

Review article

# Update on the language disorders of individuals on the autistic spectrum

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## Abstract

Inadequate language is a defining feature of the autism spectrum disorders (autism). Autism is a behaviorally and dimensionally defined developmental disorder of the immature brain that has a broad range of severity and many etiologies, with multiple genes involved. Early studies, which focused on the language of verbal children on the autistic spectrum, emphasized aberrant features of their speech such as unusual word choices, pronoun reversal, echolalia, incoherent discourse, unresponsiveness to questions, aberrant prosody, and lack of drive to communicate. Persistent lack of speech of some individuals was attributed to the severity of their autism and attendant mental retardation rather than possible inability to decode auditory language. Clinical study of unselected children with autism indicated that the language deficits of preschoolers fall into two broad types, perhaps with subtypes, those that involve reception and production of phonology (sounds of speech) and syntax (grammar), and those that do not but involve semantics (meaning) and pragmatics (communicative use of language, processing, and production of discourse). Except for the preschoolers' universally deficient pragmatics and comprehension of speech, many of their language deficits parallel those of non-autistic preschoolers with developmental language disorders. There is now biological support for the clinical observation that young autistic children are language disordered as well as autistic. Recent electrophysiological studies disclose auditory input abnormalities in lateral temporal cortex even in verbal individuals on the autistic spectrum. Severe receptive deficits for phonology enhance the risk for epilepsy. Genetic studies indicate that linkage to chromosome 7q31–33 is limited to families with evidence for phonologic impairment as well as autism. Clearly, social and cognitive disorders alone provide an inadequate explanation for the range of language deficits in autism.

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**Keywords:** Autism; Language disorders; Subtypes; Children; Genetics; Auditory processing; Epilepsy

## 1. Definition: autism, the autistic spectrum, pervasive developmental disorder

Autism is a developmental disorder of brain function which has many different causes, with genetic etiologies considerably more prevalent than acquired insults to the immature brain. Autism is not a disease but a behaviorally defined – therefore dimensional – syndrome for which there is no biological confirmatory test. Because of its broad range of severity, the term autistic spectrum seems most appropriate as the subtypes defined in the International Classification of Diseases, 10th edition (ICD 10) [1] or the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM IV) [2] were carved out of this continuum of severity in order to enhance diagnostic uniformity. It seems doubtful at this time that, with the exception of Rett syndrome, these

clinically defined subtypes, listed in Table 1, will map onto particular etiologies.

To be on the autistic spectrum, which is called pervasive developmental disorder (PDD) in ICD 10 and DSM IV, individuals must have deficits in three domains of behavior: (1) sociability, empathy, and the ability to infer what another person may be thinking or experiencing; (2) the communicative use of language and imaginative, creative play; and (3) cognitive and behavioral flexibility, and range of interests and activities. Note that cognitive level is not a defining feature of autism and that this behavioral label has no exclusionary criterion. Autism is generally a static, life-long disorder with symptoms that change and improve with age and as a result of educational intervention [3]. Consequently, the diagnosis of autism is appropriate for individuals whose intelligence ranges from severe mental deficiency to giftedness – albeit usually with notably uneven abilities. It is also appropriate to use this label even in the face of obvious neurologic or sensory handicaps, or of a specific diagnosable biological cause; therefore a person

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Table 1  
Subtypes of pervasive developmental disorder (PDD) or autistic spectrum disorder [1,2]

Autistic disorder	Functionally severe deficits in (1) sociability, (2) communicative language and imaginative play, and (3) range of interests and activities, with at least six of 12 behavioral descriptors endorsed, at least two in sociability, at least one each in language and range of interests
Asperger syndrome	A less severe syndrome, with sentences acquired at the normal age, an IQ of 70 or better, and often clumsiness, but with severely impaired sociability and cognitive and behavioral flexibility
PDD not otherwise specified (PDD-NOS)	Category appropriate for individuals who do not fulfil criteria for any other PDD subtype, yet have endorsement of at least two descriptors, at least one in sociability
Disintegrative disorder	Severe regression of language with the appearance of autistic symptomatology after entirely normal early development, including speaking in full sentences. Must not be attributable to a degenerative disease of the brain
Rett syndrome	Specific genetic cause of autism in girls associated with postnatal failure of brain growth, early regression of sociability, absent or rudimentary language, severe hand stereotypies, and mental retardation, and a variety of other neurologic symptoms, including epilepsy. It is generally but not always due to mutations in the <i>MCP2</i> gene on Xq28. Less severe phenotypes and rare occurrences in boys are also known.

who is blind or has an overt hemiparesis or Angelman syndrome must also receive the additional behavioral diagnosis of autism if he or she has deficits in the three behavioral domains just mentioned.

## 2. Neurologic basis and causes of autism

It is now well established that autism does not arise except in the face of dysfunction of complex brain networks that involve the neocortex, diencephalic relays, cerebellum, and probably other subcortical nuclei involved in neurotransmitter release [4,5]. The effects of psychopharmacologic agents implicate serotonin, dopamine, norepinephrine, and no doubt other neurotransmitters and neuromodulators in the pathophysiology of autism [6,7].

The defined etiologies most likely to be associated with autism are those with widespread effects, like congenital rubella or tuberous sclerosis, or some known genetic conditions, such as fragile-X, Angelman syndrome, phenylketonuria, and others [8]. Tuberous sclerosis, caused by either of two single gene defects on 9q or 16p, has a very variable phenotype which includes autism in a quarter to a third of cases, especially those with epilepsy [9], although it accounts for no more than 1–2% of individuals on the autistic spectrum [10]. A recent imaging study in children with tuberous sclerosis and intractable epilepsy [11] indicated that, compared to the affected children who were mentally retarded with or without autism, those who were behaviorally unimpaired had significantly fewer and smaller tubers, especially in the dorsal prefrontal gyri. Positron emission tomography of non-lesional tissue revealed that those who were autistic and had impaired communication had decreased glucose metabolism in lateral temporal gyri bilaterally, cortical areas crucial to auditory and language processing; they also had bilateral increases in glucose metabolism in the deep cerebellar nuclei and in alpha-methyl-tryptophan in the caudate nuclei.

Known structural or genetic disorders associated with autism account but for a small minority, maybe 10%, of cases [12]. Twin and family studies demonstrate that genet-

ics plays the major role in the vast majority of individuals in whom there is no currently diagnosable etiology [13], but that single causal gene defects are rare and that polygenic inheritance is common. Gene linkage studies show that these genes or groups of genes vary among affected individuals. This explains the fact that recurrence risk within sibships is under 10%, and also that family members of individuals with autism are at greater risk for developmental disorders other than autism such as language disorders, obsessive–compulsive disorder, and bipolar disease [13,14].

## 3. Some early studies of language in verbal children with autism

In his original description of 11 children with classic autism, Kanner [15] elaborated on a number of communication abnormalities. Three of the children were mute, but occasionally said a full sentence. The language of those who spoke had striking characteristics: echolalia; pronoun reversal; the production of utterances with tenuous or no obvious relation to the conversational context; and, strikingly, unresponsiveness to questions and lack of drive to communicate either verbally or with gestures. At virtually the same time, Asperger [16] was equally struck by the communicative deficits of the four boys whose biographies he provided and he stated that less severely affected children with similar characteristics were not rare. His language descriptions were identical to Kanner's. He commented on the idiosyncrasy and, in some cases, verbosity of the children's language and its aberrant prosody (intonation and rhythm). Bartak et al. [17] found that comprehension of speech was more severely compromised in children with autism than in mentally retarded children matched for non-verbal cognitive level and Tager-Flusberg [18] indicated that this was also true when they were matched to normally developing children. She reported that children with autism did not differ from matched retarded controls in phonology, prosody, or syntax, but that they had more severe comprehension and pragmatic (conversational use of language) deficits than children with developmental

Table 2  
Allen and Rapin clinically defined language disorder subtypes in preschool children [23,24]

A. Mixed receptive/expressive disorders	Impaired phonologic decoding which affects all subsequent processing of language. Expression sparse and dysfluent. Language acquisition through the visual channel often unaffected
Verbal auditory agnosia	Phonologic decoding so profoundly impaired that the children understand no language and therefore are non-verbal or virtually so
Phonologic–syntactic subtype	Comprehension impaired but equal to or superior to language production. Expressive language sparse, in rudimentary, poorly articulated sentences, vocabulary impoverished
B. Higher order processing disorders	Comprehension and formulating of discourse impaired, phonology and syntax may be delayed but are not a primary deficit
Lexical syntactic subtype	Severe word finding deficit resulting in dysfluent language, syntax often immature. Expression may start as fluent jargon
Semantic pragmatic subtype	Expressive language fluent, echolalic, often verbose and scripted, with verbal perseveration, unusual word choices, and impaired conversational use of language. Comprehension more impaired than production
C. Expressive disorders	Comprehension normal or near normal
Verbal dyspraxia	Extremely dysfluent expression in the face of normal or near normal comprehension. Although verbal dyspraxia may be associated with oromotor deficits and overall clumsiness, these motor deficits are not severe enough to account for the profoundly impaired expressive deficit which is postulated to be at the level of retrieval of the commands for verbal expression
Phonologic programming subtype	The children are fluent and unintelligible, or they have small distorted expressive vocabularies and simplified syntax.

language disorders. Semantics (meaning of linguistic utterances) was more impaired than syntax (grammar); the children violated semantic constraints and did not use a semantically based strategy for recall. She concluded that in autism, phonologic (speech sounds) and syntactic development are relatively independent of semantic and pragmatic development.

Autism was not listed in the DSM until its third 1980 edition [19]. In that and all subsequent editions, language and communication, together with imaginative play, appear as the second of the key behavioral domains affected by autism. The diagnostic criteria for language listed in the most recent edition [2] are (1) late or lack of development of language without attempt to compensate with gestures, (2) impairment in the ability to initiate or sustain a conversation, and (3) stereotyped, repetitive, and idiosyncratic language. The descriptors in the accompanying text include aberrant prosody, immature syntax, impaired comprehension, failure to perceive irony or understand jokes, and severely deviant pragmatics, without any mention of phonology.

Most of these earlier studies, and many more recent ones described the language of children who could speak. In an early study, Bartolucci et al. [20] compared the phonologic errors of nine children with autism and a mean age of 12 years to those of 12 non-autistic retarded children, matched for age and non-verbal intelligence quotient (IQ). They found that acquisition of phonology, albeit delayed, followed the same trajectory as in normally developing children, but that children with autism made more substitution errors than their non-autistic matched controls. They concluded that the children with autism were language disordered as well as autistic. Because lack of speech was generally associated with either severe mental retardation or severe autism, persistent failure to develop language was widely attributed to these deficits and the language abilities

of non-verbal individuals were not studied in any detail. Miranda-Linné and Melin [21] did compare mute to verbal children and adults with autism using the Autism Behavior Checklist [22]. Comprehension, like expression, was worse in the mute individuals, who also had more severe autistic symptomatology.

#### 4. Clinical studies of the language of unselected preschool children with autism

The first study of language in an unselected sample of children with autism was carried out by the late D.A. Allen, a developmental psycholinguist who directed a therapeutic nursery for preschool children with communication disorders with either autism or severe behavior disorders, none of whom was severely retarded, and IR, a child neurologist with a particular interest in communication disorders [23–25]. Our collaboration led us to propose a clinical classification of preschool children with inadequate language development, whether or not they were on the autistic spectrum, and whether or not they could speak. Evaluating language at the levels of phonology, grammar, semantics, and pragmatics linked to a neurologic input–processing–output model, we identified three major types of disorders, each of which was divided into two subtypes on the basis of the severity and characteristics of the children’s language disorders (see Table 2 for details).

We applied this model to two non-overlapping cohorts: 109 preschoolers (mean age 3.6 years, range 2–5 years) enrolled consecutively in the therapeutic nursery; and all 382 children with inadequate communication seen in IR’s practice (mean age at first visit: 5 years) [25]. To study language, we excluded children with severe mental retardation, hearing losses, cerebral palsy, or inadequate data. We used DSM III-R criteria [26] to diagnose an autistic spectrum disorder (both classic autism

Table 3

Clinical classification of language disorders in children on the autistic spectrum and developmental language disorders (dysphasia) after exclusion of children with severe mental retardation

Subtype of language deficit	Children with autism	Children with dysphasia
a. Allen and Rapin cohort [25]		
<i>N</i> = 491	<i>N</i> = 299	<i>N</i> = 262
Mixed receptive/expressive disorders	144 (63%)	130 (50%)
Higher order processing disorders	85 (37%)	40 (15%)
Expressive disorders	0	92 (35%)
b. Tuchman et al. cohort [27]		
<i>N</i> = 412	<i>N</i> = 197	<i>N</i> = 215
Verbal auditory agnosia	17 (9%)	12 (5%)
Phonologic–syntactic disorder	117 (59%)	113 (53%)
Higher order processing disorders	62 (32%)	19 (9%)
Expressive disorders	0 (0%)	72 (33%)

and PDD-NOS) and diagnosed developmental language disorders (dysphasia) without autism clinically. The children in the nursery were well known to Dr Allen and had undergone extensive neuropsychologic and language testing. Children seen in neurologic consultation had been assigned a diagnosis of developmental language disorder or autistic spectrum disorder at the time of the visit; classification of their language disorder was made either prospectively on the basis of historical data and observation during a short play session as part of the consultation, or retrospectively by chart review.

This cohort of 491 children consisted of 229 children with an autistic spectrum disorder and 262 children with a developmental language disorder (Table 3). As expected, boys outnumbered girls 4/1 in each of the groups. There were two striking findings: first, none of these young children with an autistic spectrum disorder were judged to have normal comprehension, whereas 92 (35%) of the children with language disorders were deemed to have normal or near normal comprehension; and second, the mixed receptive/expressive disorder, in which phonology and syntax are both deficient, was the most prevalent disorder in both groups, and in fact slightly more so in autism (63%) than dysphasia (50%). This finding contradicts the widely held notion that phonology and syntax are not involved in autism. We assigned the diagnosis of a higher order processing disorder, which involves receptive and expressive semantics and, in autism especially, pragmatics, but not phonology and syntax, to only 37% of the children with autism. Higher order processing disorders, thought previously by many to be unique to autism, were not absent, but distinctly less common in dysphasia as we assigned them to only 15% of the children in that group.

Tuchman et al. [27] carried out a second more detailed clinical study of a cohort of 551 consecutive children with inadequate communication that IR had seen in consultation, a cohort that partially overlapped the neurologic cohort just described but that did not include any of the children from

the nursery (Table 3). Mean age at first visit was 5 years 2 months in the autistic sample and 4 years 11 months in the dysphasic sample. In order to compare meaningfully the language disorders of children with autism to that of children with dysphasia and make the groups more comparable, we excluded 66 children with autism and severe mental retardation. We also excluded 28 autistic hearing impaired children, 12 girls with Rett syndrome, and 11 autistic and 22 dysphasic children with inadequate data. This left 197 children with autism and all 215 with a developmental language disorder for study. The children were assigned to the same major clinically defined language disorder subtypes as in the first study, except that children with verbal auditory agnosia (VAA), the most severe mixed receptive/expressive disorder because of profoundly deficient phonologic decoding, were tabulated separately from those with the phonologic/syntactic disorder. Table 3 shows that once again children with the mixed receptive/expressive subtype were the most numerous in both groups, and that children on the autistic spectrum did not have purely expressive disorders. As in the earlier study, higher order processing disorders were more than twice as prevalent in children with autism as in those with dysphasia,

Klein et al. [28] in a retrospective study of 67 children with VAA, all but ten of whom were in IR's patient files, looked at prognosis depending on whether the disorder was developmental or acquired, and whether or not autism or epilepsy was present. Fifty-eight percent of the children had seizures, 76% had autism, and 24% had a history of language regression. Children with autism were likely to have had either absent or very abnormal early language (developmental VAA) or to have experienced language regression before 3 years of age. Language fluctuations were reported more often in children with acquired VAA than in those with developmental VAA. Seizures were more prevalent (71%) in children with acquired VAA who were also autistic than in the other groups (18% in the non-autistic children with acquired VAA, and about 50% in those with developmental VAA, whether or not they were autistic). Autism was a risk factor for poor language recovery and later learning disabilities. The study emphasized the complex interrelationships between language, seizures, and behavior in children with VAA, and the difficulty in determining whether epilepsy is responsible for both the language disorder and the autism, or whether all three are independent consequences of the underlying brain dysfunction.

Ballaban-Gil et al. [29] evaluated intelligence, language, and behavioral outcome by structured telephone interview in 99 of 163 consecutive subjects on the autistic spectrum IR had evaluated more than 5 years earlier. (Of the other 64 subjects, three had died, the others could not be reached or refused to participate.) There were 54 (55%) adolescents aged 12–17 years, and 45 (45%) adults 18 years or older. Behavioral issues remained problematic in 69% of the sample and 90% had persistent social deficits. Expressive

language had improved with age, although only 35% of those who were neither hearing impaired nor severely retarded had achieved normal or near normal fluency. Children with normal or near normal intelligence at first evaluation had improved the most, as had children first evaluated before the age of 6 years. Thus this study confirmed that, as reported in other studies (reviewed in Lord and Paul [30]), speech before the age of 5 years was a favorable prognostic sign. Of the children who had mild/moderate mental retardation when first evaluated between 6 and 12 years, none had improved in comprehension, although expression was better in 20% of them.

### **5. Empirical studies of the language of preschool children with autism compared to children with developmental language disorders (dysphasia)**

In a large multiinstitutional, multidisciplinary study, 476 preschoolers were divided into two cognitive groups at a non-verbal IQ of 80, creating a non-retarded group and a low IQ group [31]. The children were also divided into an autistic disorder group and non-autistic group on the basis of their parents' responses to the Wing autistic disorder interview checklist (WADIC) [32] and by psychiatrists' evaluations following DSM III-R [26] criteria for autistic disorder. The resultant four subgroups were thus two autistic disorder subgroups: high autistic disorder (HAD) and low autistic disorder (LAD), and two non-autistic subgroups: developmental language disorder and non-autistic low IQ. All the children were tested with standardized language instruments and the spontaneous language of those who were verbal, as well as that of a comparison group of normal children, was recorded during a standardized play session for transcription and analysis. The neurologists who evaluated the children described as non-verbal or minimally verbal 56% of those in the LAD subgroup, compared to 6% of the HAD subgroup, and as dysfluent 50% of the verbal LAD and 27% of the HAD children. Severely impaired fluency was confirmed by scores on the fluency subtest of the McCarthy scales of children's abilities [33]. The scores of the testable LAD group were lower than those of the HAD group and those of the other two groups on all the language measures, notably Verbal Reasoning of the Stanford Binet 4th edition [34]. Analysis of spontaneous language confirmed that the verbal LAD group was the most impaired on virtually all measures. Co-varying for age, non-verbal IQ, and socioeconomic status, the LAD children produced more echoes than the HAD children and produced shorter utterances, although the two groups did not differ in number of meaning or structural errors. Overall, the HAD children had uneven language skills, with deficits in expression of connected speech, verbal comprehension, and rapid naming, and relative strengths in single word labeling and written language. The testable LAD children were impaired on all these language measures.

One hundred and six of the children in the autistic group were restudied at school-age, 23 at age 7 years and 59 at age 9 years (we used the 9 year data in children tested at both ages). (in preparation) There were 37 children with preschool non-verbal IQs of 80 or more and 45 with non-verbal IQs below 80. A direct, non-hierarchical cluster analysis of the entire sample with nearest centroid clustering was performed. Because of the findings in the earlier studies, the variables chosen for clustering were a measure of expressive phonology, the Photo Articulation Test [35], and a measure of comprehension of connected speech, the Clinical Evaluation of Language Fundamentals [36], its appropriate subtests being Sentence Structure at age 7 years and Semantic Relations at age 9 years. A three cluster solution gave better subgroup separation than a four cluster solution. Clusters at ages seven and nine were virtually identical. Cluster 1 included children who were low on both expressive phonology and comprehension of connected speech, a cluster corresponding to the clinically defined mixed receptive/expressive group. Children in cluster 2 and 3 had average phonologic scores, like the children in the clinically defined higher processing disorders and like the children in the classical descriptions in the literature of language in autism [37]. Age and non-verbal IQ did not determine cluster membership. Predictably, verbal IQ was lower than non-verbal IQ in the children in all three clusters and was frankly low (63 and 65) in the children in both clusters. Children in all three clusters had deficits in sociability. How well these clusters will map onto the clinically defined language subtypes requires further research.

### **6. Electrophysiologic and imaging studies**

Electrophysiology provides biological support for an auditory processing deficit affecting language development in autism. Studies evaluating auditory brainstem responses and middle latency responses have yielded inconsistent results. Recent cortical evoked response studies provide evidence for more consistent dysfunction in the lateral surface of the superior temporal gyrus, as indicated by the negative wave N1c, a component of the obligatory auditory event related potential. In an early study, Narita and Koga [38] identified a delay in the latency of N1c over the left hemisphere in response to speech stimuli. Klein et al. [39] studied six young adults with a history of childhood VAA and variable severity of deficits in auditory discrimination, compared to six normal adults matched for age and gender. Brain stem and middle latency auditory evoked responses did not differ between subjects and controls. In contrast, the N1c to tones and speech syllables was delayed bilaterally in the VAA subjects. This strongly suggested a persistent abnormality in secondary auditory cortices even in the subjects who had regained normal verbal expression many years earlier. Bruneau et al. [40] described diminished amplitudes and prolonged latencies of N1c in response to

pure tones in 4–8 year old children with autism and mental retardation, compared to age-matched normal and mentally retarded children. In contrast, Dunn et al. [41] reported significantly delayed N1c in response to words, but not tones, in a semantic classification task in verbal school-age children with autism, even in those who were not phonologically impaired. In short, electrophysiology indicates consistent abnormalities in the neural processing of words in lateral superior temporal gyrus and of simple non-verbal auditory stimuli by a subset of individuals with autism.

Electrophysiologic evidence also provides evidence for abnormal neural processing of semantic information. The processing negativity known as N4 does not show the normal increase in response to deviance from semantic context in children with autism [41], perhaps reflecting impaired selective activation of the meaning of words by semantic context.

Most imaging studies in children with autism were performed carried out in sedated subjects and thus could not address functional changes during the accomplishment of language tasks [42–44]. Investigators have described a variety of structural abnormalities in individuals with autism compared to normal controls, notably in the cerebellum, brain stem, corpus callosum, diencephalon, and cerebral white matter. The volume of the white matter of the cerebrum is subtly larger in early life [45]. Maturation of serotonin synthesis differs in children with autism compared to controls [7]. But with the exception of the study in children with tuberous sclerosis quoted earlier [11], no abnormality has been consistent or characteristic of autism or its language disorders.

## 7. Discussion

Both the clinical and the empirical studies support the view that the language disorders of children on the autistic spectrum have much in common with those of children with developmental language disorders, although those with autism differ strikingly by their universally impaired pragmatics and comprehension of discourse. They also differ by the prevalence of ‘pure’ expressive disorders – much more prevalent in dysphasia, and of higher order processing disorders – more prevalent in autistic spectrum disorders.

Very recent genetic evidence provides underpinnings for the several behaviorally defined language disorder subtypes in autism and their clustering driven by expressive phonology together with comprehension of discourse. Genetics offers biological support for the broad division of the language disorders of children with autism into those that involve phonology and those that do not. A gene, *FOXP2* in the *SPCH1* region of 7q31 had been identified in a large British family with a dominantly inherited severe global language disorder [47]. Wassink et al. [46] reported linkage of probands with autism who had spoken later than the age

of 3 years and had non-autistic first degree relatives with either late development of language or reading or both, that is with auditory phonologic processing deficits, to chromosome 7q31–33. This led to the speculation that *FOXP2* might be a gene for autism with language disorder, but a more recent study [48] questions this linkage. It is now well accepted that a major cause of dyslexia is difficulty with phonologic processing [49,50]. As stated earlier, families of children on the autistic spectrum on average have an excess number of individuals with language and other developmental disorders, which bolsters evidence for the multigenic inheritance and widened phenotype of autism [51]

The fact that many descriptive and comparative studies of the language characteristics of selected children on the autistic spectrum were carried out at schoolage or in adolescents and included only verbal children provides a plausible explanation for their surprising conclusions that phonology and syntax are not involved in autism. The DSM and ICD systems cite delayed development of speech as a key characteristic of language in autism, and delay is acknowledged in many descriptions of the language characteristics of individuals with autism. Allen and Rapin specify that expression often starts with a fluent jargon and immature syntax in verbal preschool children of the lexical–syntactic subtype, which suggests imperfect phonologic decoding. They also stress word retrieval deficit, which is a semantic deficit, impaired comprehension of discourse and, especially, question forms. Semantic deficits in children with higher order processing deficits may be ascribable to problems with semantic organization rather than to delayed maturation of phonology and syntax.

Most highly intelligent adolescents and adults with Asperger syndrome or PDD-NOS (autism residual form) no longer manifest deficits in comprehension of discourse. What remains life-long are the semantic and pragmatic deficits such as difficulties in sustaining conversation, turn-taking and allowing the conversational partner to introduce his or her topic, in prosody and direction of gaze. Much remains to be done for identification of the involved brain circuits and the nature of their deficits before a comprehensive understanding of the neurologic basis of the language disorders of autism is achieved.

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