

Autism and Child Psychopathology Series

*Series Editor:* Johnny L. Matson

Jonathan Tarbox

Dennis R. Dixon

Peter Sturmey

Johnny L. Matson *Editors*

Handbook of

# Early Intervention for Autism Spectrum Disorders

Research, Policy, and Practice

 Springer

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# Autism and Child Psychopathology Series

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Jonathan Tarbox • Dennis R. Dixon  
Peter Sturmey • Johnny L. Matson  
Editors

# Handbook of Early Intervention for Autism Spectrum Disorders

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## Preface

Early intervention for children with autism spectrum disorders (ASD) is an increasingly popular and well-researched field of inquiry. However, as both popular attention and scientific activity grow, so too does the spread of misinformation. The purpose of this handbook is to put together in one place the latest in scientific information pertaining to the assessment and treatment of young children with ASD. To that end, the editors are pleased to assemble a world-class collection of scientists who have authored chapters touching on a wide variety of topics relevant to the field.

The first part of this volume, “Diagnosis and Background,” brings together a collection of chapters that lay the foundation for autism treatment. The final editing of this volume coincided with the publishing of the DSM V, which significantly changed the criteria for autism diagnosis. Therefore, many of the chapters in Part 1 dance a delicate balance between being current with the new diagnostic criteria, yet summarizing and analyzing the results of relevant research, nearly all of which was done with respect to the old diagnostic criteria.

Part 2 of this volume, “Intervention,” aims at addressing a relatively comprehensive scope of topics on evidence-based treatment for young children with autism. Hundreds of treatments for autism have been proposed but the vast majority retain little-to-no scientific support. Accordingly, all the intervention chapters in this volume focus strongly on aspects of autism intervention which have been the subject of rigorous scientific research, most of which are founded largely or wholly in applied behavior analysis. Chapter 23 rounds out the collection of intervention topics by directly addressing controversial treatments for which there is little or no evidence. Finally, Chaps. 24, 25, and 29 expand the scope by addressing issues related to family systems, general medical disorders, and multicultural issues, respectively. It is hoped that this volume provides a useful reference in the daily work of researchers and practitioners, as well as a springboard to spur further research into still under-addressed areas of assessment and treatment of children with ASD.

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## Acknowledgments

The editors would like to thank all of the world-class authors whose contributions made this volume possible. The editors would like to especially thank Brian Belva and Adel Najdowski for their organizational support for the project.

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**Part I**  
**Diagnosis and Background**

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# Evolution of Autism: From Kanner to the DSM-V

1

B. Andrew Adler, Noha F. Minshawi and Craig A. Erickson

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## Keywords

Diagnosis · History

Leo Kanner was a man who possessed the truly rare gift of being able to step back and see the big picture. At the time when his initial case series was published in 1943, the children Kanner described would have been diagnosed with childhood onset schizophrenia. At that time, children with severe psychiatric illness were frequently placed into this single, all-encompassing diagnostic category. However, Kanner recognized several distinguishing characteristics of these children which made them different from those with schizophrenia. He identified the disorder impacting this group of children as “autistic disturbances” and proposed a new diagnostic category. In his observations of a unique psychiatric disorder, Dr. Kanner saw a forest while others before had seen only trees.

In the nearly 70 years since Kanner’s first description of autistic disorder (autism), we have seen this diagnosis evolve considerably. This chapter will review the history of the diagnosis of autistic disorder. We will discuss the evolution of the diagnosis of autism from Kanner’s work and through the various editions of the Diagnostic and Statistical Manual (DSM), with some specu-

lation on the much anticipated fifth edition. Current terminology will be discussed from a historical perspective including the relatively new terms “Classic Autism,” “Atypical Autism,” and “High Functioning Autism.” Finally, a general outline is provided for use in diagnosing individuals suspected to have an autism spectrum disorder.

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## Kanner’s Autism

Leo Kanner, an Austrian born psychiatrist, founded the first psychiatry clinic devoted solely to the treatment of children at the Johns Hopkins University School of Medicine in 1930 (Alan Mason Chesney Medical Archives of the Johns Hopkins Medical Institutions *n. d.*). He was 36 years old at the time and had recently immigrated to the USA, having worked first in South Dakota before taking an offer to work at Johns Hopkins. In 1935, Kanner published the first textbook for child psychiatry (Alan Mason Chesney Medical Archives of the Johns Hopkins Medical Institutions *n. d.*). He wrote about various aspects of child psychiatry ranging from education to folklore; however, when he died in 1981, Dr. Kanner was best known for his work on establishing the diagnosis of autism.

Before Kanner published his seminal article, *Autistic Disturbance of Affective Contact* in 1943,

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no one had yet made a distinction between what Kanner called “Infantile Autism” and childhood onset schizophrenia (Matson 2008). The word autism, not coincidentally, was one of the “4 A’s” originally coined by Swiss psychiatrist Eugene Bleuler, who used it in reference to another term he also coined: schizophrenia (Gallo 2010). Bleuler took the term from the Greek “auto” meaning “self” to describe a focus on the self which he observed in individuals with schizophrenia (Gallo 2010). Persons with schizophrenia seem to actively withdraw from the outside world and enter a world marked by distortions of both perception and cognition (Gallo 2010). Kanner borrowed the term from Bleuler in order to describe children who appeared to have a similar self-turned focus (Gallo 2010).

### **Schizophrenia and Autism; Related but Unique Disorders**

The connection between schizophrenia and autism is not purely based on the historical overlap of these diagnoses, but includes clinical connections that are observed in treating individuals with these two disorders. As Kanner noted, individuals with schizophrenia manifest a variety of negative symptoms including social withdrawal which make them appear clinically similar to individuals with autism. The symptoms related to social dysfunction in both disorders can also be severely disabling, and include limited motivation to engage in social interaction, flat affect, and the lack of social and emotional reciprocity (Woodbury-Smith et al. 2010). On tasks of social cognition, patients with both autism and schizophrenia show similar functional abnormalities in multiple brain regions, including the cerebellum, insular cortex, and fusiform gyrus (Meyer et al. 2011). In autism-related irritability, psychiatrists often prescribe antipsychotic medications, originally developed to treat schizophrenia (Woodbury-Smith et al. 2010; Melville et al. 2008). In both disorders, symptoms can be very resistant to both pharmacologic and psychotherapeutic intervention.

The long-term outcomes in both autism and schizophrenia were not known to Kanner when

he made his initial distinction between the two disorders. Over time, further observations have been made by following these individuals longitudinally. Interestingly, it has been found that individuals with autism also commonly suffer from psychosis and are actually ten times as likely as the general population to develop new onset psychotic symptoms in a given year (Melville et al. 2008). Several genes have been discovered that may be associated with both autism and schizophrenia indicating, perhaps, a common neurodevelopmental pathway or disease process (Burbach and Van der Zwaag 2009). For example, some authors suggest that a common etiology of maternal infection or inflammation may be involved in both disorders (Meyer et al. 2011). Others have observed that individuals with autism have a significant decrease in life expectancy which is also characteristically seen in individuals with schizophrenia (Shavelle and Strauss 1998), although the mechanism underlying this outcome is not clearly understood.

Kanner made the crucial observation that individuals with autism differed from those with schizophrenia in that they lack a period of normal development prior to the onset of symptoms (Gallo 2010). This observation is the reason that autism is categorized as a pervasive *developmental* disorder. Symptoms of autism are present throughout early development and diagnosis is typically made in early childhood. Further differentiation includes the concrete and reality-based thought process seen in autism. In addition, hallucinations and delusions, which are the hallmark symptoms of schizophrenia, are often not appreciable in individuals with autism.

When brain imaging technology developed in the 1970s research methods turned to finding differences among and between individuals with mental illness. On a structural level, neuroimaging studies have shown that individuals with autism have increased white matter, gray matter and cerebellar white matter volumes (Stanley 2002). This is in contrast to gray matter reduction in the frontal and temporal lobes found in individuals with schizophrenia (Stanley 2002). Though there are some functional similarities in both patient populations, as mentioned previously,

neuroimaging studies have noted significant structural differences.

In addition to differences in developmental history and neuroanatomy, there are also many distinctions between autism and schizophrenia in other domains. In 1972, Sir Michael Rutter published one of the most important articles to define differences in these disorders and enabled clinicians for the first time to see the distinction between them. He noted that, although genetic factors were prominent in both disorders, parents of children with autism were often highly functional, intelligent, and of higher social class (Rutter 1972). Schizophrenia, in contrast, is one of the most inheritable disorders in all of psychiatry. Genetic studies in autism show that schizophrenia is exceedingly rare in families with autism and vice versa (Rutter 1972).

Regarding cognitive functioning, individuals with autism are much more likely to suffer from intellectual disability than individuals with schizophrenia and individuals with schizophrenia are more likely to suffer from cognitive decline over the course of their lifetime (Rutter 1972). With respect to comorbid conditions, individuals with autism are more likely to have epilepsy (Rutter 1972). Regarding distribution within the population, autism differentially affects male children at a rate of 4:1 while the gender distribution in schizophrenia is evenly split between the two sexes (Rutter 1972). Observing disease course, marked remission of symptoms can occur in schizophrenia and remission is exceedingly uncommon in autism (Rutter 1972). Much of the evidence which supported Kanner's initial hypothesis did not become available until 30 years after his publication, when Rutter used additional research findings to fully differentiate the two disorders.

### Initial Diagnostic Criteria for Autistic Disorder

Based on a series of 11 children (eight boys and three girls) described in his initial publication, Kanner provided the following observations which became his initial criteria for a diagnosis of autism (Jordan 1999; Kanner 1943).

**Profound autistic withdrawal** Kanner originally observed that his patients were “happiest when left alone, almost never cried to go with... mother, did not seem to notice... father’s homecomings, and [were] indifferent to visiting relatives...” (Kanner 1943, p. 218). Clearly, Kanner considered social dysfunction to be the most essential diagnostic criteria for patients with autism. Many of the other criteria are derived from this initial feature. For example, a “*need for sameness*” is one way in which individuals with autism avoid acknowledging others, and the effect others have on changing the surroundings. A “*tendency to be overstimulated*” is one way in which the outside world intrudes on the patient’s purposeful withdrawal. Deficiencies in *language* may be the result of individuals with autism lacking the motivation to interact effectively with others. Thus, Kanner understood that social withdrawal and a failure to relate to others had many overriding effects on the symptoms experienced by his patients.

**Need for sameness** Kanner noted that routines and the maintenance of an unchanging environment were important to his patients. He gives an example of one patient who repeatedly threads buttons in a particular order for no reason other than that this was the order in which he was originally taught (Kanner 1943). In Kanner’s observations, disturbances in routine and environment often caused significant distress and led to violent outbursts. One way he conceptualized the need for sameness was that it promoted the integrity of the individual’s world of solitude. In other words, in the absence of environmental change they could more effectively ignore the existence of others. That does little to explain why these children had restricted, repetitive, and stereotyped behaviors. In speaking, these individuals commonly used repetition in intonation, as well as in making sounds and phrases. Interestingly, Kanner’s patients required the behavior of those around them to maintain the same type of repetition and consistency.

**Excellent rote memory** Kanner noted outstanding cognitive abilities in some of the children in his series, one of whose intelligence quotient (IQ)

was upwards of 140. Four of the children in his study, incidentally, were the offspring of physicians. At the age of 2 or 3 years many of his subjects could recite a multitude of words, numbers, and poems which had very little meaning to anyone else (Kanner 1943). Kanner notes that many of the individuals in his series requested information from parents which was then incorporated into their long-term memory. He also notes that many of the parents spent a lot of time teaching their children by rote methods. Kanner implied that parents may have shared this information in order to compensate for lacking the opportunity to share other more meaningful experiences with their children.

**Mutism or language that lacks communicative purpose** Three of the patients in Kanner's case series were mute. Of the eight patients with verbal ability, words for them had a literal and inflexible meaning (Kanner 1943). He noted that many of his patients yelled nonsense words or phrases and other "irrelevant utterances" were a common part of their speech (Kanner 1943). Often words were used without the purpose of communication. Many of Kanner's patients had *echolalia*, or the repetition of words or phrases which lack an intended meaning. *Pronoun reversal* was also common. Abnormalities of speech were evident early in life and Kanner's patients frequently failed to meet developmental milestones.

**Tendency to be overstimulated** Kanner observed several sensory oddities in his series of patients. He reported that intense lights and sounds were a problem for them. Kanner felt that these stimuli were troubling to his patients because they intruded on the child's aloneness and could not be purposely avoided or ignored (Kanner 1943). Kanner also notes several of his patients had problems in feeding early in life that one may postulate to be related to a propensity towards overstimulation by textures and taste.

**Skillful relationship with objects** Kanner noted that many of his patients had dexterity with telephones, scissors and other objects, though they frequently used them without a meaningful

purpose. His patients often were quite adept at completing tasks involving tools, but would only be able to complete them if the same pattern of behaviors was carried out in the same order. For instance, one of the children would be able to fetch something for his mother in another room but only if the object was in the exact place where the child had last found it. If the object had been moved, even within plain sight, he failed at this task. Many of the children in Kanner's series had spatial reasoning abilities that were above average. For instance, regarding another patient he says "I have seen her with a box filled with the parts of two puzzles gradually work out the pieces for each" (Kanner 1943, p. 230). Although his patients were quite adept at using tools, they often used these tools in a repetitive way which ultimately limited their usefulness.

**Appearance of intelligence** Kanner noted the appearance of intelligence in his patients which he felt was different from other severely impaired individuals who suffered from mental retardation. He described these individuals as "quiet, solemn, composed... self-sufficient and independent" (Kanner 1943, p. 230). Strangely enough, this was one of the reasons that Kanner recognized autism as unique from other previously defined disorders and thus felt it important enough to mention in his publication. Kanner saw a series of physically attractive and intelligent appearing patients with normal or above normal IQ. These children shared a number of other gifts including *good rote memory*, and a *skillful relationship with objects*. At the same time they were also severely socially impaired to be considered very limited in their ability to function in almost any setting. Many of these observations highlight Kanner's tendency to identify and focus on the strengths of the patients he treated.

### **Kanner's Revised Diagnostic Criteria for Autism**

Later, Kanner revised and simplified his diagnostic criteria in order to develop a more general categorization method (Jordan 1999). His goal

was to define the most basic features underlying the disorder. Kanner felt that some features of autism (such as repetitive language) could be explained as deriving from higher order symptoms (Jordan 1999). In 1956, Kanner proposed three criteria for the diagnosis of autism. These included extreme isolation, insistence on sameness, and an onset prior to 2 years of age. Simplifying these ideas even further, Kanner stated that the one central feature of autism was the patient's inability to relate to others from early on in life (Volkmar and Lord 1998). This concept, which he initially called "profound autistic withdrawal" and later referred to as "extreme isolation," was the highest order symptom that explained all of the others (Jordan 1999). Thus, in Kanner's eyes, the term "autism" was intended to fundamentally describe a disorder of social relatedness.

### **Kanner's Disproved Assumptions**

It is notable that from his initial observations, Leo Kanner made several assumptions which later proved to be false. This is not meant to detract from the revolutionary accomplishment of his initial observations. To the contrary, many of these false assumptions have carried over to current practice and are still commonly held beliefs by mental health providers. We seek here to clarify some of these assumptions which lack empiric evidence.

Kanner assumed that children with autism had normal or above normal intelligence (Jordan 1999). More recent observations, however, show that around 75% of individuals with autistic disorder also meet diagnostic criteria for intellectual disability (Volkmar and Lord 1998). Some research has sought to find strengths of individuals with autism based on Kanner's observations of superior memory. However, the scores of individuals with autism on cognitive tests of local versus global processing has been mixed without the clear advantage in so-called "rote memory" observed by Kanner (Spek et al. 2011). More recent estimates have shown that the so-called "idiot-savants," or individuals with autism or intellectual disability who display extraordinary talents in highly specialized areas, are exceeding-

ly rare and constitute only around 1 in 2,000 individuals with autism (Heaton and Wallace 2004).

Another Kanner observation shown to be inaccurate was that autism is not associated with medical illnesses. Sixty years of observation and research have shown that this is also false. As was previously stated, as many as 25% of children who are diagnosed with autism will go on to develop epilepsy (Volkmar and Lord 1998). Additionally, numerous correlations have been found associating autism with deficits in hearing, vision, prenatal exposure to valproic acid, and genetic conditions such as fragile X syndrome.

Early explanations for autism blamed parents and minimized biological factors. This may have had origins in Kanner's observations of parents of the children in his original case series. However, assumptions about the etiology of autism further developed as a response to his initial publication. "Refrigerator Mothers" who withheld affection and failed to bond with their children were believed to be coupled with overachieving though emotionally distant fathers in order to yield autistic offspring (Volkmar and Lord 1998). Later research repudiated the theory that autism was caused by a dysfunctional child-parent relationship, showing no increased incidence of autism in orphanages in which children were brought up deprived of human contact (Volkmar and Lord 1998). Many children who were raised in social isolation had delays in language development but lacked ritualistic behavior (Rutter and Bartak 1971). Although some of what Kanner originally observed has not weathered the test of time, it is difficult to overestimate Kanner's impact on the diagnosis of autism. We now understand autism to be a complex neuropsychiatric condition associated with diffuse central nervous system dysfunction and multiple medical conditions.

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### **Diagnostic Progress Beyond Kanner**

After Kanner's original proposal of a distinct diagnostic category for autistic disorder in 1943, many alternative diagnostic systems were proposed. Creak, O'Gorman, Rendle-Short, Rutter and Wing all proposed criteria during the 1960s and 1970s (Jordan 1999). Rutter summarized

much of the prevailing opinion which emerged from the diagnostic debate in this list of four criteria which were published in 1971 (Jordan 1999; Rutter and Bartak 1971). These criteria included delay in speech, failure to develop interpersonal relationships, ritualistic and compulsive phenomena, and an onset before 30 months.

### Delay in Speech

Rutter, like Kanner before him, noted many abnormalities in the speech of individuals with autism (Rutter and Bartak 1971). He identified pronoun reversal and echolalia as the most prominent features in the speech of individuals with autism. Rutter also noted a general paucity of speech reflecting little motivation to engage others in conversation. In addition, Rutter found that individuals with autism frequently had abnormalities in tone and pronunciation.

### Failure to Develop Interpersonal Relationships

Rutter identified social dysfunction as the “key feature” of autism in agreement with Kanner’s previous work (Rutter and Bartak 1971). In his paper, he provides several examples of how this feature is manifested, including poor eye contact, little variation of facial expression, and a lack of interest in people. He saw that individuals with autism lacked normal development of sympathy and empathy and did not effectively express their emotions. This was seen very early in babies preferring not to cuddle with parents and developing very little attachment to them. Later on, these same children did not spontaneously seek out others and did not engage in normal play.

### Ritualistic and Compulsive Phenomenon

Rutter delineated four ways in which individuals with autism act in ritualistic or compulsive ways (Rutter and Bartak 1971). Some had an unvary-

ing attachment to unusual objects. Others had restricted interests and still others quasi-obsessive ritualistic behaviors. Rutter also identified the resistance to change, which Kanner had initially identified as a separate diagnostic category, falling within the realm of ritualistic or compulsive phenomenon.

### Onset Before 30 Months

Rutter noted that while the vast majority of patients developed symptoms of autism within the first year of their life, some patients developed symptoms later on (Rutter and Bartak 1971). He chose to restrict the diagnosis of autism to those individuals who presented symptoms prior to 30 months. Rutter somewhat arbitrarily chose this number since it captured the population of patients he identified to have autism and excluded other patients similarly impaired by other illnesses, such as early onset schizophrenia.

### Standardized Diagnosis Within the Diagnostic and Statistical Manual

*The Diagnostic and Statistical Manual of Mental Disorders* (DSM) did not accept the diagnosis of autism until the third edition which was published in 1980 (Volkmar and Lord 1998; American Psychiatric Association 1980). This reflected the body of work Rutter had accumulated over the previous decade since his initial publication in 1971 (Volkmar and Lord 1998). Though initially called “Infantile Autism” in the *Diagnostic and Statistical Manual—Third Edition Revised* (DSM-III-R; American Psychiatric Association 1987) changed the name to autistic disorder and removed the onset requirement which was initially present in the DSM-III (Volkmar and Lord 1998). Aside from requiring the onset of symptoms prior to *36 months*, reinstated after DSM-III-R, the criteria in the *Diagnostic and Statistical Manual—Fourth Edition* (DSM-IV; American Psychiatric Association 1994) reflected the core ideas proposed by Rutter. The three symptom clusters of social dysfunction, speech

delay, and ritualistic or stereotypic behavior were all preserved in this version, as well as in the *Diagnostic and Statistical Manual—Fourth Edition Text Revision* (DSM-IV-TR; American Psychiatric Association 2000). As Kanner understood, autism fundamentally is a disorder of social relatedness. This emphasis is reflected by requiring two criteria from this symptom cluster while requiring just one each from the other clusters (American Psychiatric Association 2000).

Diagnostic criteria for 299.00 autistic disorder (American Psychiatric Association, 2000).

(A) A total of six (or more) items from (1), (2), and (3), with at least two from (1), and one each from (2) and (3):

1. 1. qualitative impairment in social interaction, as manifested by at least two of the following:
  - a. marked impairment in the use of multiple nonverbal behaviors such as eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction
  - b. failure to develop peer relationships appropriate to developmental level
  - c. a lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g., by a lack of showing, bringing, or pointing out objects of interest)
  - d. lack of social or emotional reciprocity
2. 2. qualitative impairments in communication as manifested by at least one of the following:
  - a. delay in, or total lack of, the development of spoken language (not accompanied by an attempt to compensate through alternative modes of communication such as gesture or mime)
  - b. in individuals with adequate speech, marked impairment in the ability to initiate or sustain a conversation with others
  - c. stereotyped and repetitive use of language or idiosyncratic language
  - d. lack of varied, spontaneous make-believe play, or social imitative play appropriate to developmental level
3. 3. restricted, repetitive, and stereotyped patterns of behavior, interests, and activities, as manifested by at least one of the following:
  - a. encompassing preoccupation with one or more stereotyped and restricted patterns of

interest that is abnormal either in intensity or focus

- b. apparently inflexible adherence to specific, nonfunctional routines or rituals
- c. stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping or twisting, or complex whole-body movements)
- d. persistent preoccupation with parts of objects

(B) Delays or abnormal functioning in at least one of the following areas, with onset prior to age 3 years: (1) social interaction, (2) language as used in social communication, or (3) symbolic or imaginative play.

(C) The disturbance is not better accounted for by Rett's Disorder or Childhood Disintegrative Disorder.

As clinicians sought to further define what constituted a diagnosis of autism, they encountered numerous individuals who met some but not all of the required criteria. As a result, a new diagnostic category, pervasive developmental disorder not otherwise specified (PDD NOS), was created.

**Pervasive developmental disorder not otherwise specified** PDD NOS is another diagnosis which first appeared in the DSM-III (Buitelaar et al. 1999). PDD NOS is known by several other names including “atypical PDD” and “atypical autism” (Volkmar and Lord 1998). The DSM-III and DSM-III-R defined this term quite vaguely as a severe social impairment which does not meet criteria for autistic disorder. The DSM-IV, which defined new diagnoses of Asperger's Disorder, Rett's Disorder, and Childhood Disintegrative Disorder, defines PDD NOS as a severe social impairment not meeting criteria for any of the other PDD (Volkmar and Lord 1998). According to the DSM-IV-TR, possible reasons for selecting this diagnosis and thus reasons that the patient would not be diagnosed with autistic disorder, include such things as later age of onset or more commonly atypical or subthreshold symptomatology (American Psychiatric Association 2000). This can be primarily manifested in three scenarios. There are individuals with PDD NOS who have *mild* social dysfunction but sig-

nificant speech delay and stereotypic movements (Buitelaar et al. 1999). Another possibility is that individuals have significant social dysfunction but lack either speech delay or stereotypic movements (Buitelaar et al. 1999). Finally, this category may include individuals which have an onset of the disorder after 36 months of age (Buitelaar et al. 1999).

**Asperger's disorder** At the same time that Kanner defined autism, another Austrian named Hans Asperger published a case series of four children suffering from a slightly different type of social impairment in Austria in 1944 (Volkmar and Lord 1998). In contrast to Kanner's subjects, these children had higher language capacity and were often excessively verbal. Also unlike individuals with Kanner's definition of autism, many described by Asperger would seek out others in attempt to engage in social interaction. They would talk in depth about a narrow range of topics such as a "little professor." When speaking about their specific interests, these individuals had much difficulty appreciating social cues to stop speaking or to invite reciprocal dialog.

Asperger observed his condition to be marked by delays in social maturity, to occur almost exclusively in males, to be associated with strong cognitive skills and to run in families (Volkmar and Lord 1998). Ironically, in order to describe the social dysfunction experienced by these individuals, Asperger used the same word as Kanner (and Bleuler before him). For 40 years this condition was referred to as "Autistic Personality Disorder," a phrase coined by Asperger and first published in German (Attwood 2006). Asperger's initial case series went largely unnoticed in English-speaking countries until a review of his work was published by Lorna Wing in 1981 (Attwood 2006). Wing noted some differences in a subgroup of individuals being classified as having autism. She recognized that they were similar to individuals seen previously by Asperger, and invented the term "Asperger's Disorder" (Attwood 2006). It was not until 1991 that Asperger's writing was translated into English by Uta Frith and became widely disseminat-

ed through English speaking circles (Attwood 2006).

Diagnostic criteria, which pre-dated the DSM-IV, were proposed by Christopher Gillberg in 1991 (Attwood 2006). Gillberg's criteria included social dysfunction, narrow interest, compulsive need to introduce interest to others, peculiar or pedantic speech, deficiency in nonverbal communication and motor clumsiness (Attwood 2006). Asperger's Disorder was overlooked by the first three editions of the DSM but ultimately included in the 4th (Szatmari et al. 1995). Diagnostic criteria in the DSM-IV are dissimilar to those proposed by Gillberg, due to the omission of motor clumsiness and speech peculiarities. The DSM-IV includes no reference to pedantic speech or the need to introduce their interest to others. Similar to the way in which the DSM had defined autistic disorder, two symptoms from the social domain emphasize that Asperger's Disorder is primarily a social disturbance. Also, the social dysfunction and stereotypic behavior criteria are identical in phrasing to the same domains used to define autistic disorder. Many clinicians find that the unique variation of social dysfunction and the specific narrowed interests seen in Asperger's Disorder are distinct from autism and prefer to use Gillberg's criteria for this reason (Attwood 2006). As noted initially by Asperger, exclusion criteria in the DSM-IV include language and cognitive delay.

Diagnostic criteria for 299.80 Asperger's disorder (American Psychiatric Association 2000).

(A) Qualitative impairment in social interaction, as manifested by at least two of the following:

1. marked impairment in the use of multiple nonverbal behaviors such as eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction
2. failure to develop peer relationships appropriate to developmental level
3. a lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g., by a lack of showing, bringing, or pointing out objects of interest to other people)
4. lack of social or emotional reciprocity

(B) Restricted, repetitive, and stereotyped patterns of behavior, interests, and activities, as manifested by at least one of the following:

1. encompassing preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus
2. apparently inflexible adherence to specific, nonfunctional routines or rituals
3. stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping or twisting, or complex whole-body movements)
4. persistent preoccupation with parts of objects

(C) The disturbance causes clinically significant impairment in social, occupational, or other important areas of functioning.

(D) There is no clinically significant general delay in language (e.g., single words used by age 2 years, communicative phrases used by age 3 years).

(E) There is no clinically significant delay in cognitive development or in the development of age-appropriate self-help skills, adaptive behavior (other than in social interaction), and curiosity about the environment in childhood.

(F) Criteria are not met for another specific PDD or schizophrenia.

**Differential diagnosis** With the similar diagnostic phrasing used in the DSM-IV the question arises as to whether these three disorders are truly unique or just variations on a spectrum of disturbances marked by social impairment. According to DSM-IV definition, individuals lacking significant language delay but having impairment in social relatedness and stereotypic movements have Asperger's Disorder, though prior to the 4th edition of the DSM they would have been diagnosed with PDD NOS. Significant confusion has persisted in differentiating PDD NOS from Asperger's Disorder and misdiagnosis is common (Volkmar and Lord 1998). Additionally, as it is currently defined, Asperger's Disorder is unique from Autistic Disorder only in the lack of abnormal language and cognitive development. There arises a problem in distinguishing individuals with Autistic Disorder who have normal intelligence or so called "High Functioning Autism" from individuals who have Asperger's Disorder

which is not resolved by the DSM. By excluding many of Gillberg's defining criteria, many clinicians feel that the DSM-IV inadequately describes the unique pattern of pathology seen in individuals with Asperger's Disorder (Attwood 2006). This debate however, may be a moot point with anticipated changes the DSM-V which are discussed later.

**Rett's disorder and childhood disintegrative disorder** The two remaining diagnosis in the PDD category of DSM-IV are Rett's Disorder and Childhood Disintegrative Disorder. Rett's Disorder (also known as Rett Syndrome) is a rare disorder first observed by Andreas Rett in 1966 and described in a case series of 22 female individuals (Haas 1988). According to the largest patient registry in the world, the prevalence of Rett's Disorder is estimated at 1 per 22,800, making it 36 times less common than autism (Chakrabarti and Fombonne 2001; Kozinetz et al. 1993). Rett observed females who initially had normal development, but later developed stereotypic movements, decelerating head growth, mental retardation as well as a loss of social and language abilities (American Psychiatric Association 2000). Similar to Asperger's work, Rett originally published in German and the disorder did not reach widespread recognition by English speaking audiences for several decades until a larger case series was published in English in 1983 (Haas 1988). Several studies have found an association between Rett's Disorder and a gene mutation (MECP2) found on the X chromosome (ADAM Medical Encyclopedia 2011).

Childhood Disintegrative Disorder (also known as Heller's Syndrome) was first described by Heller in 1908 and initially called Dementia Infantilis (Volkmar 1997). Heller described a case series of six children who had developed normally but subsequently developed a severe regression and appeared to have symptoms similar to autism (Volkmar 1997). The disorder was included in the DSM-III but amid significant controversy over whether it should be considered a PDD or a Neurodegenerative Disorder (Volkmar 1997). The disorder disappeared from the DSM-III-R due to removal of the onset criterion for