test were normal. At age 8, EE began taking 7.5 mg of Ritalin twice a day for attention problems.

Current Presentation

At age 9–1, EE was reevaluated. She was in a typical second grade classroom with the support of a one-to-one aide 4 days a week. She also had language therapy, physical therapy and continued ABA therapy to work on academic, language, and social skills. EE's mother reported that EE had good eye contact, was interested in her peers, was sensitive to the feelings of others, and was able to engage in age-appropriate games and activities. EE was flexible in response to changes in her routine and her environment. EE displayed a full range of appropriate emotions and was affectionate with family members. She engaged in symbolic and creative play, and she enjoyed playing with other children. However, she continued to have difficulty sustaining conversation with other children. Also, her mother reported that she had recently begun toe walking. EE's many phobias had abated. However, EE's mother reported that EE had difficulty regulating herself and her activities, had a hard time extracting key information from verbally presented material, had difficulty maintaining her attention on tasks, was easily overloaded by newly presented information, and had difficulty keeping track of more than one simple task at a time. EE also had difficulty attending to multi-step directions; she needed directions repeated many times or presented as single steps. EE's mother also

reported that EE had a significantly easier time maintaining her attention on tasks when she was working one-on-one with an adult in a very structured environment.

During the evaluation, EE made good eye contact and appropriate verbal responses to the examiner's questions. She easily understood verbal directions, and was cooperative with the task demands. Her affect was somewhat flat, but she was not socially avoidant or internally distracted. No perseverative behaviors or motor stereotypies were noted. However, she was very easily distracted by external stimulation and she had difficulty maintaining her attention on testing. EE required several breaks during the session and much encouragement from the evaluator. EE still showed some below average cognitive abilities but most of her abilities were in the average range.

On interview, EE's mother endorsed all nine of the symptoms of inattention (poor attention to detail, difficulty sustaining attention in tasks, difficulty organizing activities, avoids tasks that require mental effort, easily distracted, forgetful in daily activities, does not seem to listen when spoken to, does not follow through on instructions, and loses things). She also endorsed three of the nine symptoms of hyperactivity (fidgets, difficulty awaiting turn, and interrupts others). Additionally, EE's mother completed the Behavior Assessment System for Children (BASC). On this measure, EE had clinically significant attention problems. Therefore, EE meets criteria for a diagnosis of ADHD-Inattentive Type. Her only residual PDD symptoms are difficulty sustaining a conversation with others and occasional toe walking.

DISCUSSION

We have presented three cases of children with clear-cut disorders on the PDD spectrum which evolved into clear cut cases of ADHD. Key clinical characteristics of the larger group of 11 cases are summarized in Table I. In six cases, the presentation was of ADHD-Inattentive Type; in five cases, it was of ADHD-Combined Type. The common residual features of PDD included a tendency for perseverative interests and occasional but mild motor stereotypies. Social skills tended to remain poor, but in a way more characteristic of impulsive, aggressive, immature children insensitive to the subtleties of sustained interactions than of aloof or odd children. In all cases, the social awkwardness or delay was mild. All of the children wanted friends and most had

at least one real friend; three were unsuccessful in making friends because of impulsivity or bossiness.

The age at which the diagnostic transition took place is difficult to determine. In many cases, the clinical picture was changing at a midpoint evaluation, but the PDD diagnosis was not withdrawn because PDD features were still present and because explicit withdrawal of the diagnosis would have endangered services that were still needed. The average age at which the ADHD picture had become clear and PDD symptoms were no longer present was 7–8 years, with three children clearly evolving into ADHD by age 4–5 and an additional three children by age 6–7.

It should be noted that there was no apparent difference in presentation or severity for the children with early diagnoses of Autistic Disorder vs. PDD-NOS (from other diagnosticians). The differences seemed to be accounted for primarily by the diagnostic practices of the clinicians involved and some reluctance to diagnose Autistic Disorder in very young children.

There seems to be a relatively high proportion of children in this group (9/11) who show the regressive type of autism. Whether these constitute real regression, decreased display of behaviors still in the repertoire, or the emergence of positive symptoms cannot be determined retrospectively, but in these nine cases, parents told convincing accounts of previously acquired skills or emotional relatedness very noticeably declining.

Eight of the 11 children received ABA treatment, often combined with preschool experiences. One might conclude that this aggressive treatment that targets autistic symptoms and systematically teaches skills tends to have this relatively positive outcome. Several ABA programs refer children to the first author for independent evaluations of their progress, however, so this is not an unbiased sample. Ten of the 11 children had recurrent ear infections, which is higher than estimated prevalence in the general population (18% of children under 4 years) (Albrant, 2000).

The percent of children on the PDD spectrum who follow this developmental clinical course is not known, but is not insignificant; there were many more similar (although perhaps less clear cut) cases that could have been presented. A follow-up of a large number of consecutive PDD cases, or epidemiological study, would be needed to estimate how common a pattern this is. Clearly, children who remain functioning in the retarded range or have severe

language disorders that cannot be remediated would not fall in this group, although impairments of attention often appear to be a prominent and limiting factor for them as well.

The pattern of clinical development presented here raises theoretical speculations. It is possible that these cases represent comorbid autism and ADHD (with a chance occurrence or because of elevated comorbidity) and that when the more prominent autistic features resolve, the ADHD features are more clearly revealed. Another possibility is that the children described here represent a subtype of severe ADHD that presents as autism or PDD in the preschool years, but that evolves distinctively because the underlying impairment is different from other cases of PDD. Against this possibility is the fact that these children look typically autistic in early childhood and could not be distinguished from the 'mainstream' of children on the autistic spectrum. Another possibility is that the attentional features of autism are more difficult to remediate than core social and language features, and that when aggressive intervention successfully addresses the core features, the attentional features remain and markedly resemble those of primary ADHD. It is also possible, as has been suggested by Kinsbourne (1991), that attentional features should be regarded as a core deficit in autism and that, in fact, ADHD and autism may lie on a continuum of impaired attention and arousal. He proposes that perhaps because of the centrality of attention deficits to autism, attentional features tend to persist when other features have resolved. Alternatively, it may be that a PDD-ADHD subtype exists, in which the attentional difficulties become more salient after the social and language impairments are remediated.

Although the cases presented seem to clearly delineate the children's diagnostic progression from PDD to ADHD, there are some limitations to the observations. Not all of the children received their initial diagnosis from the same psychologist. Some of the initial diagnoses were confirmed by history and reports written by separate practitioners. Therefore, not all of the children received the same measures of attention and PDD behaviors. Additionally, the ultimate outcome for many of the children described in this paper is also not known. At least one of these cases (CC) who has been followed has lost some of his ADHD features and is indistinguishable cognitively and socially from typical peers but remains troubled by persisting mild attention problems that interfere with complex academic tasks and a heavy

homework load. Of the others who are still being followed, all still have marked ADHD features, but some of the children have not been seen for several years and this may suggest clinical improvement and no further need for clinical services. A systematic follow-up would be needed to elucidate outcome in adolescence and adulthood.

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