

Attention-Deficit Disorders and  
Comorbidities in Children, Adolescents,  
and Adults

# Attention-Deficit Disorders and Comorbidities in Children, Adolescents, and Adults

Edited by

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**Note:** The authors have worked to ensure that all information in this book concerning drug dosages, schedules, and routes of administration is accurate as of the time of publication and consistent with standards set by the U.S. Food and Drug Administration and the general medical community. As medical research and practice advance, however, therapeutic standards may change. For this reason and because human and mechanical errors sometimes occur, we recommend that readers follow the advice of a physician who is directly involved in their care or in the care of a member of their family.

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Manufactured in the United States of America on acid-free paper

First Edition

06 05 04 03 8 7 6 5

American Psychiatric Publishing, Inc.

1000 Wilson Boulevard

Arlington, VA 22209

[www.appi.org](http://www.appi.org)

### **Library of Congress Cataloging-in-Publication Data**

Attention-deficit disorders and comorbidities in children, adolescents, and adults / edited by Thomas E. Brown.— 1st ed. p. ; cm.

Includes bibliographical references and index.

ISBN 0-88048-711-9 (alk. paper)

1. Attention-deficit hyperactivity disorder. 2. Comorbidity. I. Brown,

Thomas E., Ph.D.

[DNLM: 1. Attention Deficit Disorder with Hyperactivity.

2. Comorbidity. WS 350.8.A8 A883 2000] RC394.A85 A88 2000 616.85\_89—dc21

99-047756

**British Library Cataloguing in Publication Data**

A CIP record is available from the British Library.

*To my wife, Bobbie,*

*With love and gratitude for all you are, all you give, and all we share together.*

TEB

Perhaps the most indispensable thing we can do as human beings, every day of our lives, is remind ourselves and others of our complexity, fragility, finiteness and uniqueness.

Antonio R. Damasio (1994)

*Descartes' Error: Emotion, Reason and the Human Brain*

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# Acknowledgments

The idea for this book emerged in casual conversation over lunch with colleagues. Several were complaining of their difficulty in locating persons with uncomplicated attention-deficit/hyperactivity disorder (ADHD) to participate in a research project. Heads nodded in agreement when someone commented on the irony that most research studies focused on “pure” ADHD cases, while the ADHD of most children, adolescents, and adults seen in our clinical practices is complicated by multiple comorbidities. That was 7 years ago.

In the intervening years, we have learned much more about the complexities of ADHD and its comorbidities across the life span. Some of our understanding has come from research, some from conversations with colleagues, and much of it from talking with thousands of boys and girls, men and women who have come seeking assessment and treatment for their frustrating struggles with ADHD and related disorders. I am deeply grateful to my patients, whose sharing of their unfolding life experiences continues to teach me about the amazingly complex interaction of human impairments and strengths.

Many colleagues have enriched and helped to refine my understanding of ADHD and its comorbidities. I appreciate the contributions of Dr. G. Davis Gammon to my earlier work and am grateful for ongoing collaboration and support from Dr. Don Quinlan, Director of the Clinic for Attention and Related Disorders at Yale. I am also grateful to Dr. Tom Spencer, Dr. Rosemary Tannock, and Dr. Tim Wilens, from whom I have learned much as we have worked together in teaching many courses and symposia.

Many thanks are due to all of the colleagues who contributed chapters to this book. Every one of these outstanding researcher–clinicians is overworked in an extremely demanding schedule of research, teaching, and clinical practice. Their generosity in giving precious time, effort, and care to prepare and revise their chapters is deeply appreciated.

A special word of thanks is due to the very competent staff at American Psychiatric Press, Inc., for their support and hard work in moving this book from a germinating idea to a finished volume. Special thanks to Dr. Carol Nadelson, Editor-in-Chief and CEO, and Claire Reinburg, Editorial Director, for their kind support and their patience with multiple delays; to Pam Harley, Managing Editor, Books, who has skillfully coordinated the production process; to Greg Kuny and Beth Rosenfeld, who have done a masterful job of editing the manuscript; to Pam Maher and Abdul Kargbo for their careful preparation of the manuscript for press; and to Bob Pursell, for his insight and expertise in marketing.

Most of all, I am grateful to my wife, Bobbie, for her boundless love, delightful wit, and generous support in the lengthy vicissitudes of this project and throughout our many years together. In countless ways, our daughter and son, Liza and Dave, as well as my mother and mother-in-law, Dorothy and Rose, have also provided love and support that nurtures my work and sustains and enriches my life. For all my family, I am continually grateful.

Thomas E. Brown, Ph.D. Hamden, Connecticut October 15, 1999

# Introduction

For many years assessment and treatment of the disorder currently known as attention-deficit/hyperactivity disorder (ADHD) seemed relatively simple. Typically, the diagnosis was made by a pediatrician when parents or teachers reported a child, usually a young boy, to be disruptive, with extraordinarily hyperactive and impulsive behavior. Such children were usually placed on a regimen of stimulant medication, to be continued until puberty, at which time it was expected that the disorder would naturally remit.

Over the past decade extensive research and clinical experience have dramatically altered our understanding of this disorder. We now know that ADHD afflicts not only young boys who are hyperactive but also a much wider segment of the population. It results in impairment in a substantial percentage of children, adolescents, and adults of both genders, probably at least 5% of the population, many of whom are not hyperactive (Barkley 1990; Gaub and Carlson 1997). For over 50% of those who have ADHD in childhood, these impairments persist into adulthood, although often they do so with a changing profile of complex symptoms (Biederman et al. 1993; Spencer et al. 1994; Weiss and Hechtman 1993; Wender 1995).

No longer can ADHD be viewed as a simple or insignificant behavioral disorder. Recent studies demonstrate that for many of those affected, the ADHD diagnosis encompasses chronic impairments in cognitive functions that are essential to consistent, effective adaptation in school, work, and family and social relationships. Severity of these impairments can range from chronic frustration and underachievement to devastating inability to complete one's education, to hold a job, or to maintain a relationship.

“Inattention” impairments of ADHD encompass not just chronic difficulties in listening to a speaker but also significant problems in a wide variety of cognitive functions, including ability to activate and organize for work, ability to sustain alertness and effort for work, and ability to utilize short-term “working memory” effectively (Barkley 1996, 1997; Brown 1995, 1996; Douglas 1988, 1999; Pennington et al. 1996; Pennington and Ozonoff 1996).

Some of these attention impairments are included or adumbrated in current diagnostic criteria for ADHD, but emerging findings suggest that persons with ADHD, particularly affected adolescents and adults, have a wider range of chronic cognitive impairments than those incorporated in DSM-IV. Increasingly, researchers in this field are recognizing that ADHD inattention symptoms overlap with “executive functions,” which play critical and complicated roles in integrating, regulating, and managing ongoing mental activity (Barkley 1997; Castellanos 1999; Tannock and Schachar 1996).

Since this complicated set of ADHD-related cognitive impairments is linked to attention but is not essentially linked to hyperactive-impulsive behavior, the persistence of the term “hyperactivity” in the name of this disorder may be misleading. The title of this book uses the term *attention-deficit disorders* to emphasize the centrality of attention impairments, with or without hyperactivity, in the disorder. The plural form is utilized in the title to emphasize the diversity of ways in which attention-deficit disorders are manifest in children, adolescents, and adults. Within the book the terms *attention-deficit disorder* and *attention-deficit/hyperactivity* are used interchangeably.

Attention-deficit disorders are complex not only because of the variegated cognitive functions impaired in inattention. Very often these disorders are further complicated by comorbidities. The

term *comorbidities* refers to other psychiatric disorders that impair an individual concurrent with his or her primary diagnosis. Many studies have found that over 50% of persons diagnosed with an attention-deficit disorder also meet the diagnostic criteria for one or more additional psychiatric disorders—for example, mood disorder, anxiety disorder, substance use disorder, learning disorder, or behavior disorder. The problem is not only that persons with an attention-deficit disorder can have other concurrent psychiatric disorders; there is evidence that individuals with an attention-deficit disorder have a markedly increased probability of having one or more additional psychiatric disorders (Biederman et al. 1991; Jensen et al. 1997).

Researchers and clinicians are now recognizing that appropriate assessment and treatment of persons with attention-deficit disorders requires careful attention to a wide range of cognitive impairments and also to the extensive variety of other psychiatric disorders that may be comorbid. Comorbid disorders may mask or be masked by symptoms of an attention-deficit disorder and thereby confuse the diagnostic process. Comorbidities, recognized or unrecognized, may also seriously complicate the process of treatment for attention-deficit disorders.

Those who assess, treat, educate, and care for children, adolescents, or adults with attention-deficit disorders need to appreciate and understand the complexity of these disorders and their comorbidities. It is not sufficient for evaluation of a person with an attention-deficit disorder to include only an assessment of possible attention-deficit disorder symptoms. Nor is it sufficient in the treatment of persons with attention-deficit disorders to focus only on symptoms of the attention-deficit disorder without regard for possible psychiatric comorbidities. Clinicians who provide assessment and treatment for attention-deficit disorders need to be well informed about the complexities of attention-deficit disorders and their comorbidities as well as about the implications of these complexities for effective therapeutic and educational interventions.

This book is an effort to consolidate what is currently known about attention-deficit disorders and their common comorbidities as they occur in children, adolescents, and adults. The chapters are written by outstanding clinical researchers, many of whom work on the cutting edge of current research in this field. Each chapter summarizes what its author or authors see as important in what is currently known about its particular topic, but all of the chapters should be regarded as reports of current understandings that await elaboration and correction by findings from additional research.

Each chapter incorporates many references to the relevant scientific literature so that readers can examine directly the details of the studies being summarized. The authors have attempted to focus their discussion of research toward practical implications for clinicians, educators, and others concerned with direct service to individuals with attention-deficit disorders. Although there are similarities among the views of many of these authors, there is also considerable diversity within the group in the way that they currently conceptualize attention-deficit disorders and comorbidities and their assessment and treatment.

## **Overview of the Book**

The first section of this book begins with two chapters in which the authors describe the emerging understandings of the nature of attention-deficit disorders and of genetic factors implicated in their transmission. These are followed by 10 chapters, in each of which is described a specific cluster of psychiatric disorders often found to be comorbid with attention-deficit disorders, including mood disorders; anxiety disorders; learning disorders; oppositional defiant, conduct, and aggressive disorders; obsessive-compulsive disorder; sleep disorders; substance abuse and substance use disorders; tic disorders; and developmental coordination

disorders. Each of these chapters describes what is currently known about a particular cluster of comorbidities and how such comorbidities may have an impact on the patient and modify the process of assessment and treatment. The section concludes with one chapter describing problems in assessing and treating attention-deficit disorders in preschoolers and another summarizing various types of outcomes of individuals with attention-deficit disorders as these have been reported in existing longitudinal studies.

The second section of the book focuses on assessment and treatment interventions for attention-deficit disorders and comorbidities in children, adolescents, and adults. Beginning with a chapter on clinical assessment, the section also includes chapters on pharmacotherapy, psychosocial interventions, cognitive therapy, and educational interventions for individuals with attention-deficit disorders and comorbid disorders. The volume concludes with a chapter on tailoring of interventions for individuals with attention-deficit disorders, in which is emphasized the need for individualizing treatment to take into account comorbidities and combinations of comorbidities as well as other specifics of the individual, his or her family, and their social setting.

*Thomas E. Brown, Ph.D.*

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## 1

## **Emerging Understandings of Attention-Deficit Disorders and Comorbidities**

*Thomas E. Brown, Ph.D.*

Over the past 10 years, interest in attention-deficit disorders (ADDs) has burgeoned throughout North America. Once seen simply as behavioral disorders in young boys, ADDs are now being more widely recognized in females (Arnold 1996; Biederman 1999; Gaub and Carlson 1997; McGee and Feehan 1991), in adolescents (Barkley 1990; Biederman et al. 1996b, 1998; Schaughency et al. 1994; Weiss and Hechtman 1986, 1993; Wilson and Marcotte 1996), and in adults (Biederman et al. 1993; Millstein et al. 1997; Spencer 1994; Wender 1995).

Increasing numbers of boys and girls, and men and women, are being diagnosed with and treated for chronic attention problems and related difficulties that have seriously disrupted their functioning in school, at work, and in their family and social relationships. Many patients report that treatment has produced dramatic and continuous improvement in their long-standing ADD symptoms. Anecdotal reports on the usefulness of medications for alleviating ADD symptoms are supported by research evidence of the efficacy of medication treatment for preschoolers (Musten et al. 1997), children (Spencer et al. 1996), adolescents (Smith et al. 1998), and adults (Spencer et al. 1995).

Some question the reasons for the increased number of people who are reporting ADD

symptoms, suggesting that this may simply be a passing fad in psychiatric diagnosis. Yet clinical experience in assessing those who seek treatment for and are diagnosed with ADDs makes clear the substantial impairments and genuine suffering experienced by persons with these disorders, and clinical experience in the treatment of them makes clear the substantial and continuous improvements in multiple life functions that result from effective treatment of ADD symptoms.

Moreover, clinical impressions on the validity of the diagnosis of attention-deficit/hyperactivity disorder (ADHD) and the efficacy of ADHD treatment are substantially supported by empirical research. Clearly, more people are being recognized as having ADHD and are being successfully treated for it. This has led to increasing concern among some professionals and laypeople, who wonder why the number of persons with ADHD has been increasing so rapidly.

It seems likely that what has been increasing is not the number of persons who have inattention problems, but the number of such persons who recognize these impairments as symptoms of a treatable disorder that occurs in females as well as males and in adolescents and adults as well as young children. Individuals, male and female, both young and old, with chronic attention impairments have probably existed in every generation; however, they may have been seen simply as underachieving, lazy, immature, or unmotivated. Among those who erratically demonstrated the ability to perform well in school or at work or to have successful social relationships but seemingly lacked the ability or motivation to sustain success may have been many who had the disorder currently recognized as ADHD.

Such individuals now may seek treatment for ADHD because they have heard that this disorder can be treated effectively with medication. As increasing numbers of children, adolescents, and adults have achieved significant improvement from ADHD treatment, reports of their diagnosis

and successful treatment have spread, reaching family members, friends, and acquaintances who have struggled with similar difficulties.

As teachers, psychologists, and physicians witness improvements obtained by treatment of ADHD symptoms, many are becoming more alert to these symptoms and are suggesting appropriate assessment and treatment for those who might benefit. Educational and advocacy efforts by support groups such as CHADD (Children and Adults with Attention Deficit Disorder) and the National Attention Deficit Disorder Association, as well as books and widespread media coverage, have further expanded public and professional awareness of ADHD as a treatable disorder. Yet increasing popular interest in ADHD appears to be sustained not so much by the media but by expanding awareness of the successful treatment of ADHD symptoms in adults as well as in children.

Recognition of the validity of ADHD and the effectiveness of its treatment was supported by a recent report from the American Medical Association (AMA). In response to public and professional concern regarding possible overdiagnosis of ADHD and possible overprescription of stimulant medications used to treat the disorder, the AMA commissioned a study to review relevant evidence. The study concluded, "ADHD is one of the best-researched disorders in medicine, and the overall data on its validity are far more compelling than for most mental disorders and even for many medical conditions" (Goldman et al. 1998, p. 1105). In addition, the report noted the potentially deleterious impact of this disorder: "ADHD is associated with significant potential comorbidity and functional impairment, and emotional problems at subsequent stages of life" (p. 1106). The AMA report also noted the effectiveness of medications used to treat ADHD, concluding: "Medications have been unequivocally shown (i.e., by double

blind, placebo-controlled studies) to reduce core symptoms of hyperactivity, impulsivity, and inattentiveness. They improve classroom behavior and academic performance; diminish oppositional and aggressive behaviors; promote increased interaction with teachers, family and others; and increase participation in leisure time activities” (Goldman et al. 1998, p. 1103). The report further noted, “Adverse effects from stimulants (used to treat ADHD symptoms) are generally mild, short-lived, and responsive to dosing or timing adjustments” (p. 1104).

Although researchers and clinicians in the United States and Canada have pioneered the assessment and treatment of ADDs, recognition of these disorders as a significant public health problem has been gradually expanding worldwide. Differences in cultural expectations, diagnostic criteria, and ascertainment methods cause variability in reported incidence rates from one country to another (Mann et al. 1992), but significant rates of ADDs have been recognized in countries as diverse as New Zealand (Anderson et al. 1987; Fergusson et al. 1993), Germany (Baumgaertel et al. 1995), Italy (Gallucci et al. 1993), China (Leung et al. 1996; Tao 1992), Japan (Kanbayashi et al. 1994), India (Bhatia et al. 1991), as well as in Puerto Rico (Bird et al. 19885).

As increasing numbers of medical and educational professionals in other countries have become aware of ADDs among their patients and students, and laypeople recognize these disorders in their offspring, interest has been growing steadily in learning about the disorders and their treatment. Well- attended national conferences on ADDs have been held recently for medical and educational professionals, as well as laypeople, in England, Norway, Sweden, Venezuela, India, and many other countries. With increasing recognition of these disorders internationally, there has been increasing interest worldwide in improving assessment and treatment services for

children, adolescents, and adults with ADDs.

One important result of the burgeoning numbers of people being assessed and treated for ADDs is an increase in efforts to identify and understand ADD impairments and why they often respond to existing treatments. As more information about ADDs has accumulated, particularly in adolescents and adults, understanding of these disorders has begun to shift from an emphasis on disruptive behavior, which has long characterized them.

### **Former Understanding of ADDs as Disruptive Behavior Disorders**

Currently, the diagnosis for chronic symptoms of inattention remains closely linked to problems with hyperactive/impulsive behavior, even though research has demonstrated that many individuals with ADDs are not hyperactive. This linkage has persisted from early studies of young children who were hyperactive and disruptive in school. Researchers studying these children noticed that many had chronic problems with sustaining attention. As a result, attentional problems were incorporated into diagnostic criteria for hyperactive/impulsive behavior disorders and have remained closely linked to disruptive behavioral diagnoses ever since.

The first major recognition of inattention symptoms as central to ADDs came with the publication of DSM-III (American Psychiatric Association 1980), which shifted emphasis to inattention as the primary symptom of the disorder known until then as “hyperactivity.” In DSM-III the name of the diagnosis changed from “hyperkinetic reaction of childhood” to “attention deficit disorder” and diagnostic criteria for ADD with and without hyperactivity were established. For 7 years, criteria for diagnosing ADD without hyperactivity were included in

DSM.

Recognition of attentional impairments as central to ADDs was lost in DSM-III-R (American Psychiatric Association 1987). The revision committee challenged the notion of ADDs without hyperactivity because of a lack of supporting research. In DSM-III-R, diagnostic criteria for ADDs without hyperactivity were dropped and inattention symptoms were combined into a single list, along with symptoms of hyperactivity and impulsivity. This hybrid diagnosis was then named “attention-deficit hyperactivity disorder” and remained classified as a disruptive behavior disorder.

Although numerous research studies in the late 1980s clearly demonstrated the validity of the diagnosis of ADD without hyperactivity, there were no officially accepted diagnostic criteria for this type of ADD from 1987 until DSM-IV was published (American Psychiatric Association 1994). DSM-IV recognized a “predominantly inattentive type” of ADD, which required no symptoms of hyperactivity or impulsivity, but the name of the diagnosis remained “attention-deficit/hyperactivity disorder.” The slash between the defining terms was introduced to allude to the separability of symptoms of the hybrid disorder ADHD.

Despite these changes in the official definition of this diagnosis, much of the professional and lay literature about ADHD continues to describe the disorder as characterized by three cardinal symptoms: inattention, hyperactivity, and impulsivity. Few publications focus on attention impairments of ADHD without linking these to hyperactive and impulsive behavior. As a result, the disruptive behavioral focus continues and individuals whose significant attention impairments are not accompanied by hyperactive-impulsive symptoms are less likely to be recognized as having ADHD.

## **Inattention**

### *Inattention Without Hyperactivity*

Lahey and Carlson (1991) reviewed the substantial research supporting the validity of the diagnosis ADD without hyperactivity. Among studies reviewed were several that demonstrated, by factor analysis, that ADD symptoms yielded two separate factors: one comprising chronic inattention and the other including combined hyperactivity and impulsivity. These studies showed that children whose ADDs were with hyperactivity-impulsivity (ADD/H) differed from children whose ADDs were without hyperactivity (ADD/WO) in their emotional and behavioral patterns, peer relationships, and cognitive functioning.

Using this base of research, the DSM-IV committee on ADHD conducted a field study to test new diagnostic criteria for ADDs with and without hyperactivity-impulsivity. Results of this study led the committee to recognize that an individual may have an ADD without symptoms of hyperactivity or impulsivity. The name given to this diagnosis was “attention-deficit/hyperactivity disorder, predominantly inattentive type” (Lahey et al. 1994).

In addition to the inattentive type of ADHD, another new diagnosis, “predominantly hyperactive-impulsive type,” was introduced in DSM-IV. This category was added primarily to identify preschool children who were extremely hyperactive, though their young age precluded identification of significant problems of inattention. This category may turn out to identify a prodromal form of combined type ADHD.

“Attention-deficit/hyperactivity disorder, combined type” is offered in DSM-IV to diagnose individuals with significant problems with both inattention and hyperactivity-impulsivity.

Although DSM-IV introduced the predominantly inattentive type of ADHD, this name

perpetuated the continuing linkage of inattention to hyperactivity.

Subsequent research on the incidence of the various types of ADHD identified in DSM-IV has indicated significant differences in frequency of each type, depending on the setting. In clinical psychiatric settings, the combined type is found with the greatest frequency: 45%–62% of clinical samples of patients with ADHD (Eiraldi et al. 1997; Faraone et al. 1998; Paternite et al. 1995). Probably for this reason, the combined type has claimed the largest share of attention in most research done on ADHD. Yet, in the wider community, the predominantly inattentive type is most common. According to epidemiological studies, prevalence of the predominantly inattentive type ranges from 4.5% to 9.0% of the general population of children, whereas the combined type occurs at frequencies ranging from 1.9% to 4.8%. The predominantly hyperactive-impulsive type is even less common, ranging from 1.7% to 3.9% (Baumgaertel et al. 1995; Gaub and Carlson 1997; Wolraich et al. 1996).

Shortly after DSM-IV was published, additional research documented significant differences in the life course of the two primary types of ADHD. A 4-year longitudinal study by Hart et al. (1995) demonstrated, in a sample of boys diagnosed with DSM-III-R ADHD, that inattention symptoms tended to persist, whereas hyperactivity-impulsivity symptoms tended to diminish. In Australia, Levy et al. (1997) found a similar pattern in a 3-year study of a large sample of male and female children with ADHD. Together, these studies highlight the developmental separability of inattention symptoms from hyperactivity-impulsivity, even when both symptom sets initially coexist (e.g., a child with combined-type ADHD).

### *Persistence of Inattention Into Adolescence and Adulthood*

The phenomenon of hyperactivity symptoms diminishing with age may account for the long-held

assumption that children with ADDs tend to outgrow these disorders in their teens. When only behavioral symptoms are considered, many individuals with ADDs in childhood may be seen as outgrowing their symptoms in adolescence. Yet when attention impairments are present in childhood, with or without hyperactivity, they tend to persist into adolescence and adulthood, often in ways that create problems for individuals at school, at work, and in social relationships (Biederman et al. 1998; Millstein et al. 1997).

Achenbach et al. (1995) published results of a large 6-year national study of male and female adolescents making the transition into adulthood. Results indicated that childhood attention deficits tend to persist into early adulthood, usually without persistence of significant problems with hyperactivity-impulsivity. Attention problems were found to be not only more persistent into adulthood than hyperactivity-impulsivity symptoms but also more impairing. In young adulthood, attention problems were found to be associated with significant impairments in employment and social relationships.

Increasingly, research is establishing the validity and importance of inattention not only as separate from hyperactivity-impulsivity but also as the most persistent and impairing of the two symptom sets in the current understanding of ADHD. Yet identification of which cognitive impairments should be included within “inattention” continues.

### *A Broad Concept of Inattention*

The concept of inattention referred to in DSM-IV is a broad one; it involves much more than simply not paying attention while someone is speaking. DSM-IV criteria refer also to excessive problems with distractibility and chronic difficulties in organizing tasks and activities, attending to details, following instructions and completing tasks, and undertaking tasks that require

sustained mental effort, as well as to problems with losing things and excessive forgetfulness. This broad range of impairments encompassed within the DSM-IV criteria for inattention delineates impairment of not a single unitary function but a composite of diverse, but related, cognitive functions subsumed under the concept of “attention” (see Parasuraman 1998).

Consistent with the broad DSM-IV list of inattention symptoms, patients diagnosed with ADDs tend to report chronic impairments across a wide range of cognitive functions. From interviews with adolescents and adults diagnosed with ADDs, Brown (1995, 1996) derived 40 self-report items regarding aspects of inattention. These items clustered into five independent, but related, factors: 1) organizing and activating for work, 2) sustaining attention and concentration, 3) sustaining energy and effort for work, 4) managing affective interference, and 5) utilizing working memory and accessing recall. Persons diagnosed with ADDs who reported chronic difficulties in one of these factors tended to report similar levels of impairment in the other four.

Symptoms in Brown’s patient-derived factors extend beyond DSM-IV inattention criteria by incorporating several functions adumbrated, but not explicitly included, in DSM. For example, items related to activation for work tasks extend the DSM-IV item “often avoids tasks that require sustained mental effort” and are closer to the item “often seems unmotivated” used in the DSM-IV field studies (Lahey et al. 1994).

Some items on the Brown ADD scales are not directly linked to DSM-IV criteria but do reflect aspects of inattention identified by other studies of ADDs. For example, items related to managing affective interference are similar to reports by Wender (1995) that adults with ADDs but with no diagnosable mood disorders often have marked impairment in their ability to manage affective responses to stress or frustration to avoid excessive disruption in their work and

relationships.

### *Executive Function*

#### **Inattention as Impairment of Executive Function**

The broader view that inattention is an impairment of executive function is consistent with current neurological and neuropsychological understandings of attention. Unlike the psychiatric model, in which inattention has been linked primarily to disruptive behavior, neurological models tend to emphasize linkage of attention to memory and to a cluster of other cognitive functions often labeled “executive function” (Lyon and Krasnegor 1996).

Denckla (1996a, 1996b; see also Chapter 8 in this volume) has described how ADHD often overlaps with the neurological diagnosis of impairments in executive function. Executive function refers to a wide range of central control processes in the brain that connect, prioritize, and integrate operation of subordinate brain functions. Taylor (1995) highlighted the need to distinguish between modular and executive processes of attention wherein modular processing is integrated by executive functioning into strategically appropriate programs to complete necessary tasks. Denckla emphasizes that this central management system, often attributed to operations in the prefrontal cortex, is crucial to organizing and integrating cognitive processes over time and plays an increasingly important role as the young child matures and takes on more complex tasks and more independent activities.

Although the concept of executive function has not yet been rigorously defined, it provides a framework for recognizing that some brain functions manage other functions. These management, or executive, functions might be compared to the functions of the conductor of a symphony orchestra, the chief of a large city fire department, or the management team of a

corporation.

One metaphor for executive function might be the conductor of a symphony orchestra, who does not play a musical instrument in the orchestra but does play a critical role by enabling the orchestra to produce complex music. The conductor organizes, activates, focuses, integrates, and directs the musicians as they play. With his or her instructions and motions, the conductor weaves together the sounds of the various instruments, controlling the musicians' pace and signaling them to bring in and fade out their particular sounds to bring each musical score to life. The brain's executive functions, like the functions of the conductor, organize, activate, focus, integrate, and direct, allowing the brain to perform both routine and creative work.

Executive function might be compared to the chief of a large city fire department, who carries no hoses or ladders but does play an essential role in planning how firefighters should respond to a wide range of emergencies and in providing moment-to-moment directions to those who lay the hoses, climb the ladders, and enter burning structures to save life and property. The chief's role involves rapidly assessing unexpected dangers as they arise, deploying needed equipment and personnel, and carefully monitoring the situation, being alert to collapsing structures, sudden wind changes, and hazardous materials. Like the fire chief, the brain's executive functions respond to unexpected situations as they arise, by selecting from a variety of problem-solving tactics stored in long-term memory, implementing them, monitoring their execution, and changing them as needed to meet challenges.

Executive function might also be compared to the management team in a corporation. The corporation might have many divisions: procurement, human resources, manufacturing, marketing, and distribution. These separate divisions, however competent, could not function as

a corporation without effective management to organize, integrate, monitor, and modify their functions to meet constantly changing needs. Likewise, the managers could not produce and profit from the corporation's products without support from these divisions. Such is the relationship between executive function and other components of the working brain.

These metaphors cannot do justice to the complexity of the brain's executive functions, but they may help illustrate the important multiple roles involved in managing complex human activities. Executive function refers to the wide variety of functions within the brain that activate, organize, integrate, and manage other functions to allow the individual to function effectively. Many of the symptoms classified as ADD symptoms of inattention are actually symptoms of executive function impairments.

### **Developmental Demands on Executive Function**

Impairments in executive function may not become apparent until the individual is required to use this particular function. Preschool children, for example, are not expected to manage much for themselves; there is little demand on them for self-management beyond complying with parental directions. Usually, parents or other caretakers are responsible for starting, directing, and stopping most of the young child's activities. These adults often adapt their demands to the wishes and moods of the particular child, but they typically provide most of the child's "executive management."

Entry into nursery school or other group care situations gradually introduces the child to more demands for self-regulation and accommodation to the needs of caretakers and other children. Kindergarten and first grade increase demands for the child to regulate behavior (e.g., to sit still, refrain from talking, complete tasks within given time frames). The school environment also

increases demands for the child to regulate cognition (e.g., to maintain focus on specific tasks, organize and prioritize activities, remember particular concepts and skills). Children whose executive function impairments include severe difficulties with inhibiting impulsive behavior are likely to be identified very early in their school careers. Children who can manage basic behavioral self-control adequately, but who have significant impairments in the inattention spectrum of ADDs, may not be identified until they advance to the upper grades.

Demands on executive function usually escalate rapidly in junior high school, high school, and the first 2 years of college. This is when most individuals are faced with the widest range of demands to organize and direct themselves in the broadest range of cognitive and social activities, with the least opportunity to escape from activities for which they are not well suited. Moreover, early and middle adolescence is when parents and teachers are expected to decrease their management efforts and gradually require the teenager to take primary responsibility for self-management (i.e., for the exercise of executive function).

Another important fact about executive function is that it becomes progressively more necessary and complex as an individual gets older. Denckla (1996b) suggested that growing up is essentially the development of competence in executive function. Complex tasks such as dealing with multiple courses and different teachers in high school, driving a car, managing finances, and providing day-to-day parenting for children are a few of the many tasks that place strong demands on executive function. The increasing challenges to executive function as an individual matures may explain why inattention symptoms of some individuals with ADDs, particularly those who are bright and not hyperactive, are noticed not in early childhood but in middle to late adolescence or early adulthood, as demands on executive function increase.

Viewing ADDs as developmental impairments in executive function, which may not be recognizable until an individual confronts the demands of the upper grades, has important implications for determining the age at onset of ADD symptoms. DSM-IV diagnostic criteria for ADHD stipulate that at least some of the symptoms must have been present before age 7 years. Recently, the validity of this age requirement has been challenged by further analysis of the empirical field study data on which it presumably was based (Applegate et al. 1997). Barkley and Biederman (1997), following this same reasoning, argued that the age-at-onset criterion for ADHD should be either abandoned or “generously broadened” because it has no sound scientific basis and may impede recognition of ADD cases in which the onset of symptoms was not apparent as early as age 7.

Among the most important aspects of executive function as an individual matures is “working memory” (see below). Neuropsychologists (e.g., Pennington and Ozonoff 1996; Pennington et al. 1996) have highlighted working memory as a central aspect of executive function.

## **Attention**

### *Short-Term Memory as a Critical Element of Attention*

The linkage of attention to memory has long been recognized (see R. A. Cohen 1993; Cowan 1995). Recent developments on this linkage, however, focus on the role of working memory, a construct introduced into the psychological literature by Miller et al. (1960, p. 65) in their groundbreaking book on the organization of human behavior.

The concept of working memory has been developed by Baddeley (1986) and elaborated by Goldman-Rakic (1987, 1991, 1994, 1995) and others (see Richardson et al. 1996) to refer to a

subset of short-term memory functions that hold and manipulate information currently being processed. Working memory holds the focus and immediate context of current attention and refers to the brain's capacity to keep "online" and actively use bits of information crucial for current functioning, while carrying on other functions.

What separates current understandings of working memory from earlier notions of short-term memory is the recognition that working memory is not just a temporary storage system but an active processing system that helps the mind deal with immediate situations, whether novel or routine, in light of relevant information remembered from the immediate and/or distant past. Kosslyn and Koenig (1995, p. 388) described working memory as comprising "activated information in the long-term memories, the information in short-term memories, and the decision processes that manage which information is activated in long-term memories and retained in short-term memories."

Kosslyn and Koenig's point of view follows that of Anderson and Bower (1973), cited in Richardson (1996, p. 121), who emphasized that working memory is not structurally separate from long-term memory but rather is a currently active partition of long-term memory. Much remains to be learned about the nature and functioning of working memory, but two characteristics are generally agreed on: 1) working memory has a limited capacity and there are functional constraints on how much information can be activated simultaneously in it, and 2) individuals differ in the effective capacity of their working memory (Richardson 1996, p. 124). In many ways, working memory is similar to the random access memory (RAM) in a computer. Neuropsychologists and neuroscientists are still at an early stage in conceptualizing, operationalizing, and testing models of working memory. It is clear that many of the attention

impairments associated with ADD are closely tied to chronic ineffectiveness of working memory. Yet several other cognitive functions, which extend beyond working memory, apparently are impaired in persons with ADD. These include problems with insufficient arousal, energy, and effort for work tasks, which tie directly to the role of activation and emotion/affect in cognition.

### *Activation/Arousal Aspects of Attention*

Although neuropsychological concepts of working memory and executive function are as complex as the wide-spectrum concept of attention, existing models seem overly intellectualized and detached from the varying intensities of arousal and energy, which are crucial elements of attention. Sergeant (1996, 1999) noted that an adequate model of attention must incorporate elements of arousal, activation, and effort. Attention involves not only the flow of information within the cortex but also varying intensities of arousal and activation, engaged with and disengaged from a constant flow of internal and external stimuli. Arousal and activation may vary considerably from one individual to another and within persons from one situation to another (cf. Revelle 1993).

Two DSM-IV inattention symptoms address problems related to insufficient arousal, activation, and sustained effort for work tasks. The later-revised item “often seems unmotivated to do schoolwork or homework” from the DSM-IV field study picks up the problem of chronic procrastination regarding work tasks. “Often does not follow through . . . and fails to finish schoolwork, chores or duties in the workplace” alludes to chronic problems in sustaining effort for timely completion of work tasks. Several other inattention symptoms (e.g., “difficulty sustaining attention” and “easily distracted”) can also be understood as reflecting problems with

insufficient arousal/engagement or possibly excessive arousal.

### *Affective/Emotional Aspects of Attention*

Although the DSM-IV diagnostic criteria for ADHD include no affective symptoms, Wender's pioneering work (1987, 1995) and more recent formulations of ADD symptoms (e.g., Barkley 1997; Brown 1996; Conners 1997) recognize modulation of affect as a significant aspect of executive function that is often impaired as a result of ADDs. Moreover, as Rothbart et al. (1995) noted, processing of emotional information is an important aspect of attentional neural networks.

Energetic and arousal aspects of attention involve emotion as well as cognition. In *The Emotional Brain*, LeDoux (1996) argues that there is no unified emotional system in the brain.

He argues that, instead, there are a variety of ways cognitive and emotional arousal may be activated, sustained, or modulated by a rapid flow of unconscious linkages in working memory between current stimuli and activated memories unconsciously assumed to be relevant.

Regardless of whether LeDoux's specific formulations become widely accepted, his argument highlights the critical influence of unconscious emotional associations, often virtually inseparable from perceptions, as activators and modulators of attention. What remains to be clarified is the variety of mechanisms by which emotional influences (e.g., terror, longing, affection, jealousy, rage) both facilitate and impair the exercise of attention.

It is important to note that emotion affects attention not only as an internal influence that may disrupt attention and need to be managed but also as a vital element in generating and sustaining attention. This was highlighted by Taylor et al. (1997), who discussed emotions as "readiness for action" and "motivators and organizers of behavior." The researchers noted that "interest," probably the most frequently experienced positive emotion, "is an extremely important

motivation in the development of skills, competencies and intelligence” (p. 11). The motivating power of such “interest” may be most apparent when it is absent, as described in the chronic complaints of many adults with ADDs who report that although they can “hyperfocus” on activities in which they have special interest, they chronically find themselves unable to mobilize effort for tasks in which they do not feel any special immediate interest, even when they are fully aware that their failure to do that uninteresting task may cause significant problems later.

Negative impact of emotion/affect on attention is also apparent in many emotional disorders. Wells and Matthews (1994, p. 320) argued that emotional disorders of anxiety, depression, and obsessiveness are all associated with a general core dysfunctional attentional syndrome upon which features of the specific disorder and the individual personality and cognitive style are superimposed. The authors described this dysfunctional attentional syndrome as consisting of heightened self-monitoring and intensified processing of internal events, coupled with activation of self-beliefs and appraisal that strains the individual’s attentional capacity and reduces the overall efficiency of cognitive functioning (p. 266). By these and other mechanisms, emotion/affect may affect the broad range of attentional functions in ways that impair and/or enhance cognitive functioning.

### *Complexity of Attentional Systems in the Brain*

Given the wide spectrum of cognitive functions associated with attention, it is not surprising that many aspects of brain function seem to be involved. Attention is clearly not a unitary or modular function in the brain; it is not identified specifically with any singular brain structure. As Colby (1991) noted, “Attention is a distributed process . . . subserved by many brain structures” (p. S90).

In a review of findings from neuroimaging studies of the human brain, Posner and Raichle (1994, pp. 154–179) showed evidence of at least three anatomic networks that function separately and together to support various aspects of attention. These interacting networks are 1) an orienting network consisting of parietal, midbrain, and thalamic circuits; 2) an executive attention network including the left lateral frontal areas and the anterior cingulate; and 3) a vigilance network comprising the right frontal and right parietal lobes as well as the locus coeruleus.

Although many researchers rightly emphasize the role of the prefrontal cortex in problems of executive function, working memory, and ADHD, it seems likely that the wide spectrum of impairments associated with inattention will be found to involve many aspects of brain function. Denckla (1991b) emphasized this when she wrote “ADHD . . . exemplifies the need to broaden our differential localization beyond the prefrontal cortex to include neural substrates of activation, orientation, motivation and vigilance as these connect with and influence executive function” (p. x).

Among these neural substrates, which may be especially important in executive function, working memory, and ADDs, are those of the nigrostriatal structures. Crinella et al. (1997) reported findings from animal studies suggesting that nigrostriatal structures contribute essential, superordinate control of functions such as shifting mental set, planning action, and sequencing (i.e., executive functions).

Yet structural localization and dispersion of functions may not be the most important way to understand the impairments associated with inattention. As Pennington and colleagues (1996) pointed out, many developmental disorders may result

from a general change in some aspect of brain development such as neuronal number, structure, connectivity, neurochemistry, or metabolism . . . such a general change could have a differential impact across different domains of cognition, with more complex aspects of cognition such as executive functions being most vulnerable and other aspects being less vulnerable. (p. 331)

In this same context, Pennington et al. noted that the executive function impairments associated with ADHD and some other developmental disorders may all involve varying degrees of dopamine depletion in the prefrontal cortex and related areas (p. 330).

### *Neurochemical Contributions to Attentional Impairments*

For many years, researchers have suspected that the attentional impairments of ADDs are related to inherited neurochemical deficiencies in the brain. Pliszka et al. (1996) reviewed evidence that ADD symptoms may be related specifically to the catecholamines, a specific set of neurotransmitter chemicals manufactured in the brain. Yet most of the research on the role of catecholamines in ADDs has not differentiated between inattention and hyperactive-impulsive symptoms.

The possibility that attention impairments resulting from ADDs may be closely related to dopamine depletion in certain areas of the brain finds support in the numerous studies that have demonstrated dopaminergic medications (e.g., methylphenidate, dextroamphetamine) to be effective in alleviating a wide variety of inattention symptoms (see Levy 1991). Although noradrenergic medications (e.g., desipramine, nortriptyline) and  $\alpha_2$ -agonist medications (e.g., clonidine, guanfacine) have been demonstrated to be effective in alleviating hyperactivity-impulsivity symptoms of ADHD, there is some evidence that these nonstimulant medications are less effective in alleviating inattention symptoms (American Academy of Child and Adolescent

Psychiatry 1997; Levy and Hobbes 1988; Spencer et al. 1996).

These findings suggest that a specific neurotransmitter system, the dopaminergic system, may play a particularly important role in inattention symptoms of ADD. Servan-Schreiber et al. (1998) summarized research literature on the impact of dopamine on specific neural networks in human information processing and developed and tested a model demonstrating that dopamine has a direct positive effect on the increase of gain in the activation function of the neural networks underlying attentional processing. Additional evidence for the critical role of dopamine in management of cognitive functions comes from recent laboratory studies summarized by Wickelgren (1997), which indicate that in many species dopamine plays a critical role in mobilizing attention, facilitating learning, and motivating behavior that is critical for adaptation. The role of dopamine in facilitating these functions may be far more broad, subtle, and complex than has previously been thought. Inattention symptoms of ADD may be reflecting impairments resulting primarily from insufficient functioning of aspects of dopaminergic transmission in the human brain.

## **Diagnostic Issues in ADD Impairments**

### *Disentangling ADD Inattention From Hyperactivity*

As more is learned about the nature and complexity of inattention, it is becoming increasingly clear that the long-held linkage between inattention and hyperactivity in ADD needs to be reconsidered. Recently, both Barkley (1997; Barkley et al. 1996) and Quay (1997) proposed that the predominantly inattentive type of ADD should be considered a disorder separate from the combined type of ADHD. Both argue that combined-type ADHD results from impairment in the behavioral-inhibition system of the brain, whereas the predominantly inattentive type is a very

different cluster of problems with a different etiology to which their “impaired capacity to inhibit behavior” models do not apply. They argue that the “hyperactive-impulsive” symptoms associated with the current ADHD diagnosis constitute a cluster of developmental problems with a different cause and different course than those of the predominantly inattentive type, even though both types may respond to the same medications.

For reasons mentioned earlier in this chapter, separating the diagnosis of ADD inattention from the hyperactivity-impulsivity of ADHD may be sensible. DSM-IV has already separated the inattention symptoms of ADHD from the hyperactivity-impulsivity symptoms of ADHD. Currently, sufficient symptoms in either symptom set provide grounds for an ADHD diagnosis. The present confusion rests simply in the persistence of linking both symptom sets within the name of the disorder.

Perhaps a useful next step might be simply to change the labels of these diagnoses to further clarify their independence. What is currently called ADHD, predominantly inattentive type, might be called simply “attention-deficit disorder” without reference to hyperactivity. ADHD, predominantly hyperactive-impulsive type, might be called “hyperactivity-impulsivity disorder.” Persons with ADHD, combined type, might be given diagnoses for both ADD and hyperactive-impulsivity disorder.

Regardless of what may eventually be done in refining the official nomenclature for diagnosis, current practice in many clinical and educational settings is to use the official terms for ADHD only for formal diagnosis. The term *attention-deficit disorder* is already commonly used to refer to the attentional impairments of ADHD, regardless of whether they are accompanied by hyperactivity-impulsivity.

### *DSM-IV and the Diagnosis of ADD Inattention*

If diagnosis of the inattention impairments of ADDs can be separated from the hyperactivity-impulsivity symptoms of ADHD, it might be easier for clinicians to be alert to the wide range of cognitive impairments associated with ADDs, especially in adults. In the present diagnostic system, DSM-IV, there is no diagnostic category that picks up the wide range of cognitive impairments associated with the broad spectrum of inattention symptoms of ADHD. The DSM-IV section “Delirium, Dementia, and Amnesic and Other Cognitive Disorders” comprises disorders in which the “predominant disturbance” is a cognitive deficit that represents “a significant change from a previous level of functioning” (American Psychiatric Association 1994, p. 123) (e.g., dementia of the Alzheimer’s type or dementias due to vascular insufficiency, head trauma, or neurological disease), but all these disorders relate to impairment of cognitive functions that were once intact. Except for ADD, there is no diagnostic category in DSM-IV for cognitive impairments that are developmentally based, in which the individual of normal intelligence has had these same problems of impairment in organization, inattention, short-term memory, and so forth from early in life. In short, there is in DSM-IV no diagnostic category for developmentally based impairment of executive functions in persons of normal intelligence.

Although psychiatry has diagnostic categories to describe conditions of persons with executive functions that, once adequate, became impaired because of injury or disease, it does not yet have a diagnosis for the condition of those whose executive functioning has been significantly impaired from the outset of life, with or without accompanying hyperactivity-impulsivity. It appears that ADD is currently functioning as a category of diagnosis for many children, adolescents, and adults who have chronic, developmentally based impairments of executive functions and working memory.

In recognizing that the diagnosis of ADD is being used to label impaired executive functions, some might consider inventing a new name for this disorder of cognitive impairments; terms like “executive function disorder,” “cognitive management disorder,” and “developmental neurocognitive disorder” might be considered for future editions of the diagnostic manual. Yet it is not clear that such a change of terms would yield any greater clarity of understanding. There may be ways to adapt existing diagnostic terms—attention-deficit disorder and hyperactivity-impulsivity disorder—in ways that would preserve some continuity of terms and yet more adequately reflect the newly emerging understanding of these impairments.

### *ADD as Diagnosis for Developmentally Impaired Executive Function*

Following the lead of Douglas (1988), Denckla (1991b, 1993, 1994, 1996), Pennington and Ozonoff (1996), Pennington and Welsh (1995), Pennington et al. (1996), and others, theorists and researchers are increasingly beginning to conceptualize ADD as a diagnostic construct for developmentally impaired executive function and working memory. Tannock and Schachar (1996) observed, “There is growing consensus that the fundamental problems (in ADHD) are in self-regulation and that ADHD is better conceptualized as an impairment of higher-order cognitive processing known as <@145>executive function” (p. 129). Castellanos (1999) made a similar observation: “The unifying abstraction that currently best encompasses the faculties principally affected in ADHD has been termed executive function (EF), which is an evolving concept. . . . there is now impressive empirical support for its importance in ADHD” (p. 179).

The notion of “higher order” cognitive processing initially sounds formidably abstract, but it is quite simply illustrated in the situational variability of ADD symptoms. The problem that persons with ADD face is not that they are totally unable to sustain attention, to organize a task,

to recall what just happened, and so forth. They are able to exercise all these basic functions quite adequately under certain conditions—for example, in an emergency or when engaged in an activity in which they have a high level of immediate, spontaneous interest. The central problem of persons with ADD is that they are not able to activate and sustain these functions, which they sometimes perform quite well, in many situations when it is necessary or desirable to do so. For example, an individual may be able to “hyperfocus” and sustain intense attention for several hours while playing a sport or using a computer, yet he or she may be unable to sustain attention for more than a few minutes at a time when reading or when sitting in a class or a meeting. The functions—paying attention, organizing, recalling, and so forth—are intact; they are simply not responsive to higher-order processing. That is, the individual is not able readily to activate, deploy, and utilize these functions as needed. They are not readily turned off or on when needed; they are not responsive to “willpower.” Executive function is simply a name for those higher-order systems of the brain that activate, integrate, coordinate, and modulate a variety of other cognitive functions.

Tannock and Schachar (1996) also noted the neuropsychological evidence that persons with ADHD tend to show specific impairment in frontal lobe tasks associated with executive function: “The deficits in ADHD appear to be relatively specific to executive function rather than reflecting generalized cognitive impairment[,] because executive dysfunction is evident in ADHD children with above-average IQ and deficient performance is observed on frontal- lobe tasks, but not on measures of temporal lobe functioning or on ‘non- executive tasks’” (p. 131). A similar perspective is offered by Seidman (1997b):

Cognitive deficits, particularly impairments in attention and executive functions, are hypothesized to be a core part of ADHD [Douglas 1972] and are thought to play a major role in the difficult adaptation of

children with ADHD. These children exhibit subaverage or relatively weak performance on various tasks of vigilance and sustained attention, motoric inhibition, executive functions [such as organization and complex problem solving] and verbal learning and memory [Grodzinsky and Diamond 1992; Barkley et al. 1992; Seidman et al. 1995a, 1995b]. (p. 150)

Seidman makes a very similar report from his study of adults with ADHD: “Unmedicated adults with ADHD performed worse than controls on measures of auditory sustained attention, executive components of verbal learning and arithmetic despite similar levels of education and IQ. These impairments could not be accounted for by age, learning disabilities, psychiatric comorbidity or gender” (Seidman et al. 1998, p. 264).

### *Impairments of Executive Functions in Cognitive Versus Behavioral ADHD*

The formulations of ADHD as impairment in executive function just cited do not distinguish between combined and inattentive types. This is quite different from Barkley’s (1997) elaboration of his new theory of ADHD, in which he focuses explicitly on only the combined type and not the inattentive type. Drawing on extensive neuropsychological research, Barkley identifies behavioral inhibition as one of five aspects of executive functioning that he describes as underpinning the impairments of ADHD.

Using a rather elaborate theory extrapolated from Bronowski’s formulations about human language, Barkley (1997, p. 108) proposes that behavioral inhibition plays a primary and essential role in the development and proficient performance of four additional executive functions: nonverbal working memory, verbal working memory, self-regulation of affect/arousal, and reconstitution (i.e., capacity to manipulate stored information). He sees combined-type ADHD as a consequence of related impairments in these five aspects of executive function.

Barkley (1997) argues that behavioral inhibition is primary among these executive functions because it “probably assists with the suppression of the observable motor accompaniments associated with each form of executive function, thus facilitating the internalization of behavior” (p. 155). This assumption of the primacy of behavioral inhibition among executive functions is interesting but is thus far not convincingly supported by any empirical data. To the contrary, Barkley’s emphasis on behavioral inhibition as the central identifying feature of ADHD has been challenged by Douglas (1999): “Barkley’s attempt to establish the primacy of disinhibition differs from my own conceptualization [of ADHD] in which attentional and inhibitory deficits are viewed as different manifestations of an underlying *regulatory control problem*. . . . there is convincing evidence of *both* facilitating (activating) *and* inhibitory problems in ADHD” (p. 108).

I offer a modified view, an alternative to Barkley’s theory, as follows: 1) as Barkley and Quay have both argued, impairment of one aspect of executive function, behavioral inhibition, is the core problem in the hyperactive-impulsive type of ADHD but not in the predominantly inattentive type; and 2) other aspects of executive function (e.g., verbal working memory, self-regulation of affect) are impaired in the inattention symptoms of ADD, whether these symptoms appear in the combined type or in the inattentive type.

In this revised formulation, individuals with combined type ADHD would be seen having impairments in a wider range of executive functions—both those that modulate behavioral inhibition and those that modulate the wide variety of cognitive impairments currently listed as inattention symptoms of ADD. Individuals with the predominantly inattentive type of ADD, either primary or after remission of hyperactive-impulsive symptoms, would be seen as having impairment in those aspects of executive function related to the various aspects of inattention.

This understanding could be translated into existing diagnostic terms with a few simple changes of nomenclature. The term *attention-deficit disorder*, without any reference to hyperactivity disorder, could be used to describe the cluster of cognitive impairments described in DSM-IV as inattention symptoms of ADHD. The term *hyperactivity-impulsivity disorder* could be used to describe the cluster of impairments of behavioral control described in DSM-IV as hyperactivity-impulsivity symptoms of ADHD. Persons could be diagnosed as having both disorders so long as the criteria for both disorders are met.

### *ADD Impairments as Dimensional, Not Categorical*

Increasingly, clinicians and researchers are recognizing that ADD impairments occur along a dimension. For example, Levy et al. (1997) reported on a large-scale twin study that yielded genetic evidence that “ADHD is best- viewed as the extreme of a behavior that varies genetically throughout the entire population rather than as a disorder with discrete determinants” (p. 737). This continuum ranges from normal levels of impairments that occur sometimes in virtually everyone to more extreme levels of impairments that occur less frequently and have substantially greater negative impact on the person’s life functioning. Put simply, everybody has ADD-type impairments sometimes, but only those who chronically have significant impairment from ADD symptoms should receive an ADD diagnosis. Thus, the ADD diagnosis is analogous to the diagnosis for major depressive disorder; everyone has depressed mood occasionally, but only for those whose depression is persistent and significantly impairing is the diagnosis of major depression considered appropriate. In contrast, pregnancy is not dimensional, but categorical. There are no degrees of pregnancy; either one is pregnant or one is not. There is no defined state in between.

The two main points made by this emphasis on dimensionality of ADD are as follows: 1) The various impairments of cognitive function symptomatic of ADD inattention occur to some degree in virtually everyone. 2) The line between those diagnosed with ADD and those not meeting the diagnostic criteria for ADD is not sharply drawn; the differences between those meeting the criteria on the one side and those almost but not quite meeting the criteria on the other side are not great.

Despite findings that the dimensional view of ADD symptoms yields greater predictive utility for diagnostic purposes than does the categorical view (cf. Fergusson and Horwood 1995), Kagan (1994) and Jensen (1995) remind us that categorical understanding of a disorder like ADD may also be useful. Persons who have a cluster of significant impairments in a particular domain may have qualitatively different experiences than those who have isolated, transient, or less-impairing symptoms. The existing diagnostic criteria for ADHD have led to the identification of groups of people who chronically have a cluster of related impairments that tend to co-occur and to lead to considerable difficulty in a variety of settings.

Regardless of how the diagnostic labels for impairments of ADD may eventually be changed, or of how arbitrary the delineation may be, there will be a continuing need for those involved in diagnosis to determine a cutoff distinguishing the range of ADD impairments that are to be considered “normal” from those of sufficient severity that they can be considered to constitute a “disorder.”

### *Severity Cutoffs for ADD as a Dimensional Disorder*

Given that ADD symptoms are dimensional, it becomes clear that making a diagnosis for an ADD requires establishing a cutoff to mark a point beyond which the symptoms are considered

to constitute a disorder warranting treatment. DSM-IV appears to establish such a cutoff by its listing of nine symptoms of inattention and nine symptoms of hyperactivity-impulsivity and its stipulation that when six or more of these nine symptoms are present and exclusion criteria are met, the ADHD diagnosis can be appropriately made. Yet counting these symptoms is not like counting cancer cells in a biopsy tissue sample. Careful examination of ADHD symptom lists indicates that each item really encompasses a whole domain of functioning and requires a determination of what constitutes “often,” “maladaptive,” and “relative to developmental level.”

Consider the inattention item: “Is often forgetful in daily activities.” This might include forgetting what has been heard or what has been seen, or both, and what has been learned or what has been done, or both. “Often” might mean many times an hour, many times a day, or many times per week. “Developmental level” might mean of the same age, of the same general range of intelligence (IQ above 70), or of the same specific range of intelligence (compared with others with IQs in the superior range).

Many such judgments enter into a clinician’s determination of whether any given symptom should or should not be counted toward diagnosis. Similarly, clinical judgment is required for determination of whether any given individual is presenting “clear evidence of clinically significant impairment in social, academic, or occupational functioning” (American Psychiatric Association 1994, p. 84). Making such diagnostic judgments is not always easy; it requires empathic perception, ability to communicate effectively with the patient, a good appreciation of the wide breadth of “normality,” and a firm grasp of the multiple varieties in which psychopathology may be manifest. It should be noted that similar clinical judgments are required for diagnosis of virtually every psychiatric disorder, both in differentiating “disorder” from

“normality” and in differentiating one disorder from another, especially when they overlap or are concurrent.

## **Comorbidities With ADD**

### *“Dimensions of Impairment” Paradigm for Diagnosis*

It is not only for the ADD diagnosis that a dimensional approach to diagnosis of impairments is being recognized. Over recent years researchers and clinicians in both child and adult psychiatry have called attention to the limitations of categorical diagnosis, in which it is assumed that each psychiatric diagnosis is a discrete category with clear boundaries and substantial underlying specificity (Achenbach 1990–1991; Blacker and Tsuang 1992; Caron and Rutter 1991; Nottelman and Jensen 1995; Skodol and Oldham 1996).

As Skodol and Oldham (1996) noted:

[P]sychobiological research has led to the discovery of abnormalities in specific neurotransmitter functions in a wide variety of disparately classified disorders. Family studies have demonstrated family aggregation of disorders of apparently different types. Treatment studies have indicated that pharmacological agents, such as antidepressant drugs, can benefit patients with seemingly distinctive types of psychopathology. Thus, the notion that all 200+ DSM-IV categories represent discrete disorders with distinctive etiologies and pathogenetic mechanisms is patently naive, and the search is on for more fundamental psychopathological disturbances. (p. 2)

Many of the diverse discrete categorical diagnoses are increasingly being seen as related in clusters, spectrum, or dimensional groupings, in which the disorders in such groupings are seen as variations of dysfunctions in related functional systems. Examples include schizophrenic

spectrum disorders (Bellak 1994), compulsive-impulsive spectrum disorders (Oldham et al. 1996), autistic spectrum disorders (Towbin 1994), and depressive spectrum disorders (Angst and Merikangas 1997).

### *ADD as a Spectrum Disorder Often Comorbid With Other Disorders*

This dimensional, “spectrum” approach to diagnosis seems especially appropriate for ADDs, which, as discussed earlier, appear not to be unitary or categorical. ADDs include many variants of impairments in a wide range of cognitive executive functions. Yet these disparate impairments are related; they often appear concurrently, tend to run in families, and often respond to treatment with the same type of medications.

However, ADD impairments often do not appear alone; they can appear concurrent with a wide variety of other psychiatric disorders and do so with a frequency that greatly exceeds chance.

The medical term for this concurrence is *comorbidity*. Comorbidity can apply to the overlap of two or more disorders at the very same moment or, more commonly, to the co-occurrence of two or more disorders in the same lifetime. Generally, it is not the fact of comorbidity itself, but its frequency and causes that are important. It is possible for an individual to have diabetes and influenza (or many other combinations of ailments) at the same time; this would have no special interest unless persons with one of the disorders had an increased risk of having the other, or if the co-occurrence significantly changed the prognosis, course, treatment response, or outcome of either or both disorders.

Studies of the comorbidity of ADD have found extremely elevated rates of co-occurrence between ADD and many other psychiatric disorders (see Angold and Costello 1993; Biederman et al. 1991, 1992; Jensen et al. 1993, 1997). Assessments of comorbidity usually are made by

comparing the incidence of two given disorders in the general population and then ascertaining the incidence of one disorder among those persons identified as having the other.

Such comparisons of individuals with ADD and persons without ADD in the general population have yielded markedly higher incidence rates for a wide variety of psychiatric disorders in the ADD samples. For example, the generally reported rate of anxiety disorders in the general population of children is about 5%; among children with ADD the observed rate of anxiety disorders is approximately 25%. Similarly elevated incidences of major depressive disorder, oppositional defiant disorder, conduct disorder, learning disorders, bipolar disorder, Tourette syndrome, substance abuse, and other psychiatric diagnoses have been reported for children and/or adults with ADD (Biederman et al. 1991b, 1993).

Within the present system of diagnostic categories, ADD is very often comorbid with other disorders. Biederman et al. (1992) reported that among the children with ADDs in their sample, 51% met the criteria for at least one other psychiatric diagnosis; among adults with ADDs in this sample, the authors found 77% with at least one comorbid psychiatric diagnosis. These elevated rates of ADD comorbidity are found not only in clinic samples, where one would expect to find individuals with more severe and aggregated problems, but also in community samples, where there has been no selection for those who seek treatment.

### *Possible Reasons for the High Psychiatric Comorbidity With ADDs*

Several proposals have been advanced to explain the high rates of comorbidity of ADD with the wide range of psychiatric disorders. One possible argument is that ADD symptoms may be just one aspect of the comorbid psychiatric disorder. This argument is contradicted by Biederman et al.'s (1992) National Institute of Mental Health family genetic study of ADHD, in which they

presented evidence suggesting that the cognitive impairments observed in the sample of children with ADHD “are caused by the ADHD syndrome itself and do not appear to be accounted for by psychiatric comorbidity” (p. 352). Likewise, in a sample of adults with ADHD, Biederman et al. (1993) reported evidence of ADD cognitive impairments based on neuropsychological measures and unequivocal evidence of school failure, even in those adults whose ADD was not comorbid with any other psychiatric diagnosis in their lifetime.

Children and adults with ADDs who have a comorbid psychiatric disorder, such as depression, anxiety disorder, or conduct disorder, have all the requisite symptoms for the second disorder in addition to the requisite number of symptoms of an ADD. Moreover, ADD can sometimes appear without any comorbid disorder. ADD does not appear to be just another label for other psychiatric diagnoses with which it is comorbid.

Another explanation proffered for the high rates of comorbidity between ADD and other psychiatric disorders is that ADD may not be a single entity. Biederman et al. (1992) argued that ADD may be a name for “a group of conditions with different etiologies and risk factors, as well as different outcomes, rather than a homogenous clinical entity” (p. 339). Conners (1997) made a similar point: “Any effort to find a common mechanism, whether anatomical or purely psychological . . . [, for ADHD] seems doomed to failure as long as we treat the surface symptoms as unitary phenomena instead of the multi-component processes they really are” (p. 9).

From the perspective advanced in this chapter, one might paraphrase and modify Biederman et al.’s argument to say that ADD is a name for a spectrum of impairments of cognitive executive functions that often appear together and often respond to similar treatments, though they may

have differing etiologies, risk factors, and outcomes, and are often comorbid with a wide variety of psychiatric disorders, many of which may also be spectrum disorders. This view of ADD as a cluster of attentional/executive impairments that appear and may persist with and without psychiatric comorbidity is consistent with Seidman's findings from neuropsychological assessments of children and adults with ADD (Seidman et al. 1995a, 1995b, 1997a, 1997b, 1998): the impairments of attention/executive function in these individuals tend to be persistent and relatively independent of any comorbid psychiatric disorder that may be present.

### *Possible Subtypes of ADD Comorbidity*

The central question that emerges from recognition of the overlap of ADD with other disorders is, What difference does comorbidity make? In other words, how does the co-occurrence of ADD with another disorder alter the presentation, illness course, or response to treatment of the affected patient? The major purpose of most of the chapters in this volume is to address this important question.

Jensen et al. (1997) recently published a review of 15 years of ADD literature to ascertain the most prevalent patterns of ADD comorbidity and to determine the extent to which specific comorbid patterns may convey unique information about ADHD symptoms, treatment, and outcomes. From this review they derived data that support delineation of two new subclassifications of ADD: ADHD, aggressive subtype, and ADHD, anxious subtype. Data suggest that persons with ADD that is comorbid with conduct disorder tend to have lower IQ, increased learning/reading deficits, and evidence of neuropsychological impairments; also, demonstrably high levels of familiarity are evident in this population. Data on those whose ADD is comorbid with anxiety indicate a tendency for this group to demonstrate more inhibition on

laboratory measures of attentional processes, decreased impulsivity, and decreased severity of some other associated symptoms (e.g., aggression and conduct disorder symptoms). The authors suggested more research to refine understanding of these two proposed subtypes. At a more general level, Jensen and colleagues suggested, from their review of the data, that clinical course and outcomes of ADD are generally poorer in the presence of comorbid conditions than when there is no comorbidity, whether measured by parent-child interactions, poor school performance, automobile driving behaviors, or risks for later substance abuse and antisocial personality disorder.

### *Importance of Assessing Comorbidities With ADDs*

Clearly the impact of comorbid psychiatric disorders on the course, treatment, and outcomes of ADD is a very important area for further study. Yet, until recently, most researchers of ADD and many other psychiatric disorders have tended to design their studies to focus on individuals who manifested relatively “pure” forms of the disorder without complications of comorbidity. Jensen et al. (1997, p. 1077) strongly suggest that “comorbidity must be more fully considered in the design of future studies of ADHD.” They contend that “[r]ather than regard ADHD-associated conditions as <@145>noise,’ that is, as extraneous factors to be controlled for or eliminated,” investigators of ADHD should “vigorously study” the nature and impact of comorbidity on ADHD assessment, treatment, and outcome.

The study of the comorbidity of ADD with other psychiatric disorders is important. However, while the needed studies are being designed, carried out, and reported—a process that will take more than a few years—many patients with ADD will be seeking assessment and treatment. It is critically important that those who provide assessment and treatment for these children,

adolescents, and adults with ADD learn carefully to take into account the strong likelihood that comorbid disorders may be present with ADD. Likewise, clinicians assessing patients for other psychiatric disorders should seriously consider the possibility that ADD, perhaps masked by other symptoms, may be comorbid with another psychiatric disorder such as a mood disorder, anxiety disorder, conduct disorder, learning disorder, or substance abuse.

In actual practice, clinicians deal with the complications of individual persons, not with simplistic categories of disorder. Often a person, in addition to fully meeting the established diagnostic criteria for one or more disorders, will have some symptoms of other disorders, but not all the symptoms required for diagnosis of those disorders. Good clinicians have long recognized that these more isolated symptoms and “subclinical” forms of disorder need to be taken into account for accurate assessment and effective treatment. Ratey and Johnson (1997) highlighted the importance of these subclinical characteristics, which they have labeled “shadow syndromes.”

In any case, adequate assessment and treatment of ADD requires that the clinician take into account not only the wide spectrum of impairments associated with ADD but also the accompanying symptoms of other disorders. Some of the accompanying symptoms may fully meet the diagnostic criteria for specific disorders, whereas others may reflect remission or persistence of the disorder in subclinical form. Sometimes only one comorbid disorder may be present, either actively or in remission. In some cases, the diagnostic criteria for several comorbid disorders may be fully met, with the disorders active at the same time or intermittently.

It should also be noted that generally neither ADD nor comorbid disorders are static in their manifestations. Longitudinal research and clinical experience both indicate that symptom

profiles of individuals with ADD often change over time—for example, hyperactive-impulsive symptoms may be lost as a child gets older, and organizational problems may become increasingly evident as an adolescent takes on more adult work and financial responsibilities.

Likewise, symptoms of other psychiatric disorders comorbid with an ADD may remit or exacerbate over time. For example, obsessive-compulsive symptoms may become more or less salient, an adolescent may gradually cease to abuse alcohol or marijuana, and depressive symptoms may lift spontaneously or in response to treatment. The severity of ADD symptoms and their responses to treatment may vary considerably as a function of increased or reduced severity of one of the comorbid disorders or interaction of changes in several comorbid disorders that may be present at a given time.

### **Areas for Further Research on ADD Comorbidity**

Recognition of ADD as a spectrum of executive impairments comorbid with many other psychiatric disorders raises many questions about the nature, etiology, assessment, course, treatment, and outcome of ADD. These questions apply to ADD both in its relatively uncomplicated forms and when comorbid with various other disorders, singly and in combination. Some areas in which further research might be especially useful are described in the following subsections.

#### *Assessment of ADD Cognitive Impairments Across the Life Span*

Although there exists an enormous and continually growing research literature on ADD, most of the reported research thus far has focused primarily on young Caucasian boys with hyperactive-impulsive symptoms. There have been relatively few studies of adolescents and adults with

ADD, very few of adolescents and adults with ADD symptoms that did not include hyperactivity, and extremely few of persons with ADD who are female or from ethnic minorities. Moreover, as Jensen et al. (1997) noted, extant research on ADDs has insufficiently studied persons with ADDs that are complicated with comorbidities. The research that has been done on the comorbidities of ADD has focused mainly on varieties of behavioral disorders, especially conduct disorder. Relatively little attention has been paid to other types of comorbidity with ADD.

Since ADD appears to be a highly prevalent developmental disorder that, for at least half of those diagnosed, persists into adulthood, it is extremely important to develop a fuller empirical understanding of how ADD symptoms are manifested in childhood and of how these symptoms persist and/or change over time in the absence and presence of comorbidity.

Some ADHD symptoms may change, for better or worse, in response to developmental learning or advancing brain development. New symptoms may arise as individuals are faced with increasing demands for self-control and independent functioning. Biederman et al. (1998) presented data on the diagnostic continuity of childhood ADHD into adolescence, but far more empirical information is needed on the persistence and changes in the wider range of ADHD symptoms over the life span.

Requirements for increased executive function continue to escalate as development proceeds, from the relatively basic requirements of group activities in day care or kindergarten, to the constantly increasing academic and social challenges of each grade in elementary and high school, to the variegated challenges of preparing to work, eventually moving out of the family nest, getting and functioning in a job, paying one's own bills, managing a household, carrying on

multiple social relationships, and, possibly, functioning as a parent. Awareness of these increasing challenges over time to an individual's capacities for executive functions, when coupled with recognition of ADHD as developmentally impaired executive function, may require reassessment of the diagnostic criteria for ADHD pertaining to age at onset and age at impairment. The DSM-IV diagnostic criteria for ADHD specify that at least some of the symptoms of ADHD must have been present prior to age 7 years for the ADHD diagnosis to be used appropriately. Recently, some of those who were involved in establishing those criteria have questioned the validity of the age-at-onset criterion (Applegate et al. 1997). Barkley and Biederman (1997) extended this questioning to argue that the age-at-onset criterion for ADHD should be altogether abandoned or generously broadened.

Persons with various types of ADD and comorbid disorders may experience widely differing levels of impairment and success as they attempt to cope with the ever-changing challenges presented by the demands of changing life circumstances. Some widely circulated popular books offer very useful descriptions of the phenomenology of ADD in adult life (e.g., Hallowell and Ratey 1994; Kelly and Ramundo 1995; Solden 1995). Systematic cross-sectional and longitudinal research is needed to develop adequate empirically based understanding of the developmental course of ADD and how this course may be modified by various comorbid conditions.

One particular need for further research is to develop an empirical basis for diagnostic criteria for ADD in adults. The DSM-IV criteria for the diagnosis of ADD are intended for use with all age groups, but the criteria were developed in field studies that included no adults, only children aged 4 to 17 years. Murphy and Barkley (1996) reported that when the diagnostic cutoffs of

DSM-IV are applied to assessment of ADD in adults, only the most severe cases are identified. Their studies of adults not seeking treatment indicate that much lower cutoff points must be utilized to identify adults whose symptoms place them within the 7% of the population most impaired by ADD. To provide adequate criteria for diagnosis of ADD in adults, systematic research is needed to establish the nature, course, and prevalence of symptoms of ADD in late adolescence and throughout adulthood. More systematic research can establish impairment on a more solid foundation than that provided by simply a given percentage of the population or criteria for persistence of impairment derived from studies of samples of children.

Neuropsychological research on ADD impairments of children, adolescents, and adults is particularly important for validating the diagnosis and for developing more adequate diagnostic measures useful for various ages. As Seidman et al. (1997b) observed, cognitive performance measures “are key validating criteria for ADD because they do not share method variance with other measures . . . , directly assess performance . . . and can be given longitudinally to assess (symptom) stability over time” (p. 158). Further research will help to determine the clinical and predictive utility of neuropsychological measures in the assessment and treatment of ADD; it should also lead to development of more effective measures to augment assessment by clinical interview.

Yet there are limits to what can be expected of “objective” measures of executive function impairments of ADD. Rabbit (1997) observed that the usual research strategy of isolating and quantifying one particular variable is not feasible in studying executive function because “an essential property of all ‘executive’ behavior is that, by nature, it involves the simultaneous management of a variety of different functional processes” (p. 14). Burgess (1997) expressed a

similar concern in his review of theory and methodology of executive function research; he cited numerous studies indicating that most current neuropsychological measures of executive function are inadequate because they try to separate interacting aspects of complex integrated functions. To illustrate, he cites Goethe's comment that "dissecting a fly and studying its parts will not tell you how it flies" (p. 99).

### *Interaction of Genetic and Environmental Factors in ADDs*

Numerous studies have demonstrated that ADD impairments are highly familial, linked to genetic factors that apparently have an impact on the functioning of specific neurotransmitter systems (see Chapter 2, this volume, for a review). Yet environmental factors are not without influence on any individual's ADD symptoms. Biederman et al. (1995) replicated the earlier findings of Rutter to demonstrate that psychosocial adversity significantly influences the expression of ADHD symptoms. In this study they found that while no one particular psychosocial stressor increased poor outcome of ADD, the additive effect of multiple stressors (e.g., low socioeconomic class, large family size, parental psychopathology, foster placement) had a significant impact on the level of impairment.

As Mazure and Druss (1995) noted, the relationship between environmental stressors and psychiatric disorders, though not simple, is significant. Several different theories have been advanced to describe the role of stressors and environmental protective factors in contemporary models of psychiatric illness. What these theories have in common is an appreciation of the importance of environmental stressors and supports interacting with genetic vulnerability to psychiatric illness to shape the individual's impairment and development of strengths. Thus far, there has been very little research on the role that environmental stressors and/or supports might

play in shaping the course of impairments and development of protection and adaptation in persons with ADD (see Samudra and Cantwell 1999). Yet several workers in developmental psychopathology have reported on studies that may help begin to address these issues.

Rogeness and McClure (1996) reported on the study of environment- neurotransmitter interactions as these shape neural circuits, neurophysiology, and neurochemistry of the brain across development. Likewise, Shore (1996) discussed the role of experience in maturation of the regulatory system in the prefrontal cortex. The perspectives of these researchers suggest a mechanism and a developmental perspective that might be useful in studying these issues with regard to ADHD.

### *ADD Symptoms Associated With Severe Social/Mental Dysfunction*

Many individuals with ADD chronically have difficulties in social relationships with peers, family members, teachers, work supervisors, intimate partners, and others. These difficulties stem from the tendency of these individuals to be chronically inattentive to social communications, especially to subtle verbal and nonverbal

cues that often play a critical role in regulating social interaction. Persons with ADD symptoms that include hyperactive- impulsive behavior often have even more social problems because of their tendencies to be impatient, impulsive, and intrusive. Yet, despite such difficulties, most persons with ADD are characterized by relatively normal social development.

Yet ADD inattention symptoms are also found in some children, adolescents, and adults who, despite average or above-average intelligence, are strikingly unusual in their social and emotional development. These individuals with “atypical” characteristics appear qualitatively

different from age-mates in the way they relate to other people and/or in the way they express their emotions. In social situations some of these individuals come across as peculiarly detached, showing little evidence of emotional interest or connection to those around them; others in this group relate in uncomfortably intense ways, thrusting themselves on strangers as though they were intimates. Often such children and adolescents impress both peers and adults as “weird” because they appear surprisingly out of touch with even the most basic social expectations.

Emotional expression in these children, adolescents, and adults with atypical characteristics may be very different from that of most other persons, including most others with ADD. They may react to apparently minor frustrations with sudden and sustained “catastrophic” emotions (e.g., inconsolable sobbing or violent, threatening rage). In others of this group, emotional expression is startlingly absent, even in situations where one would expect intense pleasure, sadness, or anger; the individual may simply withdraw or appear totally unaffected.

Some of the children with these atypical characteristics also have stereotyped behavioral mannerisms that make them stand out from other children. They may flap their arms or engage in unusual repetitive hand gestures when excited. These peculiar mannerisms are not tics or flamboyant displays of emotion; they appear more as involuntary movements that occur as unwitting “motoric overflow” in times of excitement (e.g., when the child is frightened or awaiting some pleasure).

Some of these individuals with atypical characteristics are also peculiar in their thought processes. Not only do they have the usual ADD problems of losing focus in conversation, they also tend to “get stuck” on certain topics of conversation and have unusual difficulty in moving on. Sometimes they cannot let go of a particular word, image, or theme; they tend to perseverate

in retelling a joke or multiplying fantasies about a situation—for example, describing various ways they could get back at someone who has offended them.

Although these unusual mannerisms of speech, movement, or thought may be quite striking, they often appear as just one more peculiarity of an individual who stands out far more obviously as having impairment in very basic aspects of social interactions and/or emotional expression.

These individuals tend to be perceived by peers and elders as awkward, strange, weird, odd, and eccentric. Their social and emotional impairments are not just immature or delayed; they seem outside the usual range of development and quite atypical.

Although some individuals with attention-deficit disorder and these atypical symptoms do not fully meet the diagnostic criteria for another disorder (see Barkley 1990; Guevremont 1993; Rubin and Stewart 1996), some others have persistent emotional detachment that may meet the diagnostic criteria for an autistic spectrum disorder (e.g., Asperger's disorder, pervasive developmental disorder not otherwise specified, or schizoid personality disorder) (Attwood 1998; Klin 1994; Siegel 1996; Towbin 1994; Wolff 1995). Others whose atypicality may manifest more volatile, intense emotional displays may qualify for the diagnosis of borderline personality disorder (Cohen et al. 1983; King and Noshpitz 1991; Lewis 1994; Petti and Vela 1990). Still others who also manifest disordered thinking may meet the criteria for childhood-onset schizophrenia or schizotypal personality disorder (King 1994; Towbin et al. 1993).

Current DSM-IV diagnostic criteria stipulate that the ADHD diagnosis should not be made if symptoms occur “exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia . . . and are not better accounted for by another mental disorder,” including a personality disorder (American Psychiatric Association 1994, p. 85). From the point of view

described in this chapter, there may be reason to reexamine this stipulation, which is based on a hierarchical model of diagnosis in which the ADD symptoms would be essentially subsumed within the “more severe” diagnosis. From the viewpoint of our emerging conceptualization of an ADD as an impairment in executive function/working memory, ADD inattention symptoms would, it seems, warrant diagnostic attention and possible targeted treatment even if they are present in the context of another, “more severe” disorder.

This viewpoint would be consistent with that of Bellak (1979), who recognized that a significant number of his patients with “schizophrenic spectrum” disorders had “minimal brain dysfunction,” the term used at that time for what is now known as ADHD. Subsequently, he more specifically noted an overlap between the schizophrenic syndrome and ADDs (Bellak 1994). More recent research (Green 1996) has noted that the cognitive deficits of schizophrenia with the greatest functional import are not the “positive symptoms” of delusions or hallucinations, but the “negative symptoms” of impairment in information processing involving verbal memory and impaired vigilance. These cognitive impairments in schizophrenic patients, as described by Green (1996), closely resemble the executive function impairments discussed earlier as being central to ADD. Taylor (1995) noted the potential value of further research on the development of attentional impairments in schizophrenia.

Impairments of working memory in persons with schizophrenia have been emphasized by Goldman-Rakic (1991), who argued that “a fundamental problem in schizophrenia is the loss of working memory processes that inexorably lead to a deficit in the regulation of behavior by internalized schemata, symbolic representations, and ideas” (p. 1). She claims that this “singular cognitive operation may account for some cardinal features of the disease” (p. 1). Similarly,

Docherty et al. (1996) presented data to support their hypothesis that communication disturbances in schizophrenia reflect specific deficits in working memory and attention. It is not clear how similar or different the impairments of attentional/working memory in persons with schizophrenia may be from those in persons with ADD but no schizophrenia. The findings from one study (Øie and Rund 1999) suggest that ADHD patients have more impairment on some specific measures of attention, verbal memory, and learning, while patients with schizophrenia show more general impairment of brain function.

These observations about possible overlap of ADD and severe autistic or schizophrenic spectrum disorders are of more than theoretical interest; they have important implications for treatment. A recent exchange of letters in a psychiatric journal illustrates the point. One correspondent chided the writer of an earlier article for overlooking the DSM rule that children with autistic disorder should not be diagnosed as having ADHD. The author responded, “Although they may be hyperactive, persons with autistic disorder also may present with executive dysfunction, inattention, impulsivity and distractibility. These individuals may benefit from treatment with methylphenidate or clonidine and have been so treated for many years in the autistic disorder program at Johns Hopkins Hospital” (Harris 1996). Bellak (1994) made a very similar argument for use of stimulant medication in the treatment of a subgroup of patients within the heterogeneity of patients with schizophrenia whom he identified as having “ADD psychosis.” He wrote, “It is my experience that people I now consider to be suffering from ADD psychosis respond favorably to dopaminergic medication and poorly to neuroleptics[,] whereas with patients who have been diagnosed with schizophrenic syndrome, the reverse is most often the case” (p. 29). More research is needed to increase understanding of how the executive function/working memory impairments identified here as central to ADDs may be involved in

various other psychiatric disorders. Such research may have important implications both for developing more effective treatments and for understanding underlying common features in disorders previously thought to be unrelated.

### *ADDs and Specific Learning Disorders*

Another area in which further research might be especially fruitful concerns the overlap between the executive function/working memory impairments central to ADDs and the domain of specific learning disorders. Although there has been persisting debate about the nature of the relationship between ADD and specific learning disorders, there is considerable evidence of markedly elevated rates of specific learning disorders, such as reading disorder, math disorder, and disorder of written expression, among individuals diagnosed with ADD (Cantwell and Baker 1991). Some have attempted to draw a clear line between these two categories by characterizing ADD as a disorder that may be helped by medication, while viewing specific learning disorders as “hard wired” into the organism and responsive only to alternative instructional techniques. This clear demarcation may be challenged by an alternative view in which impairments of working memory are seen as playing a significant role in both ADDs and learning disorders.

Tannock and Brown (Chapter 7, this volume) note the critical role of working memory in reading, math, and written expression. Reading involves holding in mind and integrating initial portions of a word, phrase, sentence, paragraph, chapter, and so forth long enough to connect these with subsequent portions so that connections can be made and various levels of meanings can be comprehended. Connections must be made between letter shapes and phonemes; diverse associations from elements of long-term memory must be quickly sorted out to select what is appropriate to context and to discard what is not. Smooth execution of these multiple linkages

clearly involves not only the learning of phonemes and vocabulary but also ongoing exercise of short-term working memory.

Likewise, most mathematical operations, from the borrowing and carrying of the simplest arithmetic to the intricacies of the most complex calculations for theoretical problem solving, are highly dependent on working memory. Multiple steps must be prioritized and sequenced, and information must be carried from one operation into another. To do arithmetic and mathematics, one needs not only knowledge of specific procedures but also effective working memory. The problem solver's ability to transiently hold "on-line" these various numerical facts and relationships while analyzing problems and invoking appropriate learned procedures is another example of the exercise of working memory.

Similarly, working memory plays an essential role in written expression as one selects and weaves together words and verbal images to convey multiple levels of meaning. In writing, one must hold in mind an overall intention for what is to be communicated in the whole of the phrase, sentence, paragraph, essay, report, chapter, book, and so forth, while simultaneously generating the micro units of words and phrases that will eventually constitute the written work being produced. Complex and rapidly shifting interplay of micro and macro intentions is the essence of creating and self-editing that allows one gradually to shift from the glimmer of an idea, through crude approximations of rough draft, to the greater specificity and polish of a final product in which one has captured in written language what one wants to say. In addition to many more specific skills, the whole process of written expression involves ongoing and often intensive use of working memory.

Research on the differing roles of working memory in the learning and exercise of reading,

mathematics, and written expression could be helpful in improving instructional techniques in these skill domains; it could also help in developing more adequate assessment and more effective remediation for those numerous persons whose functioning in one or another of these skills is so impaired that they are identified as having a learning disorder. Existing research suggests that stimulant medication can be effective in improving a wide range of information-processing and working-memory functions for some people with ADDs (e.g., Balthazor et al. 1991). There is need for more intensive research to determine whether treatment with stimulant medications may also be helpful for certain aspects of specific learning disorders, with or without a comorbid ADD.

One would not expect stimulants to cure learning disorders. No medication can establish unlearned skills (e.g., phoneme recognition); this is the work of education. Yet stimulant medications may play a significant role, directly and indirectly, in helping to alleviate chronic executive function/working memory impairments so that persons with both ADD and learning disorder may be enabled to participate in and apply more effectively the remedial education in basic skills that they desperately need (Beitchman and Young 1997).

In addition to the academically based learning disorders of reading, mathematics, and written expression, there is another type of learning disorder that seems often to overlap with the executive function impairments of ADHD: nonverbal learning disorders (NVLDS). Numerous authors (Denckla 1991a, Chapter 8, this volume; Myklebust 1975; Rourke 1985, 1989a, 1989b; Rourke and Fuerst 1991; Semrud-Clikeman and Hynd 1990; Tranel 1987; Voeller 1991; Weintraub and Mesulam 1983) have described this syndrome, which has not yet been adequately studied or conceptualized and is not yet incorporated in the DSM system.

NVLD syndrome has been described as encompassing a wide variety of impairments in cognitive functions required for tactile and visual perception, exploratory behavior in novel situations, pragmatics of social functioning, and ability to shift and integrate visual or conceptual perspectives (Denckla 1991a, Chapter 8, this volume; Rourke 1985, 1989a, 1989b, 1995; Rourke and Fuerst 1991). These impairments may be apparent in a cognitive style characterized by excessively narrow focus and chronic difficulties in grasping the more global, contextual aspects of ideas and situations. Severe impairments of NVLDs are often associated with very severe psychosocial and/or psychiatric problems (Rourke 1989, 1991, 1995; Voeller 1991). Presumably, most NVLD impairments are associated with malfunctions in the right hemisphere of the brain, the functions of which are just beginning to be more clearly understood (see Ornstein 1997).

As Eslinger (1996) noted, current models of executive functions do not yet adequately incorporate the ways in which executive function shapes and impacts social development and functioning. This is an area of learning disorders and executive function in which further theoretical elaboration and empirical research are urgently needed to guide assessment and clinical interventions. Conceptual clarification and empirical research are also needed to illuminate the overlaps between NVLDs and ADHD. Denckla (see Chapter 8, this volume) has suggested that distinctions between these two domains of impairment may be simply differences of perspective and vocabulary among academic and clinical disciplines.

### *ADDs and Developmental Changes in Estrogens*

From the perspective taken in this chapter, ADD is a developmental disorder in that affected persons seem generally to be born with the core impairments, manifestations of which gradually emerge over the course of development as the individual is called on to learn and apply an

increasingly broad range of functions for behavioral and cognitive self-management. Results from recent studies of adult women with and without ADDs raise another possible understanding of ADD symptoms as “developmental.”

Many middle-aged women report that during the course of their menopause, regardless of whether it is naturally occurring or surgically induced, they experience for the first time a constellation of persisting symptoms that closely resembles the inattention symptoms of ADD. They note significant impairment in short-term memory, in ability to screen distractions and to sustain attention, in organization of thoughts and tasks, and so forth (see Warga 1999). Women diagnosed premenopausally with ADD often report significant exacerbation of their long-standing ADD symptoms during the protracted perimenopausal period and thereafter. These reports raise the possibility that ADD symptoms may be exacerbated or developmentally acquired by some women during menopause.

A possible mechanism for such a developmental phenomenon might be the role of estrogen as a facilitator of the release of dopamine. Basic neuroscience research (McEwen 1991; McEwen and Parsons 1982; Mermelstein et al. 1996; Thompson and Moss 1994) suggests that estrogen potentiates and modulates the release of dopamine, especially in brain areas associated with executive function, both genomically and nongenomically, in a variety of complicated ways. If this is so, significant inconsistency or persisting reduction of estrogen levels in a woman’s body such as occurs in menopause may contribute substantially to ADD symptom exacerbation in women with ADD and may even produce onset of ADD symptoms in some women who have never previously manifested ADD symptoms in any significant way.

Sherwin (1998; Phillips and Sherwin 1992a, 1992b) has reported research in which she and

others demonstrated in controlled studies that administration of estrogen to postmenopausal women enhances verbal memory and maintains the ability to learn new material. Moreover, an MRI study by Shaywitz et al. (1999) demonstrated that administration of estrogen to postmenopausal women increased activation in specific brain regions during verbal and nonverbal working memory tasks.

Much remains to be done before any direct parallels can be inferred between cognitive impairments characteristic of ADD and those often found during menopause. Yet preliminary data are sufficiently suggestive to warrant further investigation of the possible roles of gonadal hormones in the onset, exacerbation, and alleviation of ADD symptoms. If these preliminary findings are confirmed, menopausally induced ADHD symptoms might come to be seen as a form of “secondary ADHD” in the same way that Gerring et al. (1998) proposed to label as “secondary ADHD” the onset and development of ADHD symptoms after closed head injuries.

### *Longer-Term Treatments for ADDs*

Most of the research on treatment of ADDs, thus far, has focused on interventions provided over a relatively short term. The preponderance of the more than 200 studies of medication treatments for ADDs have been of less than 3 months' duration. In 1993 Schachar and Tannock found only 18 studies of psychostimulant studies for ADD with a duration of more than 3 months. Studies of psychosocial interventions have generally been of similarly short duration. Even the recently completed multisite Multimodal Treatment Study of Attention Deficit

Hyperactivity Disorder, the largest and longest- duration controlled study of ADD treatments thus far, provided its medication and psychosocial interventions for only 14 months.

Given this lack of longer-term studies of ADD treatments, it is not surprising that many discussions of ADD treatment recognize the short-term benefits of such treatments but cite the lack of evidence that the treatments provide any long-term benefits. Some have pointed to the few longer-term follow-up studies to argue that individuals treated in childhood do not seem to fare any better than matched control subjects, but this argument carries little weight once it is recognized that the studies on which this argument was based did not continue treatment into adolescence. Trying to determine the long-term effectiveness of a treatment for ADD under such circumstances would be comparable to testing the benefits of wearing eyeglasses by assessing the vision of visually impaired children 10 years after they had been forced to stop wearing their eyeglasses.

The short-term focus of most studies of the treatment of ADDs contrasts sharply with the more long-term, chronic nature of the disorder, of which there has been increasing evidence. It now seems clear that ADD is not a disease that can be cured as one might cure a streptococcal infection with a course of antibiotic treatment. Nor is it usually just a transient affliction of early childhood commonly outgrown. If, as I have argued earlier, ADDs represent a developmental impairment of executive functions of the brain, which for many of those affected appears to have significant negative impact over the life span (Hechtman 1996; see also Chapter 14, this volume), many persons with ADD are likely to need ongoing treatment over a long term, at least through adolescence and possibly well into adulthood.

There will certainly be exceptions—for example, persons with ADDs in childhood who do not need ongoing treatment. Some children do “outgrow” their ADD symptoms as they grow older. Much brain development continues into adolescence and beyond. Fischer and Rose (1994)

reviewed research and described the behavioral implications of ongoing brain development (e.g., synaptic growth and pruning) from birth through 30 years of age. For some children, the ADD symptoms appear as a transient “developmental delay,” which is eventually fully compensated for by further neurodevelopmental maturation.

For some individuals, some of the problematic ADD symptoms (e.g., hyperactivity) that are present in early childhood may decrease in severity as the individuals mature toward adolescence. Yet, as Hart et al. (1995) and others have reported, other ADD symptoms, especially those in the inattention cluster, tend to persist. Studies are needed to describe empirically which symptoms of impairment tend to persist into adolescence and adulthood, for how long, with what changes, and with what impact on education, work, and social relationships.

Some persons with ADDs may eventually find ways to avoid domains of activity in which their ADD impairments continue to present significant problems. Some specific executive function impairments are more important for certain tasks and settings than others. Boetsch et al. (1996) reported studies showing how some individuals with dyslexia and ADHD, after their schooling ends, self-select, are forced, or wander into areas of work and social relationships that are less demanding and more rewarding for their particular combination of strengths and limitations. Others, perhaps less fortunate or more bold, may attempt to persevere in areas of work, further education, or relationships in which their ADD impairments continue to be more problematic.

Although there are exceptions, longitudinal studies of children with ADD indicate that 50% to 70% or more of persons diagnosed with ADD in childhood continue to meet the diagnostic criteria for ADD at least into later adolescence. Hechtman (1996; see also Chapter 14, this volume) has reviewed research on the variety of outcomes in individuals diagnosed with ADHD

in childhood as they emerge into adulthood. These outcomes vary widely, often as a result of not one single factor but according to the additive and interactive impact of multiple protective and deleterious factors within the person and their environments. For the many whose ADD persists in problematic ways into adolescence and adulthood, some sort of continuing treatment is likely to be useful.

Thus far, there is little research to guide longer-term treatment of ADD. The research base for treatment of ADDs is not very different from that for treatment of many other chronic psychiatric disorders. In recent articles, the authors have emphasized this need and suggested guidelines for developing research on interventions designed to sustain treatment effects for the chronic course of: unipolar depression (Nezu et al. 1998), addictive behaviors (Dimeff and Marlatt 1998), eating disorders (Perri 1998), and conduct disorder (Eyberg et al. 1998). Similar research and clinical initiatives are needed for persons with ADDs who may need continuing or intermittent treatment over their life span.

## **Conclusion**

This chapter has highlighted the emerging new understandings of attention-deficit disorders as complex, multifaceted clusters of dimensional impairments in the cognitive and behavioral management functions of the brain. Described in this chapter is the emerging shift in paradigm from the old understanding of ADD as a simple disruptive behavior disorder limited to childhood toward a new understanding of ADDs as complex developmental impairments of executive functions in the brain that may cause persisting reverberations of impairment through the life cycle. Yet these understandings are emerging, not fully developed and established. The research base for such models is still fragmentary and suggestive, not complete or definitive. Much

work—theoretical and empirical—remains to be done.

The newly emerging perspectives described in this chapter are not consistently incorporated in other chapters of this volume. Some chapter authors have alluded to them, but most of the chapters use the currently orthodox definitions of ADDs provided in DSM-IV. The DSM-IV criteria are the common base of shared understanding on which more adequate conceptualizations and hypotheses can be developed. Despite the amazing progress of the past 10 years, much more research is needed to test, clarify, and enlarge our understanding of how best to conceptualize, assess, and treat these clusters of impairments currently known as ADDs.

In the title of this book and throughout this introductory chapter the plural form ADDs is primarily used to refer to attention-deficit disorders. The purpose of this usage is to highlight the multiplicity of ways in which ADDs present themselves in various individuals at various times. This usage is adopted in an effort to counter those who tend to speak of ADD in simplistic terms as a unitary disorder; it invites recognition of the rich and diverse complexity of these cognitive impairments as they appear sometimes in relatively uncomplicated form and often in comorbid combination with one or more other psychiatric disorders.

Studying the diversity of attentional disorders and their comorbidities offers unparalleled opportunity to appreciate the amazing complexity, resourcefulness, and interconnectedness of the human brain. Hopefully, as clinicians and educators gain increased awareness and understanding of ADDs, they will be better able to dispel the widespread ignorance that unnecessarily increases the frustration and pain of those many children, adolescents, and adults who suffer not only from ADD impairments but also from the blame and criticism of those who continue to believe that ADDs result from lack of willpower rather than from disorders of brain neurochemistry that

unfold developmentally in interactions with the individual's environment.

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