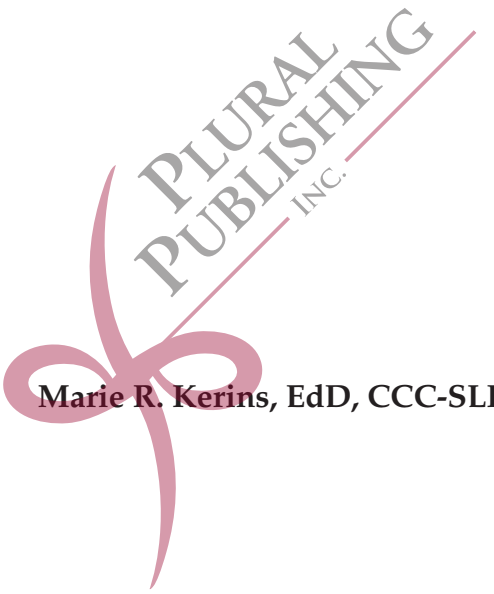
# Child and Adolescent Communication Disorders

## Organic and Neurogenic Bases

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# Contents

# Preface

One hundred years of practice, one thousand hours of assessment and treatment—that is what this collaborative text represents across the contributing authors. I am pleased and proud to present this textbook that was written by speech-language pathology and audiology faculty at Loyola University in Maryland. *Child and Adolescent Communication Disorders: Organic and Neurogenic Bases* is a textbook of disorders that we as clinicians have encountered in our own practice over the many years of working with children and adolescents. Each contributing author has had involvement and often extensive experience in both the practice and research in their respective chapter contribution. Participation in professional listservs has confirmed the need for such a text with a range of disorders from cleft palate to communication disorders concomitant with emotional and behavioral disorders.

It was important for us to include a representative sample of disorders. Organizing the content by disorder aides in text organization but also limits the reader to think beyond the restriction of the chapter's boundaries. Many of these disorders overlap and co-occur with one another. You will find this as you read through the respective chapters where there are often references to other communication disorders or other chapters. We were pleased to be able to include the new DSM-5 diagnostic criteria that serve as a common language among many health care providers. The following section will describe the text organization.

## Text Organization

The intent of this book is to introduce the reader to disorders that a speech-language pathologist will likely encounter on his or her caseload. The range of disorders varies from those that affect the client physically or organically such as cleft palate and cerebral palsy to disorders that have a more neurogenic origin such as traumatic brain injury to disorders in between that may have aspects of both organic and neurogenic origin. As research evolves, identifying the etiology of the disorders becomes more specific and objective and less behavioral. Brain studies and genetic studies further illuminate our understanding of the disorders. Each chapter begins with chapter objectives followed by definitions, information on etiology, and characteristics of the disorder.

Next, information on assessment and treatment are presented as a general introduction to these disorders giving the reader a sense of direction in both choosing assessment measures and providing intervention. As the evidence base in the profession grows, SLPs can treat clients with more scientific support. However, there is still much room for growth in developing an evidence base for many of the disorders presented. The authors have tried to bring in evidence-based approaches but will also indicate approaches that are largely theoretical and/or need further replication due to conclusions from less rigorous research designs.

It is also critical that despite the most rigorous research designs clients are individuals who are complex. There are many factors that contribute to the outcomes of treatment and at the end of the day the savvy clinician will use not only the written research but his or her own expertise and the influence of the client's individual needs.

The text is divided into six parts, all of which have been written to include and be consistent with the new DSM-5 diagnostic categories. Part I includes *Developmental Disorders* of the brain with both neurogenic and organic bases. Chapter 1 *Autism Spectrum Disorders*, Chapter 2 *Language Learning Disabilities*, and Chapter 3 *Intellectual Limitations*. Part II *Organic Bases* includes more traditionally defined organic disorders. Chapter 4 *Cerebral Palsy* and Chapter 5 *Cleft Lip and Palate* are described. Part III *Neurogenic Disorders* includes Chapter 6, *Traumatic Brain Injuries,* which affect the nervous system. Part IV *Disorders Secondary to Environmental Factors* includes a disorder that can be argued to have both organic and neurogenic bases; however, I felt it best to include this under the heading *Fetal Alcohol Spectrum Disorders*, Chapter 7. Part V, *Emotional and Behavioral Disorders,* includes a chapter entitled *Communication Disorders Concomitant with Communication Disorders;* many of these have neurogenic implications. Finally, Part VI *Central Auditory Processing Disorders* includes an overview of the implications the central auditory nervous system has on processing language. This is the last chapter, Chapter 9, *Central Auditory Processing Disorders.*

## Cover Design

As the editor, I was drawn to the cover picture illustrated by my nephew, William Reilly, for this text. Although the image of the chicken is somewhat an odd choice for a text on communication disorders it represents many thoughts and feelings that encapsulate the struggles and gifts of the children and adolescents we work with. Our students, so unique and talented in their contributions, are frequently at odds in their ability to adequately communicate their thoughts and needs whereas some also struggle to understand the essence of communication when spoken to. If asked to draw a picture of communication, a child draws an animal representing how they see themselves as a communicator—poorly understood. When asked to draw a chicken, the adolescent on the autism spectrum deliberates on which of the five breeds of chickens he/she should draw, yet fails to be able to forge friendships with his or her classmates.

Finally, the old adage of the chicken and egg comes to mind. As communication skills develop, the reciprocity between social-emotional development and skilled communication is intertwined. Communication skills develop in tandem with a rich language and literacy environment. One is dependent upon the other demonstrating that the causal relationship is not linear but reciprocal, with environmental factors contributing to language development and language development impacting how one communicates with the environment. Although difficult to disentangle, understanding their connection assists in the intervention process.

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CHAPTER 1

# Autism Spectrum Disorders

*Janet Preis*

## Chapter Objectives

Upon completing this chapter, the reader should be able to:

1. Define autism according to DSM-5 (and differentiate from DSM-IV)
2. Describe the characteristics of ASD across domains of socialization, language, and behavior
3. Describe the role of the speech-language pathologist as it relates to screening, diagnosis, and assessment
4. Explain appropriate means and content of communication assessment across life span of students with ASD
5. Describe overview of intervention for social communication
6. Delineate communication interventions according to levels of evidence

## Introduction

Autism spectrum disorders (ASDs) have become one of the most prominent and challenging disabilities for children and their families. Recent statistical information (Centers for Disease Control [CDC], 2014) indicates that one child in every 68 is classified as having an ASD. These remarkable statistics have led the CDC to encourage the health professions to consider ASD as a critical and urgent public health concern (CDC). Although many professionals work with and are responsible for assessment and intervention of people with ASD, speech-language pathologists (SLPs) play a critical role. Two of the core components of an ASD are significant impairments in communication and socialization. Although these were divided into separate "entities" in the previous diagnostic criteria (see DSM-IV-TR, American Psychiatric Association [APA] 2000), it is most appropriate (as reflected in the 2013 DSM-5 criteria) to consider these deficits as one central impairment, that of *social communication*. Therefore, due to their knowledge base and scope of practice, SLPs are on the forefront of assessment and service provision for people with ASD. This chapter examines autism spectrum disorder (referred to interchangeably as ASD and autism) as it relates to the SLP, including the diagnostic criteria, particularly the characteristics of social communication and behavior, and their influence on assessment and service provision.

In 1943, Leo Kanner first proposed the diagnosis of infantile autism. Based on

the observation of 11 children, Kanner outlined both social and communicative deficits, which, despite even the most recent diagnostic changes and research findings, are remarkably consistent with the definition currently in use more than 70 years later. Kanner used the term *autistic* to describe "the outstanding, 'pathognomonic,' fundamental disorder [that] is the children's inability to relate themselves in the ordinary way to people and situations from the beginning of life" (Kanner, p. 242), calling them "disturbances of affective contact." Until recently, autistic disorder was considered one of five conditions classified as a pervasive developmental disorder in the *Diagnostic and Statistical Manual of Mental Disorders-Text Revised* (DSM-IV-TR), along with Asperger's disorder, childhood disintegrative disorder, pervasive developmental disorder not otherwise specified, and Rett's syndrome (APA, 2000) (Table 1–1). For a diagnosis of autism, significant impairments were noted across three separate but related categories: communication, socialization, and behavior. In order for a person to qualify for a diagnosis of autism, he had to exhibit at least 6 of the 12 noted behaviors across these three categories.

Most recently, the American Psychiatric Association updated the diagnostic criteria for autism in its fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (APA, 2013). These changes are the outcome of intensive research and analysis resulting in a new category: *autism spectrum disorder* (ASD). The primary catalyst for most of the change was the lack of specificity of the previous diagnostic categories with research finding that, although the global deficit of pervasive developmental disorder (PDD) was accurate, determining *which* PDD it was among the five was not (APA, 2012). The new DSM-5 criteria meld the previously separate diagnoses of autistic disorder, pervasive developmental disorder not otherwise specified (PDD-NOS), childhood disintegrative disorder, and Asperger's disorder (a.k.a., Asperger's syndrome) into one diagnosis now entitled autism spectrum disorder (ASD), as the authors contend that they function more as a continuum of one disorder rather than four distinct entities. Rett's disorder is no longer considered a mental disorder due to its known genetic and medical origin. In addition, the categories of communication and socialization have been combined into *social communication*, requiring an exhibition of three deficits, along with two in the category of restrictive and repetitive behavior. In summary, the DSM-5 definition of ASD includes a range of severity as well as a description of overall developmental status, specific to social communication and patterns of behavior, interests, or activities.

The DSM-5 describes ASD as a lifelong, early-onset disorder characterized by marked impairments in social communication, in the presence of restricted, repetitive, and stereotypic behavior. It is a disorder that substantially impacts everyday function. Specifically, the DSM-5 definition includes the following criteria for the diagnosis of autistic disorder: "Persistent impairment in reciprocal social communication and social interaction (Criterion A), and restricted, repetitive patterns of behavior, interests, or activities (Criterion B). These symptoms are present from early childhood and limit or impair everyday functioning (Criteria C and D)" (APA, 2013, p. 53). As cited previously, current prevalence estimates yield rates of one per 68 (CDC, 2014), a significant increase from estimates less than 20 years ago of one per 1,000 (Wing,

**Table 1–1.** Progression of DSM

| *DSM (year)* | *Parent Category* | *Specific Disorders* |
|---|---|---|
| DSM-I (1952) | None | N/A |
| DSM-II (1968) | None | N/A |
| DSM-III (1980) | Pervasive Developmental Disorders (PDD) | Childhood Onset PDD<br>Infantile Autism Atypical Autism |
| DSM-III-R (1987) | Pervasive Developmental Disorders (PDD) | PDD-NOS<br>Autistic Disorder |
| DSM-IV (1994) | Pervasive Developmental Disorders (PDD) | PDD-NOS<br>Autistic Disorder<br>Asperger Disorder<br>Childhood Disintegrative Disorder<br>Rett syndrome |
| DSM-IV-TR (2000) | Pervasive Developmental Disorders (PDD) | Autistic Disorder<br>Asperger Disorder<br>Childhood Disintegrative Disorder<br>Rett syndrome<br>Same diagnoses as DSM-IV, text correction for PDD-NOS |
| DSM-5 (2013) | Autism Spectrum Disorders | No disorders, but provide the following specifiers:<br>• Associated medical or genetic condition (e.g., fragile X) or environmental factor (e.g., intrauterine exposure)<br>• Verbal abilities<br>• Cognitive abilities<br>• Severity of symptoms |



1993). The male to female ratio is almost 5:1 with little difference across racial and ethnic groups (Frombonne, 2003); non-Hispanic white children, however, are the most likely group to be identified with an ASD (CDC). Cultural factors contribute to inconsistent and even inaccurate classification and diagnosis of ASD as well as influence whether services are obtained once a diagnosis has been made (Wilder, Dyches, Obiaker, & Algozzine, 2004). Cultural differences include overall views on disabilities (e.g., acceptance, shame) and the specific perceptions on what is considered an area of need (e.g., differences in eye contact expectations across cultures).

## Epidemiology

In the 70-plus years since Kanner's original description, research has produced a myriad of products reflecting a constant search for cause, cure, and treatment. ASD by definition is a lifelong condition, and although not considered to be curable, significant progress has been made in recent years regarding potential factors influencing its presence. Although there is no single cause that can account for all cases of ASD, researchers appear to agree that both genetics and the environment play important roles (National Institute of Mental Health [NIMH], 2011). Direct examination of the brains of individuals with ASD through neuroimaging and neuropathology has found differences from those without ASD, specifically for brain growth and development, cortical connections, and neuronal organization (Grafodatskaya, Chung, Szatmari, & Weksberg, 2010). In addition, research has identified more than 100 autism risk genes (Liu et al., 2014) and in approximately 15% of the cases, a specific genetic cause of a person's ASD can be identified (Geschwind, 2011).

Some of the genetic causes are passed down in families, and others are spontaneous mutations that occur in either the egg or the sperm or very early in embryo development.

A number of studies examining the incidence of ASD in twins have provided strong support for its genetic basis (Bailey, Raspa, Olmsted, & Holiday, 1995; Folstein & Rutter, 1977; Steffenburg et al., 1989). These studies calculated a measure of genetic influence known as a *heritability index*, comparing the influence of genetics to that of the environment, each determining that this index was high. According to the NIMH, the genetic influence can be noted in identical twins who, by definition, have identical genetic makeup; both will have autism on almost 9 out of 10 occasions (2011). In addition, NIMH noted that in families with similar, but not identical genetic systems, a sibling of a person with ASD is 35 times more likely to have the disorder. Even if an ASD does not manifest itself, first-degree relatives are reported to have a greater incidence of lesser, but similar, forms of the behavioral or social language difficulties found in the disorder (Losh et al., 2009). There is also a significant association of ASD with genetic disorders such as fragile X, tuberous sclerosis, Prader-Willi, and Angelman syndromes. For example, approximately 30% and 50% to 61% of people with fragile X and tuberous sclerosis, respectively, also show traits of ASD (Sek et al., 2014; NIMH; Rutter, 2011). And the incidence of this co-occurrence, specifically for FXS, was reported to be as high as 67% for males with FXS who met "at least of one of the AD/ASD criteria on one test [ADI or ADOS], providing support to the claim that autistic behaviour is a major component of the fragile X phenotype" (p. 743, Clifford et al., 2007). However, as noted by Zafeiriou, Ververi, and Vargiami in 2007, although there is a strong correlation between autism and various genetic factors, the exact genetic background of the disorder remains unclear.

Research is not implying, however, that a genetic risk indicates automatic onset; nor does it imply that genetics is the sole cause of autism. Most recently following an extensive study of twins, researchers Hallmayer et al. (2011) concluded that the genetic influence may have been overestimated (estimated by the researchers to be a *moderate influence*

[i.e., 37%] as opposed to the previously reported 90% influence) causing the focus of research to be less on environmental triggers or causes. Their 2011 study concluded that the shared environment of twins is a critical influence on the development of ASD. The environment is considered to be a substantial "contributor" to the prevalence of ASD as, although a specific genetic cause can be identified in around 15% of cases, most cases appear to involve both the genetic risk and environmental factors that influence early brain development (Grafodatskaya et al., 2010).

The influence of multiple factors as a cause of ASD is supported by the National Institutes of Environmental Health Sciences (2012), who report that autism may be caused by an interaction between genetic and environmental factors, hypothesizing it may be triggered during early fetal development, with environmental exposures during pregnancy contributing to its cause. Some of the environmental contributors currently under examination are maternal and paternal age, illness during pregnancy, and oxygen deprivation during birth. None of these, however, have been found to be a definitive cause of ASD.

## Diagnostic Criteria

Diagnostically, ASD is a complex neurological disorder, referred to as a spectrum due to its wide range of symptoms and manifestations. In the updated DSM-5 (APA, 2013), the key components of ASD are significant impairments in social communication, and restricted and repetitive behaviors, which must be present in early childhood (although may not be noted until later) and impact everyday functioning (APA). In addition, the DSM-5 breaks down ASD into three levels of severity for the domains of social communication and behavior ranging from requiring support to requiring very substantial support (see DSM-5, p. 52, *Levels of Support*).

The designated levels of support (i.e., "requiring support, requiring substantial support, requiring very substantial support," APA, 2013, p. 52) illustrate the heterogeneity across the population of ASD. Even within the same diagnosis, a great range of functioning can occur, all causing lifelong impairment. There are, however, common characteristics and challenges central to the development of social communication skills. For the SLP it is critical to recognize and understand these core features, as they are the focus of ongoing assessment and appropriate therapeutic intervention. As we know, a diagnosis is not synonymous with an intervention plan; therefore, therapeutic approaches can both differ and overlap within the spectrum of the disorder. However, in order to most appropriately provide intervention, it is necessary first to understand how having an ASD specifically affects all aspects of communication, the realm of the SLP.

## Areas of Impairment

As noted previously, the central challenge in ASD is impaired social communication. This is a broad reaching term, encompassing the true purpose of communication where components of language and socialization merge to allow for social, human interaction. The American Speech-Language-Hearing Association (ASHA)