

Asperger's Syndrome, High Functioning Autism,
and Disorders of the Autistic Continuum
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Please note that the Appendices referred to here were not available in digitized form and are not part of this page. We apologize for not being able to include them here.

Introduction

Asperger Syndrome (AS) is a pervasive developmental disorder characterized by deficits in social interaction and motor coordination, and unusual or restricted patterns of interest or behavior. Clinically, the distinction between autism and Asperger's disorder is often made in terms of severity and in the qualitative expression of the criteria. Both syndromes are characterized by social interaction deficits, impaired communication skills, and unusual or bizarre behaviors. However, in AS, motor deficits are more pronounced, onset seems to be later, and social deficits are present without grossly impaired speech and language (Frith 1991). In DSM-IV (American Psychiatric Association, 1994) a diagnosis of AS requires the absence of any clinically significant delay in language acquisition, cognitive development, and adaptive behavior (with the exception of social interaction.) In this way, DSM-IV clearly distinguishes between the AS diagnosis and that of autism (which is characterized by more marked delays in these areas prior to three years of age). Since parameters for diagnosis have only recently been formulated such that a consensual definition can be drawn upon for research purposes, relatively little is known about AS.

Asperger's Syndrome v. High Functioning Autism

Similarities have been noted between the definition of AS and that of autism without mental retardation, or High Functioning Autism (HFA). Mental retardation co-occurs with autism in about 75% of reported cases so the DSM-IV definition allows for children who do not demonstrate measured intelligence in the mentally retarded range. Currently there are no widely accepted diagnostic guidelines specifically for High Functioning Autism (Gillberg, 1998). HFA may be most appropriately diagnosed when the criteria for autistic disorder are met (American Psychiatric Association, 1994) and Full Scale IQ exceeds the mentally retarded range. As compared to AS, HFAs generally have lower Full Scale IQs, with less apparent Verbal/Performance IQ discrepancies. In AS, Verbal IQ typically exceeds Performance IQ (Gillberg 1989, Ozonoff & Farham, 1994). There may be more of a family history in AS, especially in fathers of AS children, than in HFA (Gillberg, 1989). Motor clumsiness may be more characteristic of AS, whereas motor mannerisms may appear more in association with HFA (Gillberg, Steffenburg, & Schaumann, 1991).

Abnormalities and delays in language and communication may be more severe in HFA than in AS. Peculiarities of speech and language may nonetheless be present in children eventually identified as AS. Since most cases of AS are diagnosed at approximately age seven or later (Gillberg & Gillberg, 1989), data regarding early language development may depend largely on accurate recollections by parents, which may not be reliable (Hart, Bax, & Jenkins, 1978). The exclusionary criterion of the absence of language delays for the AS diagnosis remains controversial (Gillberg 1995). AS may also be distinguished from autism on the basis of early attachment patterns. In early childhood, AS is associated with adequate attachment to family members and with approaches to interact with peers (although inappropriate and awkward). In autism, attachment to family members is more atypical and broader social patterns are marked by withdrawal and aloofness (Klin & Volkmar, 1997).

AS may be most accurately identified through neuropsychological assessment (Lockyer & Rutter, 1970, Happe, 1994). Details of these patterns will be discussed later.

The 'Autistic Continuum'

Some researchers (e.g., Schopler, 1985) have objected to the use of a distinct diagnostic category for a disorder that represents only one point on the "autistic continuum." Frith (1991, p.5) agrees that, "Asperger syndrome is the first plausible variant to crystallise from the autism spectrum...no doubt other variants will follow." Wing (1991) concluded that both autism and AS are best regarded as falling within the continuum of social impairment which may differ in their clinical presentation due to the degree of deficit in the cognitive, language and motor realms. However, Frith asserts that, for the time being, the AS diagnostic category should be retained for clinical reasons. First, many parents of AS children will find the diagnosis more acceptable than the diagnosis of autism (which, among lay persons, may be associated with extreme withdrawal, unusual stereotypies, and self-injurious behaviors). Secondly, many children with milder forms of the disorder would be left without a diagnosis and hence, without the services and understanding they require (Frith, 1991).

In practice, children may fail to fit neatly into the diagnoses of AS or autism as sanctioned by the American Psychiatric Association (1994). When a child presents with pervasive and atypical development, the diagnosis of Pervasive Developmental Disorders- Not Otherwise Specified (PDD-NOS) may be most appropriate. Thus children who display some characteristics thought to fall on the autistic continuum, but who do not meet the criteria for AS, may receive the diagnosis of PDD-NOS. Whether a subset of PDD-NOS cases represent milder AS or autism is not known, in part because it is difficult to obtain funding for research for any disorder labeled "Not Otherwise Specified" (Klin, 1999).

History

Autism was first described by Kanner (1943, as cited in Frith, 1991). Kanner described a group of patients who, from an early age appeared aloof or indifferent to other people, resisted change, and engaged in repetitive activities. As these children grew, he observed a conspicuous absence of make-believe play, a fascination with objects which were often skillfully handled, mutism or language which seemed to lack communicative intent, and 'islets of ability' or special skills which were expressed in remarkable feats of rote memory, calculation, or other isolated skill. Shortly after the publication of Kanner's paper in the United States, Hans Asperger, an Austrian physician, published a report, in German, in which he described four boys who, despite apparently adequate verbal and cognitive skills, displayed deficits in social interaction and milder autistic behaviors (Asperger, 1944, as cited in Frith, 1991). Mental retardation was not prominent in these patients and they demonstrated deficits which resembled a milder, higher functioning form of autism. Asperger's work was published in German and his contribution went largely unrecognized until the 1980s when his work was translated by Lorna Wing (1981).

Although initially unaware of each other's work, both Kanner and Asperger used the word "autistic" to characterize the disturbances that they observed. This term had been introduced by Eugen Bleuler in 1911 to describe the extreme withdrawal from the outside world into the self, which he identified as the basic disturbance in schizophrenia (Frith, 1991). Both Kanner and Asperger independently recognized that, in contrast to Bleuler's schizophrenia, the difficulties in entering affective relationships with others seemed to be present from the beginning, among their patients. Unlike the "autism" of schizophrenia, typified by a progressive loss of contact with the external world, Kanner and Asperger's patients exhibited this difficulty early in life and with a consistent and chronic, rather than progressive, course.

Early diagnostic schemes intertwined autism with childhood schizophrenia and both were so vaguely defined as to be of no research utility (Ciaranello & Ciaranello, 1995). Although Asperger firmly held that the disorder was rooted in neurobiological causes, initially, psychodynamic theories of etiology and approaches to treatment prevailed. These theories implicated parents, specifically “refrigerator mothers” as the underlying cause of the autistic child’s delayed and atypical development. Following this theoretical framework, treatment logically necessitated residential placement of such children for extended periods (Bettleheim, 1967).

A formal diagnostic criteria for autism was not developed until the 1970s (Ritvo & Freeman, 1978, Rutter & Hersov, 1977). Autism was first included in the Diagnostic and Statistical Manual (DSM) of the American Psychiatric Association in 1980 and is now a widely recognized diagnostic entity. AS was not included until DSM-IV was published in 1994.

Clinical Presentation

The autistic continuum ranges from the most severe mentally retarded individuals with social impairment as one of several severe impairments, to highly intelligent and able persons with subtle social impairments (Wing, 1991). The various clinical pictures of autism and related disorders depend upon the combinations of different impairments, which may vary in severity independent of one another, and interact to produce various overt behaviors.

Social Interaction

The criteria for AS and autism are identical in terms of social deficits. However, in AS there are generally fewer symptoms, as well as a distinct presentation. Individuals with AS are aware of other people and desire friendship. They are often (involuntarily) socially isolated because their approaches tend to be inappropriate and peculiar. Although the AS individual may be able to correctly describe other people’s intentions, emotions, and conventions, they are unable to execute this knowledge in a spontaneous and useful manner. The lack of spontaneous adaptation is associated with an over-reliance on formalistic rules of behavior.

Autistic individuals are more apt to be withdrawn and may seem disinterested in relating to others whereas those with AS are often quite eager to relate to others but lack the requisite skills to do so (Klin & Volkmar, 1995). Meisbov and Stephens (1990) studied HFA adults’ perception of popularity among peers and found that, like their age-mates, they valued humor, attractiveness, intelligence, and athletic ability. However, they did not always agree with their age-mates perceptions of these attributes.

Communication

Although severe deficits in communication would lead to a diagnosis of autism instead of AS, several unique qualitative aspects of communication in AS have been identified. First, speech is often marked by poor prosody. Inflection and intonation typically are not as rigid and monotone as in autism. A restricted range of intonation patterns may result in utterances in which tone of voice is inconsistent or unrelated to content and communicative intent. Second, speech may also seem tangential and circumstantial. Although this may sometimes be a manifestation of a thought disorder, among AS children it is more often a reflection of their egocentric conversational approach and failure to censor output which accompanies internal thoughts. This may be evident in monologues on the topic of consuming interest (e.g., geography, railway schedules), failure to integrate what the listener can be expected to know in terms of background information; difficulty implementing the rules of conversation, such as turn-taking and topic transitioning. A third characteristic of communication among individuals with AS is verbosity. The individual with AS may launch into monologues on their favorite topic

with complete disregard of the listener's interest, nonverbal signals, or background information

Restricted Range of Interests, Activities, or Behaviors

In autism, one of the most frequently observed and most pronounced symptom is an intense preoccupation with restricted patterns of interest. In AS, this is much less commonly reported, with the exception of a preoccupation with an unusual topic about which the AS individual amasses considerable factual knowledge. Given deficits in pragmatics of social interaction, the AS individual will readily share this information, at great length and in considerable detail. The area of special interest may dominate the social interactions and activities of the AS individual (and often their families, as well). The specific subject area may change every two years or so (Klin & Volkmar, 1995).

Motor Deficits

Gross and fine motor problems are often seen in association with AS but are not part of the required criteria for diagnosis. Motor milestones may be delayed, but more typically, there are delays in the acquisition of more complex motor skills such as riding a bike, catching a ball, and climbing. AS individuals often display odd gait, poor manipulative skills, and deficits in visual-motor coordination. In autism, gross motor skills are often a relative strength.

Autistic Spectrum Disorders Through the Lifespan

Autism is a developmental disorder and its behavioral manifestations vary with age and ability. Its defining features, impairments in socialization, communication, and imagination, (Frith, 1991) are present in different forms at all stages of development. One of the earliest signs thought to be specific to autism is a lack of pointing and looking to share interest and attention with another person. However, in children with global developmental delays, this behavior would also be expected to emerge later, and hence would lack specificity to autism. Losche (1990) reviewed early home movies of autistic and nonautistic subjects and concluded that the timing and sequence of developmental gains differs between normal and autistic children only from the second year of life. It may therefore be difficult to make the diagnosis of autism with confidence prior to ages two or three years (Frith, 1991).

During the preschool years a more recognizable pattern of behavior difficulties emerge. Language may be delayed, precocious, or otherwise highly idiosyncratic (Wing, 1991). Some AS children show an early fascination with numbers and letters. Hyperlexia may be evident in which the child is able to decode words, but with little or no comprehension of meaning. Deafness is often suspected because these children seem unaware of what is going on around them. Social interaction is noticeably impaired. Make-believe play is absent and instead, the child may become fixated on simple repetitive activities or rituals. In young children deficits in 'mentalising' are common. Mentalising refers to the ability to attribute mental state (e.g., thoughts, feelings, motives) to others and to oneself. As they mature, AS and HFA children may pass mentalising tasks in formal test situations while continuing to show deficits in applying these abilities into real-life settings (Ozonoff, Rogers, & Pennington, 1991).

Among children diagnosed with autism, the grade school years bring divergent paths of development which may represent different subtypes of the disorder (Cohen, Paul, & Volkmar, 1987). With some exceptions, language and general intellectual ability go hand in hand (Frith, 1991). In AS, fluent speech is usually achieved by the age of five even though it may be noticeably odd in terms of pragmatics.

In adolescence, the AS individuals may vaguely realize that they are different from others and that they are excluded from many interpersonal relationships (Kanner, 1971). Although they amass many facts about the world, their knowledge remains fragmented and they continue to have difficulty in the meaningful, integrated execution of their

knowledge (Frith, 1991). AS individuals, despite average measured intelligence and adequate academic abilities are often described as lacking in common sense. In adulthood, the AS individual may become superficially well adapted but typically remain egocentric and isolated (Volkmar, 1987). Use of language and gestures remains stilted, and they seldom enter the natural flow of conversation. Difficulties often arise in living and working with other people and psychiatric intervention may be helpful (Frith, 1991).

Psychiatric Comorbidity

Studies of comorbid psychiatric disorders in AS subjects have been sparse. Most studies of comorbidity are limited by small sample size as well as problems in uniformly defining AS. Volkmar and Klin (1997) stated that some studies found an association between AS and Tourette's Syndrome, a finding that they were unable to replicate when examining a larger pool of AS subjects. Other disorders which may co-occur with AS include obsessive-compulsive disorder (19%); depression (15%); and ADHD (28%). Comorbidity of certain conditions may vary according to developmental level. For example, ADHD appears to be more common in younger AS individuals while depression may be more apt to emerge in adolescence and adulthood (Volkmar & Klin, 1997).

Some researchers have proposed that at least some of the adults previously identified as Schizoid Personality Disorder may actually be displaying the manifestation of AS in adulthood. Schizoid Personality Disorder is an Axis II diagnosis (APA, 1994) from adult psychiatric nomenclature. Schizoid Personality Disorder may actually represent a form of autism in adults (Wolff & Barlow, 1979). In two studies (Wolff & Chick, 1980; Cull, Chick & Wolff, 1984) children diagnosed with Schizoid Personality Disorder and those diagnosed with AS were followed into adulthood. Both groups of researchers concluded that these disorders were essentially identical. However, Wolff and Chick (1980) noted that the group identified as "Schizoid" demonstrated more distractibility and less perseveration on cognitive tasks than the subjects identified as AS. Other researchers object to the idea of collapsing these diagnostic categories (Tantum, 1988).

Etiology

Ciaranello and Ciaranello (1995) distinguish between nongenetic and genetic etiologies. Nongenetic causes are associated with disruption, usually prenatally, to the pattern of normal brain development. Genetic causes arise from mutations in genes controlling brain development. Clinically, there is no distinction between these classifications and it is assumed that both genetic and nongenetic etiologies cause damage to the same brain centers and regions (Ciaranello, VandenBerg, & Anders, 1982).

Nongenetic Causes

The most frequently cited nongenetic cause of autism is prenatal exposure to viral infection (Ciaranello & Ciaranello, 1995). Chess (1977) reported a significantly increased incidence in children born during the 1964 rubella pandemic. These children developed autism along with other birth defects characteristic of congenital rubella syndrome. Although other infectious agents have been associated with autism, these are mostly single cases (Lotspeich & Ciaranello, 1993). Varicella (Knobloch & Pasamanick, 1975), rubeola (Deykin & MacMahon, 1979), and prenatal toxoplasmosis and syphilis (Rutter & Bartak, 1971), have been linked to single cases of autism. Taken together, these and other case reports provide evidence of a possible link between prenatal infection and the disruption of brain development such that autism ensues. There is inconsistent evidence regarding prenatal, perinatal, or neonatal trauma in association with autism. In a review of the literature, Nelson (1991) was unable to find any consistent link between maternal history, pregnancy, delivery, or neonatal events with autism. However, in a study of 46 children with HFA which used normally

developing siblings as controls, it was reported that the HFA subjects had a higher frequency of reported complications during pregnancy, were more likely to have a gestation period over 42 weeks, and were more frequently first born or fourth-or-later born children (Lord, Mulloy, Weendelboe, and Schopler, 1991). In a review of the literature, Ciaranello and Ciaranello (1995) concluded that pre- and perinatal factors seem to play a larger role in cases of autism associated with mental retardation than with HFA.

Other nongenetic factors associated with autism include hypothyroidism and other medical conditions in the mother. Gillberg, Gillberg & Kopp (1992) studied five children with autistic conditions and found that three of the five had congenital hypothyroidism and the remaining two had mothers who were likely to have been hypothyroid during pregnancy. Maternal antibody formation and rejection by embryonic lymphocytes was found in association with autism in a sample of eleven mothers of autistic children (Warren, Cole, & Odell, 1990).

Teratogens have also been studied. Davis, Fennoy, and Laraque (1992) studied 70 mothers who used cocaine or engaged in poly-drug abuse during pregnancy. In this sample, 11.4% of these women gave birth to autistic children and 94% of the children born showed a pattern of delayed language development. Environmental toxins have also been implicated as possible etiologic agents in autistic spectrum disorders (Rodier, 1998).

Genetic Causes

Evidence of a genetic etiology for autism has been provided by numerous epidemiological studies. Estimates for sibling frequency have ranged from 2-6 %, 50-150 times the frequency in the general population (Rutter & Bartak, 1971). Ritvo (1989) in a study of a relatively large autistic sample, estimated the overall risk of recurrence to be 8.6%. In Ritvo's sample, if the first autistic child was male, the recurrence risk was 7%; if the first autistic child was female, the recurrence rate was 14.5%. In an overview of these studies, Ciaranello & Ciaranello (1995) concluded that all of the studies may underestimate the recurrence rate due to a tendency to stop having children after the birth of an autistic child. However, the extent to which these stoppage rules apply among parents of autistic children is unknown.

Attempts to specify a mode of inheritance for autism has been complicated by factors including: sex-influenced inheritance; reduced penetrance; variable expression of the disorder; diagnostic ambiguities; and stoppage rules. Smalley et al (1988, 1991) have proposed that autism is the result of multifactorial inheritance and genetic heterogeneity. Twin studies further support a genetic basis for autism. Studies completed by a number of researchers (e.g. Ritvo Freeman, Mason-Brothers, & Ritvo, 1985; Steffenburg, Gillberg, Hellgren, & Anderson, 1989) concur that there is a much greater degree of concordance in monozygotic than in dizygotic twins. Family studies provide further evidence of a genetic basis for autism.

Epidemiology

Autism occurs in 7-16 per 10,000 children (Wing, 1993; Baron-Cohen, 1995; Gillberg, 1995). HFA constitutes only a fraction (11-34%) of such cases. In contrast, AS may occur at a rate of 3.6 to 7.1 per 1000 children ages 7-16 years (Ehlers & Gillberg, 1993). Gillberg and Gillberg (1989) found that AS was about five times as common as autism. If these figures can be replicated and, if it becomes accepted that autism refers to a spectrum or continuum disorder, the estimate that 75% of children with autism have concomitant mental retardation would drop to only about 15% (Gillberg 1998).

The excess of autistic boys over girls was noted by both Kanner and Asperger, and is now well established, although the incidence for females is higher than originally thought. In reviews of 16 population studies of autism Wing (1993) and Gillberg (1995)

found that the male: female ratio was closer to 2-3:1. However, in HFA, the ratio is probably higher (Wing & Gould, 1979). The male:female ratio in autism tends to go down with decreasing IQ (Wing, 1981). At the lowest ability levels the ratio of boys to girls was only 2:1. At the highest ability levels, Wing's sample showed a ratio of 15:1. However, Newson, Dawson, & Everard (1982) identified a sample of 93 very able autistic people who lived all over Britain and found that only nine of them were women.

Neuropathology of Autism, AS, and HFA
The literature on the neuropathology of autism spans four decades and has produced a large body of inconsistent and often contradictory results. Autistic children, as defined in various studies, may exhibit intellectual functioning ranging from mentally retarded to intellectually superior, they may be mute or have a highly developed language skills, and stereotypic rituals and social handicaps may range from mild to severe. Given the heterogeneity of subject populations, the inconsistency in results is not particularly surprising. Postmortem and neuroimaging studies have described neuroanatomical abnormalities in Autism but the extent to which their conclusions can be generalized to AS and HFA is not known.

Autism

Structural and cellular abnormalities have been found in the hippocampus, amygdala, and cerebellum (Bauman & Kemper, 1985). These deficits have been associated with difficulties in socioemotional functioning, sensory processing, and motor planning (Aronowitz, Decaria, Allen, Weiss, & Saunders, 1997).

Evidence for pathophysiology in the cerebellum was originally proposed on the basis of clinical and neurophysiologic deficits (Ornitz, 1985). The role of the cerebellum was further investigated on an anatomic level, through autopsy studies by Bauman & Kemper (1985). The loss of granule and Purkinje cells may disrupt the developmental cytoarchitecture of the cerebellar circuitry. Immature neurons persist, and a nonfunctional fetal neuronal pattern of circuitry is retained. With maturation, the fetal neuronal pattern may be lost but it is not replaced by an adult pattern so the normal circuitry of the cerebellum does not develop.

Auditory and vestibular pathways in the brainstem were implicated in some studies (Ornitz, 1985; Ornitz & Ritvo, 1968) but these findings could not be replicated in subsequent research using a variety of anatomic, imaging, and neurophysiological methods. Although several neurophysiological abnormalities have been identified in autistic subjects, these have not been specific for autism and have also been found in non-autistic matched controls (Minshew, 1991).

Kemper and Bauman (1993) reported small, densely packed cells in the hippocampus and amygdala and suggested that this reflected an immature pattern of neuronal development in these structures. They observed neurons which appeared immature in the diagonal band of Broca, which projects cholinergic afferents to the hippocampus and amygdala (Ciaranello & Ciaranello, 1995).

Many studies have attempted to find neurochemical deficits in autism following the hypothesis that autism may be a metabolic disease which arises from a defect in some biochemical pathway. With the exception of the occasional association of autism with a known metabolic disorder, there have been no consistent findings implicating a biochemical basis for autism.

Although there is no consistent neuroanatomic defect in autism, there is evidence that implicates neuronal maturation defects, particularly in the cerebellum and limbic structures. These deficits do not appear to be reflected in the size or metabolic activity of these structures. Thus, the weight of neuroanatomic and neuropsychological evidence implicates the cerebellum and the limbic forebrain, at the cellular level, suggesting possible deficits in neuronal migration, maturation, or synaptic connectivity.

In a study which compared PET results of young men with AS and normal controls, both groups showed increased regional blood flow in the left frontal lobe in connection with mentalising tasks. However the specific area of activity differed between the two groups. These researchers suggested that difficulty in mentalising activity may be associated with dysfunctional activity in the medial portion of the left frontal lobe (Happé, Ehlers, & Frackowiak, 1996).

Unspecified abnormal electroencephalogram, auditory brainstem response, and oculomotor findings have been reported in AS and HFA, as well as in low-functioning autism (Gillberg, 1989). In a study which examined cerebrospinal fluid glial fibrillary acidic protein (GFA-p), a small group of AS children (n=4) had levels of this marker which fell in between the normal group (n=10) and an autism group, which included an HFA subsample (n=14) with high GFA-p levels equal to those of the low functioning subjects. This raises more questions regarding the etiology of AS, indicating a possible role of glial dysfunction or abnormal demyelination in the pathophysiology of this spectrum of disorders

Several case studies have documented specific medical disorders in association with AS and HFA symptomology including: tuberous sclerosis (Gillberg, Gillberg, & Ahlsen, 1994); Marfan-like syndromes (Tantam, Evered, & Hersov, 1990); Kleine-Levin syndrome (Berthier, Santamaria, & Encabo, 1992); fragile X syndrome (Hagerman, 1989); and other chromosomal anomalies (Anneren, Dahl, & Uddenfelt, 1995).

Assessment Issues

Assessment should begin with a comprehensive history. In addition to the typical practice of collecting data on early development, medical, educational, and family aspects, areas of particular relevance to the diagnosis of AS should also be explored. These include: an exploration of the onset of, or first recognition of problems; practical use of language; and special areas of interest. Emphasis should be placed on problems in social interaction, patterns of attachment to family members, development of friendships, self-concept and self-esteem, and mood presentation.

Neuropsychological Assessment

AS shares several characteristics with Rourke's (1989) concept of Nonverbal Learning Disabilities. Within the NLD framework, AS corresponds to a type of neurological impairment in which virtually the entire spectrum of NLD assets and deficits are in evidence. Many areas of functioning present a mixed bag of strengths and weaknesses. Simple and repetitive motor skills tend to improve and normalize with increasing age, however, performance on tests of complex motor skills tend to deteriorate, relative to age-based norms. Psychomotor coordination difficulties are bilateral, although a greater degree of impairment may be apparent on the left side of the body.

Bilateral tactile-perceptual deficits may similarly be more apparent on the left side. Deficits may vary according to age, with tactile imperception and suppression subsiding over time while problems with complex tactile input persist. Simple visual discrimination, especially for material that can be translated into verbal input, usually approaches normal levels. However, complex visual-spatial-organizational skills, particularly when demanded in a novel situation, tend to worsen relative to age-based norms.

Assets are often evident in the areas of rote verbal learning, however, the NLD individual may tend to overly rely on this approach and experience extreme difficulty in adapting to novel and complex situations. Demonstrated memory skills in dealing with complex verbal material is usually deficient, perhaps due to difficulties in initial comprehension of complex material.

Auditory perceptual skills are usually intact, although sometimes later to emerge as compared to normals. In linguistics, assets are apparent in a well developed vocabulary and fund of verbal information. Although sometimes judged to have unusual linguistic

prowess, deficits in this area are quite severe. Deficits are most apparent in the pragmatic use of language. Speech prosody is extremely limited. Academic functioning usually reveals the NLD pattern of deficits in mechanical arithmetic as compared to relative intact word recognition and spelling skills. Comprehension of complex reading material tends to be poor and difficulties in dealing with scientific concepts and theories are often apparent by adolescence. Errors in spelling are primarily of the phonetically accurate type.

These deficits impact interpersonal functioning in problems appreciating incongruities and humor, difficulty in adapting to novel and complex situations, and an over-reliance on rote and rigid behaviors in a constantly changing social milieu. Overall, social perception and social judgment are impaired.

Speech and Language Assessment

A speech and language evaluation should include both qualitative as well as quantitative aspects of the child's functioning. The typical test battery which focuses primarily on formal language (i.e., vocabulary, articulation, comprehension, and sentence construction) will tap only areas of strength in most AS individuals. Language assessment should thus also incorporate measures of nonverbal communication, nonliteral language (e.g., absurdities, metaphor, and humor) speech prosody (melody, volume, and pitch), and pragmatics (i.e., turn-taking, sensitivity to cues, adherence to rules of conversation). This latter group of language skills are more apt to reveal areas of significant deficits for individuals with AS. A language assessment should also note perseveration on circumscribed topics and social reciprocity.

Intervention

Securing Services

Klin & Volkmar (1997) have noted a tendency for parents and professionals to underestimate the deficits which impact on AS individuals. This is, to an extent, understandable given the proficient verbal skills, average or higher psychometric intelligence, and solitary lifestyle which may serve to mask severe deficits that become evident primarily in novel and/or socially demanding situations. Some AS children have been identified as Learning Disabled which may be successful in securing some accommodations and support but fails to address some of the most debilitating aspects of AS (e.g. deficits in reciprocal social interaction). Other AS children have been classified as PDD-NOS or autistic which often leads to placement with children who are significantly lower functioning. Such a placement may fail to make use of their unique assets. A third and most inappropriate label is "Social-Emotional maladjustment". This label is used in educational nomenclature for children with severe behavior or conduct problems who do not qualify for special education services as Emotionally Impaired.

Klin (1995) describes this as, "the worst mismatch possible, namely of individuals with a very naive understanding of social situations in a mix with those who can and do manipulate social situations to their advantage without the benefit of self-restraint."

Acceptance of AS and related diagnoses in educational settings has been limited. Children with milder autistic continuum disorders may be certified for special education services under a variety of labels. Due to the mismatch of the diagnostic category with education classifications, services may be difficult to obtain. By way of example, Appendix A shows the classifications under which an AS child is apt to receive special education services in the State of Michigan (Michigan State Board of Education, 1997). An AS child, may qualify for special education services under the classification of "Specific Learning Disability" (perhaps in reading comprehension and/or mathematics calculation), as "Speech and Language Impaired" (due to deficits in pragmatic use of language), or "Emotionally Impaired" (due to the inability to build or maintain satisfactory interpersonal relationships within the school environment). Ironically, by Michigan

eligibility definitions, it appears unlikely that a child presenting with AS would be serviced under the definition of "Autism." Some are also found ineligible for special education services because the "interdisciplinary team," does not feel that the areas of deficit have a significant impact on the child's academic functioning.

Services designed for accommodating and remediating specific learning disabilities may not address the range of problem areas in AS, especially when deficits are subtle, cause few problems for school officials, and do not directly and obviously impact on academic achievement.

To explain some of the basic concepts of AS/ NLD, the writer has developed a booklet which has enjoyed some limited, anecdotal success in enhancing awareness and understanding among a small sample of public school personnel. This is provided in Appendix B.

Although neuropsychological assessment is central to designing programs and interventions tailored to the needs of the individual, a label from the psychiatric nomenclature is essential for providing health care services and an educational classification will be needed to deem the child eligible for special education services.

Intervention Strategies

Volkmar and Klin (1997) suggest that skills and concepts be taught in an explicit and rote fashion where possible, employing a parts to whole verbal instructional approach. Learning strategies will be most effective when based on the specific pattern of strengths and deficits demonstrated in neuropsychological assessment. If motor and visual-motor deficits are identified, physical and occupational therapies are indicated.

Interventions designed to improve communication and social skills may have any or all of the following as its goals, depending on the specific needs of the individual: enhanced awareness of one's own nonverbal behaviors (e.g., the use of verbal inflection, eye contact, and gaze in social interaction); verbal decoding strategies for more accurately interpreting the nonverbal behavior of others; more integrated processing of visual and auditory stimuli; improved social awareness and perspective taking. Techniques for teaching nonverbal communication skills have been described in the literature on learning disabilities (e.g., Minskoff, 1980).

Treatments for AS and HFA are primarily symptomatic and supportive. As lifelong disorders, treatment needs and approaches will change with the individual development. Psychoactive medications may be used to target specific symptoms but are generally not indicated in AS and HFA. In high-functioning individuals with symptoms of obsessive-compulsive disorder, anxiety, or depression, pharmacotherapy may be helpful (Wing, 1991). Insight-oriented psychodynamic therapy has been extensively used to treat these disorders with little objective evidence of success (Campbell, Schopler, Cueva, & Hallin, 1996).

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