CHAPTER 16

Pervasive Developmental Disorders: Autism

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Every primary care physician can expect to treat an individual with autism.[1] Until recently autism was considered a rare disorder[2] resulting from the child's reaction to parental rejection.[3,4] Today autism is recognized as a relatively widespread disability[5] reported twice as frequently as it was in the past,[6] and it is more prevalent than childhood cancer, diabetes, spina bifida, and Down syndrome.[5] Autism is now known to be of neurobiologic origin,[7] and its presence in families is not related to parenting style, social economic status, race, or ethnicity.[8,9]

Although they require the same medical care as other children, children with autism pose unique challenges for primary care providers (PCPs).[1,10] First, as one of the most complex neurodevelopmental disorders, autism is a diagnosis based on behaviors instead of medical tests. The lack of a biologic marker for autism requires the diagnostic clinician to have specialized skills and experience in identifying the behavioral phenotype.[11] PCPs, who are often the first health care providers to learn about parental concerns,[12,13] are in a critical position to screen for autism in very young children and make appropriate referrals for a comprehensive diagnostic assessment.[14] A second challenge is that there is no known single cause or cure for autism. Confronted with this lack of information, parents are often vulnerable to unproven and invalidated treatments. As a result, physicians have to address many questions regarding therapy options and management. Health care providers must be equipped with enough knowledge to assist parents in treatment decisions. Today, it is necessary that all physicians be aware of the early features of autism, know when to refer a young child for a comprehensive diagnostic assessment, and have enough information on the disability to be able to provide ongoing advice to parents and care givers. In order to help address these challenges, this chapter will review current information on etiology and recommended practices for screening, diagnosis, and treatment in autism. Table 16–1 provides a list of resources to help clarify the recommendations.

Etiologic Theories of Autism

The etiology of autism remains unknown. Researchers have focused on several primary target areas, including genetic, neuropathologic, and environmental sources. Possible metabolic[15] and immunologic[16] causes have also been investigated. Although there is agreement that autism results from an alteration of normal brain development, to date, no single cause for autism has been identified. A consensus is building that autism results from multiple etiologies[1,10] and is likely present before birth or during early infancy.

The possibility that some individuals are genetically predisposed to developing autism has received much attention. Genetic researchers have reported monozygotic concordance rates of 60% for autism and 92% for the broader symptoms of social and communication difficulties. The concordance of autism in dizygotic twins is 0%, yet 10 to 30% of dizygotic twins develop the broader spectrum of symptoms.[17] Research suggests that several genes, perhaps as few as 3[18] and possibly more than 15,[19] contribute to the behavioral features in autism. A sibling occurrence rate from about 2 to 7% has been reported, as well as an increased risk of associated genetic and chromosomal abnormalities.[6,20] Autism, for example, has been linked with fragile X syndrome[21,22] and tuberous sclerosis.[23,24] Genetic abnormalities on all but two chromosomes (chromosomes 14 and 20) have been associated with autism.[25] Because of these associated conditions and family studies, genetic as well as other sources of etiology continue to be investigated.

Evidence for autism-associated neuropathology comes from several sources. First, many individuals with autism have additional neurologic conditions. Mental retardation occurs in about 65 to 85%[26] and epilepsy in about 30% of individuals with autism.[27] Second, neuroimaging and autopsy studies suggest an alteration of normal brain development during the prenatal period. Reduced size and number of Purkinje cells and cerebellar hypoplasia, as well as reduced neuronal

Table 16–1 Recommended Resources for Health Care Providers

Description	*Title and Source*
This article provides a general overview of autism and related research at the National Institute of Child Health and Human Development.	Autism questions and answers for health care providers[9]
These reports summarize findings from a multidisciplinary NIH Consensus Panel on screening and diagnosis.	The screening and diagnosis of autism spectrum disorders[5] Practice parameter: screening and diagnosis of autism[59]
Developed specifically for pediatricians, this policy statement summarizes information on diagnosis and management of autism including conventional and alternative treatments.	Technical report: the pediatrician's role in the diagnosis and management of autistic spectrum disorder in children[1,10]
This report, at the request of the US Department of Education's Office of Special Education Programs, was sponsored by the National Research Council and summarizes the state of the scientific evidence of the effects of early educational intervention on young children with autism spectrum disorders .	Educating children with autism[53]
This Web site provides practical information that can be printed and shared with families regarding many topics related to ASDs.	Autism Society of America, online at <www.autism-society.org>
This policy statement describes the federal, state, and local requirements of special educational and early intervention services, the pediatrician's role in collaborating with early intervention and educational professionals and parent support groups.	The pediatrician's role in the development and implementation of an Individual Education Plan (IEP) and/or an Individual Family Service Plan (IFSP)[67]

ASD = autism spectrum disorder; NIH = National Institutes of Health.

cell size, truncated dendritic growth, and increased cell packing in limbic structures have been reported.[28,29] These findings, however, are not specific to autism.

For many years, researchers have studied possible environmental causes of autism. In the 1970s, a correlation between congenital rubella and autism was reported.[30] Recently, thalidomide exposure has been linked to autism,[31] implicating its onset around the time of neural tube closure. A few researchers exploring the measles-mumps-rubella (MMR) vaccine reported a causal association with autism.[32] No conclusive scientific evidence, however, supports this hypothesis or any hypothesis of a combination of vaccines as a cause of autism.[9,33] In addition, no evidence exists for a link between autism and the type of mercury containing preservative (ie, thimerisol) used in the manufacture of vaccines. The Institute of Medicine and the American Academy of Pediatrics (AAP) have published independent reviews of the scientific evidence of the relationship between vaccines and autism; both reports identify no connection.[33,34]

Although the numbers of children with autism who have possible metabolic[15] or immunologic disorders[16] are unknown, it is hypothesized that these disorders may play a causal role. Research on the correction of the underlying metabolic dysfunction (through diet, drugs, or nutritional supplements), however, has not demonstrated a reversal of autism.[15] Similarly, no direct evidence has provided a causative link between immune system abnormalities and pathogenesis of autism.[35]

Prevalence of Autism

The prevalence of autism has recently caught the interest of researchers, service providers, and parents alike. Public agencies such as the US Department of Education have identified autism as the largest growing low-incidence disability reported by public school personnel.[36] The California Department of Health and Human Services reported a 273% increase in the number of children with autism seeking services from 1987 to 1998.[37] These alarming statistics have led some to imply an autism epidemic.[38] With careful examination, however, evidence suggests otherwise. In a careful study evaluating the prevalence of autism using a standard-

ized autism diagnostic instrument,[39] Chakrabarti and Fombonne[6,40] calculated a current prevalence rate of 62.6 per 10,000 for all pervasive developmental disorders (PDDs), a group of disabilities that includes four other categories besides autism. This number is higher than previous estimates, but analysis of the specific diagnostic groups indicates that there was a disproportionate increase in the number of children with symptoms milder than those typically seen in classic autism. Subgroup analysis determined that 16.8 per 10,000 preschool children had autism, 8.4 per 10,000 had Asperger syndrome, 36.1 per 10,000 had Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS), 1 per 10,000 had Rett's disorder, and 1 per 10,000 had Childhood Disintegrative Disorder. Analysis of the age of referral indicated that children with autism received an earlier diagnosis than those children who were later diagnosed with Asperger syndrome or PDD-NOS. The children with autism also had more cognitive and language delays. Overall, Fombonne[40] reached the conclusion that although epidemiologic studies of autism are flawed with methodologic limitations, data on increased numbers of children with autism are more likely to be reflecting (1) a usage of a broader definition of autism, (2) changes in diagnostic criteria over time, and (3) an improved recognition of autism, especially in children without mental retardation.

Diagnostic Criteria of PDD

The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR)[41] describes the diagnostic criteria for the PDDs. Each of the PDDs shares some features. But of the five PDDs, three have the most overlap with one another (ie, Autistic Disorder, Asperger syndrome, or PDD-NOS). Many researchers believe that the shared social impairments are the hallmark features of the PDDs that distinguish them from other childhood disorders.[42–44] Also, instead of the term PDD, researchers are advocating for the term Autism Spectrum Disorder (ASD) to emphasize both the shared overlap and lack of clear distinctions between the PDDs and the fact that these children often benefit from the same services.[45] Next, the detailed *DSM-IV-TR* criteria for each PDD are presented. Table 16–2 provides a brief comparison.

Diagnostic Criteria of Autistic Disorder

Although autism becomes evident within the first 3 years of life, it often remains undiagnosed until 4 years of age. The relatively late diagnosis is due to many factors. The identification of autism requires specific expertise and knowledge. Diagnosticians must have a solid understanding of normal social and communica-

Table 16–2 Diagnostic Criteria for Pervasive Developmental Disorders

	Autistic Disorder	*Asperger Disorder*	*Pervasive Developmental Disorder—Not Otherwise Specified*	*Rett's Disorder*	*Childhood Disintegrative Disorder*
Disordered social interaction	Present	Present	Present	Present	Present†
Disordered communication	Present		Present*	Present	Present†
Restricted and repetitive behaviors	Present	Present	Present*		Present†
Age of onset	Prior to 36 months			Prior to 36 months	Between 2 and 10 years
Pattern of regression in several areas				Present	Present
Average intelligence		Present			
Incidence[6]	16.8/10,000	8.4/10,000	36.1/10,000	1/10,000	1/10,000
Male to female ratio	3 to 4:1	More common in males	More common in males	More common in females	More common in males

*One of these must be present.
†Two of these must be present.
Adapted from Ruble L, Stone W.[73]

tion development in order to determine if a child is experiencing difficulties as a result of a developmental delay or autism. For very young children under the age of 2 years, establishing the consistency between a child's developmental and mental ages and social and communication skills may prove difficult, especially if the child has low nonverbal skills.[45] Another issue is that children with autism can be notably different from one another. Two children with autism can meet different combinations of the *DSM-IV-TR* diagnostic criteria. In addition, the same child with autism may meet a certain combination of criteria when younger but a different combination when older.[46] Despite these challenges, research indicates that children can be identified reliably before 3 years of age.[11,47,48]

The first component of the definition of autism, social impairment, is more challenging to detect because it represents a relative absence of behavior. Autism is distinguished by significant impairment in at least two of the following four areas: (1) coordinated use of nonverbal behaviors to regulate social and communicative interactions (eg, eye-to-eye gaze, gestures, facial expressions); (2) development of peer relationships appropriate to the child's developmental level; (3) seeking to share enjoyment, interests, and achievements with others; and (4) establishing social and emotional reciprocity (eg, engaging in social play for older children or peek-a-boo for younger children).[41]

The communication disorder, the second component of autism, is featured by significant impairment in at least one of the four areas: (1) problems in development of spoken language (also accompanied by a lack of compensation through other modes of communication like gestures); (2) inability to initiate or sustain a conversation with others in individuals with spoken language; (3) the presence of stereotyped and repetitive use of language or idiosyncratic use of language (eg, repetition of words or phrases without regard to meaning); and (4) a lack of varied, spontaneous make-believe play or social imitative play consistent with the child's developmental level.[41]

To meet criteria under the third area of impairment is to demonstrate restricted, repetitive, and stereotyped patterns of behavior interests and activities in at least one of the following four areas: (1) preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal in intensity or focus; (2) inflexible adherence to specific nonfunctional routines or rituals; (3) stereotyped and repetitive motor mannerisms; and (4) a persistent preoccupation with parts of objects.

In addition to meeting the criteria described above, the child must also demonstrate abnormal functioning in at least one of the following areas prior to 3 years of age: (1) social interaction, (2) language as used in social communication, and (3) symbolic or imaginative play. In addition, Rett's Disorder and Childhood Disintegrative Disorder, to be described later, must be ruled out.

Diagnostic Criteria of Asperger's Disorder

The next most closely related PDD is Asperger syndrome. Debate continues among researchers about whether Asperger syndrome can be distinguished from high-functioning autism (children with autism who do not have cognitive impairment).[49,50] In order to meet criteria for Asperger syndrome, the child must demonstrate impairments in two of the areas previously described for Autistic Disorder: (1) social interaction and (2) restricted, repetitive patterns of behavior, interests, and activities. The child must not demonstrate any clinically significant general delay in language and should use single words by age 2 and communicative phrases by age 3. In addition, the child also must not exhibit any significant delay in cognitive development or adaptive behavior (except for social interaction) and show curiosity about the environment in childhood.[41]

Diagnostic Criteria of PDD-NOS

The next ASD that is most closely associated with autism and Asperger syndrome is PDD-NOS. PDD-NOS is diagnosed when a child does not meet criteria for autism because of late age at onset, atypical symptomatology, or subthreshold symptomatology. Children with PDD-NOS do demonstrate the (1) social impairments and either (2) communication impairments or (3) restricted, repetitive, patterns of behavior, interests, and activities.

Diagnostic Criteria of Rett's Disorder

Rett's disorder is an X-linked neurodevelopmental disorder with an identified mutation in the gene *MECP2*.[51] Rett's disorder is unique from the previously described PDDs for several reasons. Occurring most often in females, but also present in males and resulting in multiple and specific deficits following a period of normal development after birth, children with Rett's demonstrate all of the following: (1) normal prenatal and perinatal development, (2) normal psychomotor development for the first 5 months after birth, and (3) normal head circumference at birth. After this period of normal

development, all of the following are observed: (1) deceleration of head growth between 5 and 48 months of age, (2) loss of previously acquired purposeful hand skills between 5 and 30 months of age with subsequent development of stereotyped hand movements (eg, hand wringing or hand washing), (3) poorly coordinated gait or trunk movements, and (4) severely impaired expressive and receptive language development accompanied by severe psychomotor retardation.

Diagnostic Criteria of Childhood Disintegrative Disorder

Childhood Disintegrative Disorder is diagnosed when a child experiences marked regression in multiple areas of development following a period of at least 2 years of typical development. Age-appropriate development of verbal and nonverbal communication, social relationships, play, and adaptive behavior is observed. After the age of 2 years, but before the age of 10 years, the child exhibits a significant loss of previously acquired skills in at least two of the following areas: expressive or receptive language, social skills or adaptive behavior, bowel or bladder control, play, or motor skills. Typically, acquired skills are lost in almost all areas of development.[41]

Importance of Identifying Autism in Young Children

Despite the published results demonstrating the beneficial effects of early intervention,[52,53] many children with autism miss the opportunity for specialized services because the average age of diagnosis is about 4 years.[12,13] Making an early diagnosis of autism is essential for care givers and children. Access to services, accurate information, and specially designed intervention programs are based on a diagnosis. Also, providing parents with information helps them to become more knowledgeable and informed consumers of services on behalf of their child. It also assists them in becoming organized for advocacy efforts in local and federal arenas on policy issues that affect their child and other children and adults with disabilities, such as the identified need for better-trained personnel and appropriate services.[54] PCPs who have knowledge of early symptoms of autism and listen closely to parental concerns of their child's social and communication development are more likely to refer parents and care givers to appropriate diagnosticians, allowing children and families to participate in specialized programs as early as possible.

A comprehensive evaluation that can provide both a definitive diagnosis and treatment recommendations is best conducted by a multidisciplinary assessment team comprised of a physician, psychologist, speech and language pathologist, occupational therapist, and educational specialist.[55] In order to know when to refer a child for comprehensive evaluation, it is necessary to be familiar with screening tools that identify children with possible developmental problems and instruments that differentiate children with autism from those with other developmental disorders. The AAP recommends that all physicians know and use at least one screening tool on all children.[1]

Early Indicators of Autism in Very Young Children

The *DSM-IV-TR* criteria for autism may be limited for preschool children because very young children were excluded in field tests.[56] Nevertheless, recognizing the red flags of possible autism can lead parents in the right direction. Researchers, for example, have reported behaviors that distinguish very young children with autism from a developmentally matched sample that include less frequent use of eye contact, responding to name being called, pointing, and showing behaviors.[57,58] More formal methods of identifying autism young children are now available.

Level 1 screening, which should be conducted on a routine basis at all well-child visits,[59] identifies children who are at risk for developmental delays. A list of level 1 screening instruments is available in Filipek and colleagues.[59] For children whose screening results are concerning, the next step is referral to the state early intervention program or local school system for an assessment. A level 2 evaluation is comprised of a more in-depth analysis of the developmental problems, including identifying children who are at risk for autism.[59] See Table 16–3 for recommended practices for routine developmental screening.[5,59]

Today there are research-based level 1 and 2 screening tools available for PCP. An effective screen must have a balance between its (1) sensitivity, the number of children accurately identified by the screen with the disorder, and (2) specificity, the number of children accurately identified by the screen without the disorder. The Checklist for Autism in Toddlers (CHAT) is an example of a parent report and interactive tool geared toward 18-month-olds and developed primarily for PCP to administer at well-child visits.[60] The CHAT is comprised

Table 16–3 Recommendations of Routine Developmental Screening[5]

Be familiar with the signs and symptoms of autism and refer for diagnostic evaluation
Provide developmental screening at each well-child visit (see Filipek and colleagues[5] for descriptions of instruments)
Refer for immediate diagnostic evaluation if By 12 months, the child is not babbling gesturing (eg, pointing, waving) By 16 months, the child is not using single words By 24 months, the child is not using 2-word spontaneous phrases (not just repeating) There is any loss of language or social skills at any age
Refer for immediate formal audiologic assessment when concerns include a speech, language, or hearing problem; periodic lead screens should be conducted for any child with pica
Become familiar with autism screening instruments (see Table 16–4)
Monitor the social, communication, play, and behavior development of siblings of children with autism
Refer child to early intervention (zero to three services for those less than 36 months of age) and to school system (for children older than 36 months of age) for specialized services
Become knowledgeable of the beneficial outcomes of early intervention for children with autism and the wide range of outcomes of older children
Be knowledgeable of the screening tools for older children who may have subthreshold symptoms of autism and make appropriate diagnostic referrals

of nine parent questions and five child interaction activities. Five groups of behaviors are evaluated: social interest, social play, pretend play, joint attention, and pointing to express interest in an object or event. One study demonstrated that 18-month-old children who lacked two or more of the five groups of behaviors were later diagnosed with autism at 30 months of age. Three characteristic behaviors were reported as predictive of an autism diagnosis: decreased pointing to share interest, joint attention, and pretend play.[60] In a more extensive sample of 16,000 children, the CHAT correctly identified 10 of 12 children with autism.[61] The CHAT, however, may miss some children who are later diagnosed.[62] Therefore, it is inappropriate to replace a comprehensive evaluation with a screening tool because the screening tool may be insensitive to all cases of autism. The second screening instrument, called the M-CHAT, is a modified version of the CHAT and consists of 23 yes and no parent-report items. It is designed for all parents and can be completed in the waiting room. Six items that related to social relatedness and communication discriminated children with autism from those with other ASDs.[14] Information on the sensitivity and specificity of the M-CHAT has not yet been reported.

A third instrument, the Screening Tool for Autism in Two-Year-Olds (STAT),[63] is an interactive assessment that elicits specific social and communicative behaviors from the child and is designed to discriminate children with autism from children with other developmental disabilities. The STAT consists of 12 items: two requesting, two play, four imitation, and four directing attention items. The sensitivity and specificity of the STAT are adequate. A fourth tool, the Pervasive Developmental Disorders Screening Test (PDDST),[64] is composed of three parts. Stage 1, the first part, is for use in pediatric settings; stage 2 is designed for developmental clinics, to differentiate children with autism from those with other developmental disorders; and stage 3 is for clinics that specialize in autism to differentiate autism from PDD-NOS. The sensitivity and specificity of the PDDST are not reported. A brief comparison of these tools and where they can be accessed are available in Table 16–4.

Table 16–4 Comparison of Various Autism Screening Tools

Screening Instrument	*Parent Report Items*	*Child Interaction Items*	*Target Age of Child*	*Level of Screen*
Checklist for autism in toddlers: CHAT[60]	X	X	18 months	1
Modified checklist for autism in toddlers: M-CHAT[14]	X		24 months	1
Screening tool for autism in 2-year-old children: STAT[63]		X	24 months	2
Pervasive developmental disorders screening test[64]	X		Birth to 36 months (using stage 1)	1 and 2

Treatment

Medical management for children with autism is the same as for any child. Due to the social and communication difficulties, however, children with autism may be more responsive to particular interaction strategies used during an evaluation. Tables 16–5 and 16–6 provide suggestions on ways to promote positive exchanges with individuals with autism during evaluations.

Treatment approaches alleviate behavioral symptoms and increase learning and adaptive behaviors by reversing the social and communicative impairments but do not cure the child of autism. In addition, the diagnosis of autism does not specify one treatment approach, and all intervention methods should be based on an individualized assessment of the child's needs.[65] Three broad approaches to treatment have been pursued, including pharmacologic, educational and behavioral, and alternative methods. Each mode of treatment is described.

Educational and Behavioral Treatments

Educational and behavioral treatments are the most efficacious and, therefore, the primary treatment for children with ASD. It is beyond the scope of this chapter to provide a detailed overview of the research on the educational and behavioral treatment approaches in autism, and PCPs are advised to consult other resources.[1,10,54] Unlike other treatment approaches, an educational approach has withstood the test of time and is considered the primary intervention method for autism.[53] Fortunately, as a result of federal legislation, the Individuals with Disabilities Education Act (IDEA) guarantees the education of all children with disabilities, from birth through the age of 21 years.[66] The Committee on Children with Disabilities of the AAP published a policy statement on the role of health care providers in the development and implementation of special education programming for children with disabilities.[67] This report explains the laws behind IDEA and describes components of the Individual Education Plan (IEP) and Individual Family Service Plan (IFSP) and the medical role in the IEP and IFSP. Ensuring that children with disabilities have access to services is a primary activity for every primary care physician.

Recognizing its critical role in the education and treatment of children with autism, the US Department of Education's Office of Special Education Programs requested that the National Research Council develop a committee to report on the scientific evidence regarding educational interventions for young children with autism. It is helpful to share this information with parents who have children under 8 years of age as they consider and evaluate their child's educational program.

Table 16–5 Practical Guidelines for Promoting Interactions with Individuals with Autism During an Office Visit*

Preparing for a Visit
If possible, start a conversation with the child's parents before the first visit.
Talk with parents about how their child communicates best. Also, discuss what stimuli may be most irritating or scary to the child (eg,certain objects, noises, words), and what kinds of things are reassuring or calming.
If a child's behavior is likely to make talking with parents during a visit difficult, use the phone to take histories, discuss progress made since last visit, or discuss other issues before an office visit.
Work with parents to implement a desensitization plan to your office/hospital, for procedures, and for equipment.
If possible, schedule extra time for appointments for children who have autism.
During a Visit
Being touched is very unpleasant for some children with autism; avoid touching them if it is not necessary. If you do need to touch a child, first tell him/her where and how you will touch in a neutral voice.
View the child's behavior as one way she/he communicates with you. Disruptive or aggressive behavior may be caused by confusion, fear, anxiety, pain, or other physical discomfort. Sometimes having items available that the child might enjoy may redirect anxiety and promote calmness, such as squeeze balls or toys that make noises or light up.
You may need to change the way you usually communicate with children with autism (Table 16–6).
Do not be in a hurry when interacting with these children. They may take more time than the typical child to feel comfortable with you. Also, they may need extra time to process and understand what you say and to respond to your requests.
Be kind and compassionate to parents. Nobody cares more about their children than they do.
Following a Visit
Continue supporting and communicating with parents. Phone calls can be a good way to follow up after a visit and to further discuss issues that were not thoroughly addressed during an office visit.

*Each child with autism has different characteristics and needs. Therefore, these guidelines can serve as suggestions that should be adapted to fit each situation.

Table 16–6 Communicating with Persons with Autism

Keep sentences as short and simple as possible. People with autism may process and comprehend only a portion of what you say or understand only nouns and verbs. Example: When you say, "Please stay in the room and do not go into the hallway," the child may process only the end of your sentence and think you said, "Go into the hallway."
Provide simple, clear, and concise directions. Example: Instead of gesturing at the examination table and saying, "Time for me to look at your belly," tell the child what you want him to do and make one request at a time. You could start with "Sit on table," while gesturing toward the table. Once the child is on the table, say "Lay down." When the child is laying, say "Doctor will pull up shirt." Then "Doctor will touch belly."
People with autism may not understand who, what, when, where, why, and how questions. Words with multiple meanings or meanings that are dependent on context (especially prepositions, adjectives, and adverbs) may be confusing.
Children with autism may need an extra 5 to 10 seconds to process what you say.
Some children may communicate best through means other than spoken word. Consider the use of visual pictures that depict the sequence of events of the examination, written words, gestures, and environmental cues when appropriate.
Thank children and tell them when they have done something well. Example: Say, "Good job being calm" after a procedure that is normally anxiety provoking for a child.
Use neutral tones of voice and facial expressions when telling a child what you want him/her to do.
Tell children what you would like them to do rather than what you do not want them to do. Example: Say, "Put hands on lap" rather than "Do not touch the stethoscope now."
Do not give a child a choice when the child does not have one. Example: Do not ask "May I look in your ears?" if you intend to examine his/her ears whether or not she/he gives you permission.

Adapted from Dalrymple[147] and Winner.[148]

Several types of teaching strategies have been evaluated for children with autism, and parents may consult their PCP with regard to these approaches. To name a few, these methods include structured teaching,[68] incidental teaching,[69] discrete trial training,[70] pivotal response training,[71] and functional communication training.[72] All of these approaches fall under the framework of applied behavior analysis (ABA), which is composed of systematic and planned teaching techniques designed to increase desired behaviors and decrease undesirable behaviors. No single teaching method has been reported as being more effective than any other approach; in fact, all techniques have demonstrated effectiveness, and it is likely that a multicomponent approach is most effective.[73] Regardless of any selected approach, it is essential to first generate treatment goals based on the results of individualized assessments of the child's various areas of development and make adjustments of the treatment goals and methods based on the child's progress.

Pharmacologic Treatments

Although pharmacologic treatments have not received the same research attention as behavioral or educational approaches, it is estimated that more than 50% of individuals with autism are treated with some type of medication, including psychotropics, vitamins, anticonvulsants, antidepressants, or stimulants.[74] Parents, therefore, are likely to consult with their PCP regarding these treatments. Pharmacologic treatment is considered adjunctive therapy and does not address core symptoms of autism, but rather those behaviors that interfere with learning and daily life. Medications have been used to reduce overactivity, aggression, repetitive or compulsive behaviors, self-injury, anxiety, or depression and improve attention and sleep. Descriptions of many of the classes of medications used with individuals who have autism are described below and in Table 16–7. Because little is known about the effects of some of these drugs on this population, specialists who have experience with autism should monitor pharmacologic interventions. In addition, before medication is considered for the treatment of problem behavior, it is necessary that the parents and care givers, with the assistance of trained educational and behavioral specialists, consider environmental modifications as well.[1] These types of modifications are briefly described in the educational treatment section.

Neuroleptics The neuroleptic drugs are dopamine antagonists that specifically block D_2 receptors. The degree of affinity of the neuroleptics for D_2 and other receptors depends on the medication, however. In addition to D_2 binding, other dopaminergic, serotonergic, cholinergic muscarinic, α-adrenergic, and histamine receptors may be bound. Because increased motor activity and stereotypic behavior, similar to that observed in people with autism, is seen with the activation of D_2 receptors, neuroleptics could be expected to reduce these behaviors. It has been demonstrated that some children with autism and low IQ scores have high levels of homovanillic acid, a breakdown product

Table 16–7 Medications Used for Target Behaviors

Hyperactivity, inattention, impulsiveness	α-Adrenergic receptor agonists (clonidine,* guanfacine*) Anxiolytics (buspirone) β-Blocker (propranolol) Dopamine receptor blockers, atypical neuroleptics (haloperidol, thioridazine, chlorpromazine, pimozide, risperidone, olanzapine) Opiate receptor antagonist (naltrexone†) Stimulants (methylphenidate,‡ dextroamphetamine,‡ pemoline‡) Tricyclic antidepressant (clomipramine)
Overarousal, agitation	α-Adrenergic receptor agonists (clonidine, guanfacine) Atypical neuroleptics (risperidone, olanzapine)
Aggressiveness	Anxiolytics Dopamine receptor blockers, atypical neuroleptics (haloperidol,* thioridazine, chlorpromazine, pimozide, risperidone,* olanzapine) Mood stabilizer, anticonvulsants (lithium,† valproic acid,† carbamazepine†) Noradrenergic agents (propranolol,† clonidine, guanfacine) SSRIs, tricyclic antidepressants (fluoxetine, sertraline, fluvoxamine, paroxetine, clomipramine, trazadone†)
Self-injurious behavior, stereotypy	α-Adrenergic receptor agonists (clonidine,† guanfacine) Anticonvulsants Anxiolytics β-Blocker (propranolol) Dopamine receptor blockers, atypical neuroleptics (haloperidol,* thioridazine, chlorpromazine, pimozide,* risperidone,† olanzapine) Opiate receptor antagonsist SSRIs, tricyclic antidepressants (fluoxetine,‡ sertraline, fluvoxamine, paroxetine, clomipramine)
Perseveration, obsessions, compulsions, rigidity	α-Adrenergic receptor agonists (clonidine,‡ guanfacine) Atypical neuroleptics SSRIs, tricyclic antidepressants (fluoxetine,*sertralin,* fluvoxamine, paroxetine,*clomipramine*)
Mood lability, depression	Atypical neuroleptics Mood stabilizers (lithium,† divalproex†) SSRIs,* tricyclic antidepressants*
Anxiety	α-Adrenergic receptor agonists Anxiolytic (buspirone*) SSRIs (fluoxetine,† sertraline,† fluvoxamine, paroxetine†)
Seizures, EEG abnormalities	Anticonvulsants (valproic acid, carbamazepine, lamotrigine, vigabatrin) for EEG abnormalities without seizures: glucocorticoids (corticotropin, prednisone)
Sleep disturbances	α-Adrenergic receptor agonists (clonidine,† guanfacine) Antihistamine (diphenhydramine,† hydroxyzine†) Melatonin* Sedating SSRIs (trazadone) Sedative-hypnotics (diazepam, zolpidem) Tricyclic antidepressants (clomipramine)
Social behavior	α-Adrenergic receptor agonists (clonidine, guanfacine) Atypical neuroleptics Anxiolytics β-Blocker (propranolol) Dopamine receptor blockers (haloperidol, thioridazine, chlorpromazine, pimozide) Opiate receptor antagonsist (naltrexone) SSRIs, tricyclic antidepressants
Communication	Atypical neuroleptics (risperidone, olanzapine)
Sensory issues, language	SSRIs (fluoxetine, sertraline, fluvoxamine)

EEG = electroencephalogram; SSRIs = selective serotonin reuptake inhibitors.

*First-line treatment for particular behaviors according to Tsai.[132]

†Preferred alternatives if first-line not effective (Tsai[132]).

‡First-line treatment for individuals with high-functioning autism (Tsai[132]).

of dopamine, in their cerebrospinal fluid,[75] lending support to the hypothesis that elevated dopamine levels may cause some of the behaviors exhibited by people with autism. Dopamine antagonists have been shown to decrease aggression and self-injurious behaviors, but whether this is a direct effect of the medication or the result of sedation is debated.[76] Research investigating the effects of four neuroleptics, haloperidol, pimozide, risperidone, clozapine, and other atypical neuroleptics is presented.

Haloperidol (Haldol) and pimozide (Orap) Although haloperidol acts primarily at D_2 sites, it does have some effect on other dopaminergic, α-adrenergic, and serotonergic receptors. Double-blind placebo-controlled studies involving children with autism have demonstrated a decrease in mood lability, temper tantrums, hyperactivity, stereotypies, and withdrawal and improvements in attention and social behavior.[77–79] The reduction of interfering behaviors may lead to an increase in learning as measured by discrimination tasks.[77] Haloperidol is very sedating for some children. In addition, a small number experience episodes of acute dystonia. Unfortunately, because of the extrapyramidal side effects of tardive and withdrawal dyskinesias, which have been observed in children with autism,[80,81] the use of haloperidol and related neuroleptics is limited to the treatment of severe behaviors that are unresponsive to other medications.

Pimozide mainly affects D_2 receptors. In a double-blind placebo-controlled study including children and adolescents, pimozide was shown to decrease aggressive behaviors but to have no effect on self-injury.[82] More information about the side effects of this medication is needed.

Risperidone (Risperdal), clozapine (Clozaril), olanzapine (Zyprexia), quetiapine (Seroquel), and ziprasidone (Geodon) Because haloperidol has high D_2 potency, which corresponds to extrapyramidal toxicity, the effects of atypical neuroleptics, such as risperidone and clozapine, have been studied. Other atypical neuroleptics, such as olanzapine, quetiapine, and ziprasidone, may be used, but research on the use of these drugs to treat autism needs to be completed. Risperidone is an equally potent antagonist of D_2 and serotonin receptors. Treatment of severe behaviors, including self-injury, aggression, explosivity, agitation, and hyperactivity in children and adults in open-label trials [83–85] and in a double-blind placebo-controlled study of adults,[84] has been demonstrated. Improvement in social relatedness may be observed also.[10] Sedation and weight gain, however, are common side effects.

Clozapine differs from other neuroleptics because it binds D_4, α-adrenergic, and serotonergic receptors more potently than either D_2 or D_1 receptors. Double-blind placebo-controlled studies of this medication[86] and a single-blind dose escalation study with adults[87] demonstrated decreases in self-injurious behaviors, aggression, and stereotypies. Clozapine may cause sedation, lethargy, and extrapyramidal side effects that are mild at peak effective doses.[87] However, clozapine use is limited by its most serious side effect, agranulocytosis.

Serotonin Agonists Because it has been hypothesized that serotonin function may play a role in autism, interest in medications that influence serotonin levels has arisen. Evidence for serotonin-related abnormalities in autism includes high peripheral serotonin levels, decreased responses to neuroendocrine challenge studies,[88–89] and changes induced by tryptophan-free diets.[90] In addition, antibodies to central nervous system serotonin receptors may be found in people with autism, but the research exploring this possibility has been contradictory.[91–93]

Fenfluramine An early study of the use of fenfluramine in three boys with autism suggested that using this medication may have beneficial effects on social, affective, motor, communicative, and cognitive functioning.[94] Since that study, the effect of fenfluramine on children who have autism has been studied further. This medication may not be more effective than placebos in treating autistic behaviors.[95–97] In addition, the negative effects are thought to outweigh the potential benefits of this drug.[98] Although fenfluramine increases serotonin levels over the short term by causing presynaptic release and blocking serotonin reuptake, it eventually leads to a reduction in brain serotonin and 5-hydroxyindoleacetic acid, the main metabolite of serotonin. Fenfluramine also decreases plasma norepinephrine levels and increases dopamine turnover. This drug may cause irreversible changes in serotonergic neurons,[99] decreased norepinephrine levels,[100] and cardiac side effects.[99,101]

Tricyclic Antidepressants: Clomipramine (Anafranil), Desipramine (Norpramin, Pertofrane), and Imipramine (Tofranil) Tricyclic antidepressants, such as clomipramine, desipramine, and imipramine, are named after their 3-ringed structure and are used primarily to treat depression and obsessive-compulsive disorder. The tricyclic antidepressants block norepinephrine and serotonin uptake into neurons. Clomipramine and imipramine are nonselective and inhibit the neuronal reuptake of serotonin and norepinephrine. Clomipramine also has some D_2 blocking and opioid

effects. Desipramine acts mainly as a noradrenergic agonist. It is hypothesized that these medications could be useful if the serotonin system is involved in the pathophysiology of autism.

In a double-blind study of the effects of clomipramine and desipramine, clomipramine was reported to be superior to placebo in reducing anger and obsessive-compulsive behavior. Clomipramine and desipramine were equally effective, but better than a placebo, in decreasing hyperactivity.[102] In addition, open-label trials have shown that clomipramine use may lead to improved social relatedness and reduced obsessive-compulsive behaviors, aggression, and self-injury [103–106] in individuals with autism and other pervasive developmental disorders.

Although tricyclics may prove to be very helpful in the treatment of autism, serious side effects can result from elevated serotonin levels and anticholinergic activity. For example, imipramine is not recommended for the treatment of children with autism because it may produce seizures, withdrawal, abnormal speech, and negative behavioral changes.[107] Possible side effects of clomipramine include seizures, cardiac abnormalities, aggression, tremor, agitation, sedation, weight gain, sleep problems, and constipation.[108] No extrapyramidal effects are associated with clomipramine treatment of autism.[109] Clomipramine may be more effective and produce fewer negative side effects in adolescents and adults than in children.

Selective Serotonin Reuptake Inhibitors: Fluoxetine (Prozac), Fluvoxamine (Luvox), Sertaline (Zoloft), and Paroxetine (Paxil) Selective serotonin reuptake inhibitors (SSRIs) act by blocking serotonin reuptake specifically. SSRIs have fewer side effects than tricyclics, yet, when treating depression or anxiety in people with autism, hyperactivity, agitation, and insomnia may result and smaller doses than those used to treat depression or anxiety may be needed.[110] In addition, children with autism appear to be more likely than adults to develop negative side effects as a result of SSRI use. [108] A family history of affective disorders is associated with a positive response to these medication in people with autism.[111] An overview of four SSRIs, fluoxetine, fluvoxamine, sertaline, and paroxetine, is provided.

Fluoxetine has been used successfully to reduce obsessive-compulsive behavior and depression in people who do not have autism. One open-label case series demonstrated global behavioral improvements, as measured by the Clinical Global Impression Scale, in 15 of 23 participants with autism ranging in age from 7 to 28 years.[112] DeLong and colleagues reported that children with autism make global behavioral, cognitive, language, affective, and social progress when taking fluoxetine and that those who respond to treatment are more likely to have a family history of major depressive disorder than children who have no response.[111] Fluoxetine use was associated with hyperactivity, agitation, aggression, decreased appetite, and sleep disturbance in some of the participants of the above studies.

In a double-blind placebo-controlled study, fluvoxamine was found to decrease aggression and obsessive-compulsive behaviors and improve language skills in adults with autism.[113] Side effects occurred in only a small subset of patients and included nausea and sedation that subsided with time. Fluvoxamine may have very different effects on children. In a double-blind placebo-controlled study of children and adolescents with autism and other PDDs, only 1 of 16 children benefited from the use of fluvoxamine. In addition, more side effects, including sleep disturbance, hyperactivity, agitation, aggression, ritualistic behaviors, anxiety, appetite changes, irritability, problems with concentration, and impulsivity were observed in this study[113] than in the earlier investigation involving adults.[108]

Data from open-label trials show that sertaline may be helpful in improving social interaction skills, aggression, self-injurious behavior, anxiety, irritability, transitioning behavior, and repetitive behavior in adults and children with autism, other PDDs, or mental retardation. Unfortunately, this medication has been associated with increased anxiety, agitation, and syncope in some individuals.[108] Two case reports[114,115] suggest that paroxetine may be helpful in reducing self-injurious behaviors, irritability, and tantrums in children with autism. Posey and colleagues found that agitation and insomnia may result from the use of doses above a certain level.[114]

Anxiolytics: Buspirone (Buspar) Buspirone is used in the treatment of generalized anxiety disorder, and problems with anxiety are often reported in children with autism. Buspirone is a partial serotonin receptor agonist that may also serve as a D_2 receptor antagonist. In a study of 4 children with autism taking buspirone, Realmuto and colleagues[116] found decreased hyperactivity and stereotypies in two children. Other researchers have asserted that buspirone reduces self-injurious behaviors in adults with developmental disabilities.[117]

Opiate Receptor Antagonist: Naltrexone Theories that elevated levels of β-endorphin and other brain opioids may cause self-injurious behavior in some individuals with autism have provided the basis for the hypothesis that opiate receptor antagonists, such as nal-

trexone, may reduce these behaviors. Although double-blind placebo-controlled studies have demonstrated modest decreases in self-injurious behaviors and/or motor hyperactivity with the use of naltrexone in children,[118,119] a double-blind placebo-controlled study of adults with autism showed that naltrexone produced no decrease in self-injurious behaviors and led to an increase in stereotypic behavior.[120] A study that used videotapes of six children in natural settings to judge changes in behavior suggests that naltrexone produces improvements in social behavior (including initiations), stereotypy, and attention relative to a placebo.[121] One benefit of naltrexone is that it does not have to be administered every day because of its long half-life. However, liver function tests should be monitored while one is taking naltrexone,[109] and it may cause an increase in self-injurious behaviors in some people.[122] In addition, its bitter taste may lead to decreased compliance.[121]

Stimulants Stimulants increase the activity of dopamine and other catecholaminergic neurotransmitters. Medications such as methylphenidate (Ritalin) and dextroamphetamine have been used in attempts to improve attention and hyperactivity in children with autism.[123] In fact, Aman and Langworthy assert that stimulants may be used more frequently than any other prescription medication with children who have autism.[123] Conflicting results have been obtained in studies of stimulant effects on the behavior of children with autism. Stimulants may be more effective in reducing inattention and hyperactivity in children with autism who have high-functioning autism than with those who have below-average IQ scores[109]; however, stimulants may actually increase stereotypies, activity level, fearfulness, separation anxiety, tachycardia, delusions, tics and aggression in other children.[123]

Noradrenergic Agents: Propranolol (Inderal), Nadolol (Corgard), and Clonidine (Catapres) Although there is little evidence that norepinephrine (NE) abnormalities are related to autism, drugs that reduce NE activity have been used in the treatment of autism.[124] β-Blockers, such as propranolol and nadolol, inhibit NE action by blocking NE receptors. Clonidine acts as an α2 noradrenergic agonist. Perhaps a decrease in overall level of arousal is responsible for the effect of these drugs on patients with autism. A review of three noradrenergic agonists, propranolol, nadolol, and clonidine is provided.

The results of an open-label trial suggested that propranolol and nadolol may be helpful in the treatment of autism. In this open-label study adults with autism received either propranolol or nadolol. All except one of the study participants was taking neuroleptics or mood-stabilizing drugs also. Improvements in aggression, impulsive behavior, self-injurious behavior, social skills and interest, and speech were seen.[125,126]

Clonidine has been shown to have beneficial effects in studies with double-blinded placebo-controlled designs.[127,128] In these studies, parents reported that their children were less hyperactive and irritable and more attentive, calm, and social. Clonidine may not be appropriate for treating all children with autism, however.[128] Side effects experienced by some children include fatigue, sedation, hypotension, clonidine tolerance, and increased irritability.[127,128] Guanfacine has been proposed as an alternative α2 noradrenergic agonist, which may have fewer side effects, but research with people who have autism needs to be conducted.[123]

Mood Stabilizer: Lithium Lithium is typically used prophylactically to treat mood swings in people with bipolar disorder. This drug's mechanism of action is unknown but may involve ion transport, neurotransmitters, and/or inositol phosphates. The use of lithium to change mood or behaviors in individuals with autism has not been shown to be effective unless the individual has been diagnosed with bipolar disorder or has a family history of this illness. However, lithium has been reported to decrease the aggressiveness and impulsiveness of one adult with autism when used in conjunction with fluvoxamine.[98]

Anticonvulsants: Valproic Acid (Depakote), Carbamazepine (Tegretol), and Lamotrigine The anticonvulsants valproic acid, carbamazepine, and lamotrigine have been used in individuals with autism who have epilepsy or epileptiform electroencephalograms (EEGs), without clinical seizures. There is some evidence based on studies, which did not include participants with autism, that valproic acid and carbamazepine may be helpful in reducing aggression regardless of the person's diagnosis or EEG status.[129,130] However, the efficacy of these drugs in people with autism has not been proven.[76] Belsito and colleagues found lamotrigine was not more effective than placebo in improving a variety of behaviors in a double-blind study of children with autism.[131]

Sleep Aids: Melatonin, Clonidine (Catapres), Diphenhydramine (Benadryl), Hydroxyzine (Atarax, Vistaril), Trazadone (Desyrel), Zolpidem (Ambien), Diazepam (Valium) Tsai recommends melatonin as a first-line pharmacologic treatment for sleep disturbances.[132] Melatonin is a neurohormone that is associated with the regulation of sleep-wake cycles. Although little is known about the potential side effects,[133] melatonin can be an effective treatment of insomnia in peo-

ple who have autism.[134] However, there is a concern about the quality of the products available because melatonin is not classified as a medication so that there is less scrutiny over its production. The AAP recommends occasionally withdrawing sleep aids so the effects of these medications can be monitored over time.[1]

Medication with sedating effects may be helpful in inducing or maintaining sleep. Clonidine, an α-adrenergic blocker discussed above, has been used to improve sleep patterns in children with autism.[134] Antihistamines with sedating side effects, such as diphenhydramine and hydroxyzine, may be useful in some patients; however, these medications may produce excitation rather than sedation in some children.[132]

Other drugs are available for the short-term treatment of severe sleep problems that do not respond to other medications. Trazadone is an SSRI with sedating properties. Benzodiazepines, such as diazepam, bind central nervous system GABA receptors, producing hyperpolarization and neuronal inhibition. Some benzodiazepines induce sleep, but psychological and physical dependence may develop. The hypnotic zolpidem also produces a GABA-mediated reduction in neuronal firing. Zolpidem is not a benzodiazepine and is less likely to produce dependence.

Alternative Treatments

Because neither the cause nor the cure of autism is known, alternative approaches to treatment will continue to be pursued by parents and care givers. Many of the approaches used for autism, including some of the pharmacologic treatments discussed in the previous section, have not been proven to be beneficial using rigorous studies. Some alternative treatments that have been endorsed in the past or that are currently being used with some children include the administration of megavitamins and trace minerals (particularly a pyridoxine/magnesium combination), dimethylglycine, intravenous immunoglobulin, adrenocorticotropic hormone (ACTH), and secretin. In addition, special diets (including a low-casein and/or low-gluten diet), anti-*Candida* therapy, and chelation of toxic substances (especially lead) have been used. Alternative behavioral approaches, such as Dolman-Delcato patterning, holding therapy, imitation of autistic behaviors, sensory integration and auditory integration training, and facilitated communication, have been attempted also. Impressive anecdotal accounts of success exist for most of these methods. Nevertheless, there is a paucity of well-designed studies exploring the claims made about many of these therapies. It is possible that a placebo effect or the changing natural course of the autism underlies the apparent efficacy of some of these approaches; therefore, more research is needed. Reviews of the evidence supporting or refuting the effectiveness of many alternative treatments are provided by the AAP,[10] Dawson and Watling,[135] Farber,[136] Goldstein,[137] Gupta,[16] Johnston,[138] Nickel,[139] Page,[15] and Zimmerman.[35]

Choosing Treatments

With regard to any treatment, it is essential to help families to understand the important cost-benefit issues.

What are the costs of a particular treatment? Consider the physical, emotional, and financial burdens imposed by particular therapies. Have the potential harmful short- and long-term effects of a treatment been explored? Can ongoing approaches be continued while a new one is implemented? This question is particularly important to consider if the new treatment is not successful. Parents should be reassured that it is okay to not attempt treatments that may come at too high a price for the child and his or her family, especially when the efficacy of such treatments is questioned.

What is the evidence supporting the use of a particular treatment? Anecdotal accounts are important to consider, but pediatricians can educate families about the importance of scientific investigation and how to evaluate different types of evidence. Consider the communication, language, social, cognitive, and physical characteristics and age of the individuals with which a treatment has been successful in the past when evaluating whether or not a treatment is likely to be effective with a particular child.

How will outcomes be evaluated? Health care providers can emphasize the importance of gathering information as systematically as possible about a child's baseline level of functioning and his or her progress so that future decisions about whether or not to continue a treatment can be made. Additionally, changing only one aspect of a child's treatment plan at a time is crucial in being able to attribute success to the appropriate combination of approaches.

Freeman offers other considerations.[140] These include approaching new treatments with hopeful skepticism; beware of programs that claim to be appropriate for all individuals with autism; beware of programs that obstruct an individualized treatment approach; know that there are several treatment options for individuals

with autism; know that all treatments should be based on individualized assessment; know that no new treatments should be provided until the treatment givers demonstrate assessment procedures that determine its appropriateness for the person with autism; and know that new treatments have often not been scientifically validated.

Longitudinal Outcomes of Autism

Many myths are associated with autism. People, for example, often mistakenly believe that all children with autism have hidden savant skills. Two other misconceptions are that people with autism can be cured and that as adults, individuals with autism will be dependent and nonparticipating members of their community. These latter two myths reflect the traditional method of judging outcomes, the comparison of the abilities of people with autism to the development of those who do not have disabilities. The traditional view defines outcome as a function of typical social development and levels of achieved independence,[141–145] which are best predicted by level of IQ and development of speech. It is not surprising that researchers have reported poor outcomes for most individuals with autism using these outcome criteria.[141, 142]

Because autism is a lifelong disability that causes most people with it to have persistent social problems and is often associated with some degree of mental retardation, an alternative view of outcome may be more useful and valid for parents, care givers, and treatment providers.[146] Using an alternative framework, outcome is based on the achievement of individualized goals that are established for each person. Also, this conceptualization of outcome encourages the consideration of an individual's quality of life (QOL). As individuals with autism grow older, QOL becomes especially important. A list of QOL variables for families and care givers to consider is provided in Table 16–8. Enhancing competence is the goal of intervention and results from the interactions between children and their environments. This definition of competence de-emphasizes the degree to which pathology exists only within the child and recognizes the contribution of the environment to development and learning. Competence, defined as the achievement of functional and meaningful life skills, serves as a protective factor that offsets risk factors such as underlying impairments seen in autism.[146] The evidence for the environmental influences on outcome in autism is confirmed by the positive research results on the effects of early intervention.[52] Thus, the influence of the environment on development and outcome is substantial and ongoing long after the preschool years.

Table 16–8 Quality of Life Variables to Judge Outcomes for Older Individuals with Autism[146]

Quality of Relationships with Others
Participate in activities with family members and friends
Included in family or friends' events (eg, holidays, weddings, birthdays)
Contact with family and friends as much as desired
Quality of Community Participation
Assess transportation (eg, use bus, walk, ride bike, ride in car)
Shop for items (eg, groceries, clothes, gifts)
Make choices (eg, what video to watch, movie to see, place to eat)
Attend special events (eg, sports, concerts)
Participate in extracurricular activities (eg, YMCA, bike club, philanthropic clubs)
Quality of Work Experience
Work at job that is enjoyable and provides self-satisfaction
Supported by co-workers
Able to work competently
Know job performance is good
Quality of Ongoing Learning Experiences
Opportunity to learn to try new things and meet new challenges
Opportunity to meet new people
Quality of environmental supports
Opinions and choices are considered valid and important
Is provided time and space to be alone when desired and has personal space for special possessions
Is provided enough information to make valid choices and not have to refuse them because of a lack of information, experience, or support
Quality of Personal Responsibility
Takes responsibility for personal and home chores as much as possible and in return takes pride through this accomplishment and receives recognition as contributing to the family
Bathe, wash and style hair, shave, maintain personal hygiene
Cook, clean, maintain clothes
Maintain health and wellness through understanding of nutrition, weight, medication
Manages own money

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