CHAPTER 1

History of ADHD

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Attention-deficit/hyperactivity disorder (ADHD) continues to be the current diagnostic label for children and adults presenting with significant problems with attention, and typically with impulsiveness and excessive activity as well. Children and adults with ADHD represent a rather heterogeneous population that displays considerable variation in the degree of members' symptoms, age of onset, cross-situational pervasiveness of those symptoms, and the extent to which other disorders occur in association with ADHD. The disorder represents one of the most common reasons children with behavioral problems are referred to medical and mental health practitioners in the United States and is one of the most prevalent childhood psychiatric disorders. Currently, referrals of adults for ADHD are also increasing at a rapid pace; until the 1990s and even to date, this age group has been a markedly underrecognized and underserved segment of the ADHD population.

This chapter presents an overview of ADHD's history—a history that spans more than two centuries in the medical and scientific literature. Whereas the previous edition noted that the medical history of ADHD began with Still's description of childhood cases in 1902, we now know that a number of earlier physicians described such cases dating back to the textbook by Melchior Adam Weikard published in German in 1775 (Barkley & Peters, 2012). This extends the history of ADHD in the medical literature back another 127 years. These new additions to the history of ADHD are described below. But given that the history of ADHD as understood from 1902 through 2006 has changed little since the preceding edition of this text (Barkley, 2006), little has been needed to update those sections of this chapter. In contrast, developments since that previous edition are described at the end of this chapter.

In the history of ADHD reside the nascent concepts that serve as the foundation for the current conceptualization of the disorder as largely involving self-regulation and executive functioning, as discussed here by Eric Willcutt (Chapter 15) and myself (Chapter 16). In this history also can be seen the emergence of current notions about its treatment. Such a history remains important for any serious student of ADHD, for it shows that many contemporary themes concerning its nature arose long ago. They have recurred throughout the subsequent history of ADHD to the present as clinicians and scientists strove for a clearer, more accurate understanding of the condition, its comorbid disorders, life course, impairments, and etiologies. Readers are directed to other and earlier sources for additional discussions of the history of this disorder (Accardo &

Blondis, 2000; Goldstein & Goldstein, 1998; Kessler, 1980; Ross & Ross, 1976, 1982; Schachar, 1986; Taylor, 2011; Warnke & Riederer, 2013; Werry, 1992).

THE HISTORICAL ORIGINS OF ADHD

The Late 1700s

One can find literary references to individuals having serious problems with inattention, hyperactivity, and poor impulse control in Shakespeare, who made mention of a malady of attention in *King Henry VIII*. But as of this writing, the medical history of ADHD-like descriptions traces back nearly 240 years to 1775. This early history has been expertly detailed in several sources (Taylor, 2011; Warnke & Riederer, 2013) but should be amended by more recent discoveries in that history, as discussed below.

It now appears that the first description of disorders of attention, at least as of this writing, occurred in the medical textbook by Melchior Adam Weikard in German in 1775 (or perhaps even 1770; see Barkley & Peters, 2012). Initially published anonymously, hence the difficulty with ascertaining the year of its initial publication, the medical textbook by Weikard described adults and children who were inattentive, distractible, lacking in persistence, overactive, and impulsive, which is quite similar to today's description of ADHD. Weikard implied that the disorder could result from poor childrearing but also suggests some biological predispositions as well. For treatment, he recommended sour milk, plant extracts, horseback riding, and even seclusion for severe cases.

This textbook would be followed in short order in 1798 with much more detailed descriptions of ADHD-like symptoms in the medical textbook by the Scottish physician Alexander Crichton (see Palmer & Finger, 2001), who may well have studied with Weikard in his medical training. Crichton described two types of attention disorders. The first was a disorder of distractibility, frequent shifting of attention or inconstancy, and lack of persistence or concentration, and aligns more closely with the attention disturbance evident in ADHD. The second was a disorder of diminished power or energy of attention that seems more like the attention problem evident in current descriptions of children and adults with sluggish cognitive tempo (SCT), which is briefly discussed in Chapter 2 on ADHD symptoms and subtypes and far more detailed coverage in Chapter 17, this volume. Crichton had little to say about this second disorder of attention other than it may be associated with debility or torpor of the body that weakens attention and results in individuals who are often characterized as retiring, unsocial, and having few friendships or attachments of any kind; even those few friendships seldom were of a durable nature. He argued that the faculty of attention can become sufficiently weakened that it may leave an individual insensible to external objects or to impressions that ordinarily would awaken social feelings.

The 1800s

In 1809, John Haslam described what may have been a case of ADHD in a 10-year-old boy who was uncontrollable, impulsive, and "a creature of volition and the terror of the family" (p. 199). Three years later, the famous American physician Benjamin Rush (1812) discussed three cases involving "the total perversion of moral faculties" (p. 359), which included the inability to focus attention. In the mid-1800s, the German pediatrician Heinrich Hoffman (1865) published a book of poems about psychological conditions of children based on observations from his clinical practice. He described both a very impulsive fidgety child he called "Fidgety Phil" and a very inattentive, daydreamy child he called "Johnny Head-in-Air" (see Stewart, 1970). Two years thereafter in England, Henry Maudsley (1867) published a report about a child who was driven by impulsiveness and was also quite destructive. In 1899, the Scottish psychiatrist, Thomas Clouston discussed cases of impulsive children who had learning problems. Much later in the United States, William James (1890/1950) noted in his *Principles of Psychology* a normal variant of character that he called the "explosive will," which may resemble the difficulties experienced by those who today are described as having ADHD.

In France the concept of ADHD may have originated in 1845 in the description of children and adults with attention problems by Jean-Etienne Dominique Esquirol, who believed that the insane no longer "enjoy the faculty of fixing, and directing their attention" (p. 28). Or perhaps the French history of ADHD began in the notion of "mental instability" that appears in the French medical literature in the 1885–1895 period under the leadership of Désiré-Magloire Bourneville (1885, 1895; see Bader & Hidjikhani, in press) at the Hospital Bicêtre in Paris. He observed children and adolescents who had been labeled "abnormal" and placed in medical and educational institutions, many

of whom were characterized by attention and other behavioral problems. Charles Baker, a student of Bourneville, wrote a clinical description of hyperactive and impulsive symptoms in 4 children in his 1892 thesis, according to Bourneville (1895). Attention problems were also mentioned in one case in this work.

THE PERIOD 1900 TO 1959

Still's Description in 1902

In the earlier editions of this text, credit for authoring the first medical description of cases resembling ADHD was awarded to George Still in 1902, owing to the lack of information on the earlier works of Weikard and Crichton. While this no longer remains the case, having been ousted from this credit by the discovery of Weikard's description noted earlier, Still did provide probably the most detailed account of the symptoms of these cases and the largest sample of such cases to that time. For these reasons, his observations deserve some recognition here. In a series of three published lectures to the Royal College of Physicians in 1902, Still described 43 children in his clinical practice who had serious problems with sustained attention; he agreed with William James (1890/1950) that such attention may be an important element in the "moral control of behavior." Most were also quite overactive. Many were often aggressive, defiant, resistant to discipline, and excessively emotional or "passionate." These children showed little "inhibitory volition" over their behavior, and they also manifested "lawlessness," spitefulness, cruelty, and dishonesty. Still proposed that the immediate gratification of the self was the "keynote" quality of these children, among other attributes. Passion (or heightened emotionality) was the most commonly observed attribute and the most noteworthy. Still noted further that such children had an insensitivity to punishment, for they would be punished (even physically) yet engage in the same infraction within a matter of hours.

Still believed that these children displayed a major "defect in moral control" over their behavior; a defect that was relatively chronic in most cases. He believed that in some cases, these children had acquired the defect secondary to an acute brain disease, and it might remit on recovery from the disease. He noted a higher risk for criminal acts in later development in some, though not all, of the chronic cases. Although this defect could be associated with intellectual retardation, as it was in 23 of the cases, it could also arise in children of near-normal intelligence, as it seemed to do in the remaining 20.

To Still (1902), the moral control of behavior meant "the control of action in conformity with the idea of the good of all" (p. 1008). Moral control was thought to arise out of a cognitive or conscious comparison of the individual's volitional activity with that of the good of all—a comparison he termed "moral consciousness." For purposes that will become evident later, it is important to realize here that to make such a comparison inherently involves the capacity to understand the consequences of one's actions over time and to hold in mind forms of information about oneself and one's actions, along with information on their context. Those forms of information involve the action being proposed by the individual, the context, and the moral principle or rule against which it must be compared. This notion may link Still's views with the contemporary concepts of self-awareness, working memory, and rule-governed behavior discussed later in this text. Still did not specifically identify these inherent aspects of the comparative process, but they are clearly implied in the manner in which he used the term "conscious" in describing this process. He stipulated that this process of comparison of proposed action to a rule concerning the greater good involved the critical element of the conscious or cognitive relation of individuals to their environment, or "self-awareness." Intellect was recognized as playing a part in moral consciousness, but equally or more important was the notion of volition or will. The latter is where Still believed the impairment arose in many of those with defective moral control who suffered no intellectual delay. Volition was viewed as being primarily inhibitory in nature, that a stimulus to act must be overpowered by the stimulus of the idea of the greater good of all.

Still concluded that a defect in moral control could arise as a function of three distinct impairments: "(1) defect of cognitive relation to the environment; (2) defect of moral consciousness; and (3) defect in inhibitory volition" (p. 1011). He placed these impairments in a hierarchical relation to each other in the order shown, arguing that impairments at a lower level would affect those levels above it and ultimately the moral control of behavior. Much as researchers do today, Still noted a greater proportion of males than females (3:1) in his sample, and he observed that the disorder appeared to arise in most cases before 8 years of age (typically in early childhood). Many of Still's cases displayed a

proneness to accidental injuries—an observation corroborated by numerous subsequent studies reviewed in a later chapter. And Still saw these youngsters as posing an increased threat to the safety of other children because of their aggressive or violent behavior. Alcoholism, criminality, and affective disorders such as depression and suicide were noted to be more common among their biological relatives—an observation once again buttressed by numerous studies published in recent years. Some of the children displayed a history of significant brain damage or convulsions, whereas others did not. A few had associated tic disorders, or "microkinesia"; this was perhaps the first time tic disorders and ADHD were noted to be comorbid conditions. We now recognize that while 10–15% of children with ADHD may manifest some form of tic disorder, as many as 50–70% of children with tic disorders and Tourette syndrome may have ADHD (Simpson, Jung, & Murphy, 2011).

Although many of Still's subjects were reported to have a chaotic family life, others came from households that provided a seemingly adequate upbringing. In fact, Still believed that when poor childrearing was clearly involved, the children should be exempt from the category of lack of moral control; he reserved it instead only for children who displayed a morbid (organic) failure of moral control despite adequate training. He proposed a biological predisposition to this behavioral condition that was probably hereditary in some children but the result of pre- or postnatal injury in others. In keeping with the theorizing of James (1890/1950), Still hypothesized that the deficits in inhibitory volition, moral control, and sustained attention were causally related to each other and to the same underlying neurological deficiency. He cautiously speculated on the possibility of either a decreased threshold for inhibition of responding to stimuli or a cortical disconnection syndrome, in which intellect was dissociated from "will" in a manner that might be due to neuronal cell modification. Any biologically compromising event that could cause significant brain damage ("cell modification") and retardation could, he conjectured, in its milder forms lead only to this defective moral control.

Also in England, Alfred Tredgold (1908) described children of low intelligence having abnormal behavior and limited powers of attention, impulse control, and willpower. He extended Still's theories and observations that early brain damage might present as behavioral and learning problems in later childhood. Foreshadowing current views of treatment, both Still (1902) and Tredgold found that temporary improvements in conduct might be achieved by alterations in the environment or by medications, but they stressed the relative permanence of the defect even in these cases. They emphasized the need for special educational environments for these children. We see here the origins of many later and even current notions about children with ADHD and oppositional defiant disorder (ODD), although it would take almost 70 years to return to many of them—owing in part to the ascendance in the interim of psychoanalytic, psychodynamic, and behavioral views that overemphasized childrearing as largely causing such behavioral disorders in children. The children described by Still and Tredgold would probably now be diagnosed as having not only ADHD but also ODD or conduct disorder (CD), and most likely a learning disability as well (see Chapters 5 and 6).

Around this same time, in Spain, the physician Rodriguez-Lafora (1917) wrote about his interests in childhood mental illness and described a group of children having psychopathic constitutions, a subset of which he called the "unstables." His description of them matches closely the modern view of ADHD (Bauermeister & Barkley, 2010), including inconstancy of attention, excessive activity, and impulsive behavior, as does his observation that such children get carried away by their adventurous temperament.

The Influence of the Encephalitis Epidemic

The history of interest in ADHD in North America can be traced to the outbreak of an encephalitis epidemic in 1917–1918, when clinicians were presented with a number of children who survived this brain infection but were left with significant behavioral and cognitive sequelae (Cantwell, 1981; Kessler, 1980; Stewart, 1970). Numerous articles that reported these sequelae (Ebaugh, 1923; Strecker & Ebaugh, 1924; Stryker, 1925) included many of the characteristics we now incorporate into the concept of ADHD. Such children were described as being impulsive and having impaired attention and regulation of activity, as well as impairments in other cognitive abilities, including memory; they were often noted to be socially disruptive as well. Symptoms of what is now called ODD, as well as delinquency and CD, also arose in some cases. "Postencephalitic behavior disorder," as it was called, was clearly the result of brain damage. The large number of affected

children resulted in significant professional and educational interest in this behavioral disorder. Its severity was such that many children were recommended for care and education outside the home and away from normal educational facilities. Despite a rather pessimistic view of the prognosis of these children, some facilities reported significant success in their treatment with simple behavior modification programs and increased supervision (Bender, 1942; Bond & Appel, 1931).

The Origins of a Brain Damage Syndrome

This association of a brain disease with behavioral pathology apparently led early investigators to study other, potential causes of brain injury in children and their behavioral manifestations, including birth trauma (Shirley, 1939); other infections besides encephalitis, such as measles (Meyer & Byers, 1952); lead toxicity (Byers & Lord, 1943); epilepsy (Levin, 1938); and head injury (Blau, 1936; Werner & Strauss, 1941). All were studied in children and found to be associated with numerous cognitive and behavioral impairments, including the triad of ADHD symptoms noted earlier. Other terms introduced during this era for children displaying these behavioral characteristics were "organic driveness" (Kahn & Cohen, 1934) and "restlessness" syndrome (Childers, 1935; Levin, 1938). Many of the children seen in these samples also had mental retardation or more serious behavioral disorders than what is today called ADHD. It would take investigators several decades to attempt to parse out the separate contributions of intellectual delay, learning disabilities, or other neuropsychological deficits from those of behavioral deficits in the maladjustment of these children. Even so, scientists at this time would discover that activity level was often inversely related to intelligence in children, increasing as intelligence declined in a sample—a finding supported in many subsequent studies (Rutter, 1989). It should also be noted that a large number of children in these older studies did in fact have brain damage or signs of such damage (epilepsy, hemiplegias, etc.).

Notable during this era was also recognition of the striking similarity between hyperactivity in children and the behavioral sequelae of frontal lobe lesions in primates (Blau, 1936; Levin, 1938). Frontal lobe ablation studies of monkeys had been done more than 60 years earlier (Ferrier, 1876), and the lesions were known to result in excessive restlessness, poor ability to sustain interest in activities, aimless wandering, and excessive appetite, among other behavioral changes. Several investigators, such as Levin (1938), used these similarities to postulate that severe restlessness in children might well be the result of pathological defects in the forebrain structures, although gross evidence of such was not always apparent in many of these children. Later, investigators (e.g., Barkley, 1997a; Chelune, Ferguson, Koon, & Dickey, 1986; Lou, Henriksen, & Bruhn, 1984; Lou, Henriksen, Bruhn, Borner, & Nielsen, 1989; Mattes, 1980) would return to this notion, but with greater evidence to substantiate their claims. Milder forms of hyperactivity, in contrast, were attributed in this era to psychological causes, such as "spoiled" child-rearing practices or delinquent family environments. This idea that poor or disrupted parenting causes ADHD would also be resurrected in the 1970s, and it continues even today among many laypeople and critics of ADHD.

Over the next decade, it became fashionable to consider most children hospitalized in psychiatric facilities with this symptom picture to have suffered from some type of brain damage (e.g., encephalitis or prenatal–perinatal trauma), whether or not there was evidence of such in the clinical history of the case. The concept of the "brain-injured child" was born in this era (Strauss & Lehtinen, 1947) and applied to children with these behavioral characteristics, many of whom had insufficient or no evidence of brain pathology. In fact, Strauss and Lehtinen argued that the psychological disturbances alone were de facto evidence of brain injury as the etiology. Owing in part to the absence of such evidence of brain damage, this term would later evolve into the concept of "minimal brain damage" and eventually "minimal brain dysfunction" (MBD) by the 1950s and 1960s. Even so, a few early investigators, such as Childers (1935), would raise serious questions about the notion of brain damage in these children when no historical documentation of damage existed. Substantial recommendations for educating these "brain-damaged" children were made in the earlier text by Tredgold (1908) and later in the classic text on special education by Strauss and Lehtinen (1947), which served as a forerunner to special educational services adopted much later in U.S. public schools. These recommendations included placing these children in smaller, more carefully regulated classrooms and reducing the amount of distracting stimulation in the environment. Strikingly austere classrooms were developed,

in which teachers avoided wearing jewelry or brightly colored clothing, and few pictures adorned the walls so as not to interfere unnecessarily with the education of these highly distractible students.

Although the population served by the Pennsylvania center in which Strauss, Werner, and Lehtinen worked principally contained children with mental retardation, the work of Cruickshank and his students (Dolphin & Cruickshank, 1951a, 1951b, 1951c) later extended these neuropsychological findings to children with cerebral palsy but near-normal or normal intelligence. This extension resulted in the extrapolation of the educational recommendations of Strauss to children without mental retardation who manifested behavioral or perceptual disturbances (Cruickshank & Dolphin, 1951; Strauss & Lehtinen, 1947). Echoes of these recommendations are still commonplace today in most educational plans for children with ADHD or learning disabilities, despite the utter lack of scientific support for their efficacy (Kessler, 1980; Routh, 1978; Zentall, 1985). These classrooms are historically significant because they were predecessors as well as instigators of the types of educational resources that would be incorporated into the initial Education for All Handicapped Children Act of 1975 (Public Law 94-142) mandating the special education of children with learning disabilities and behavioral disorders, and its later reauthorization, the Individuals with Disabilities Education Act of 1990 (IDEA; Public Law 101-476).

The Beginnings of Child Psychopharmacology for ADHD

Another significant series of articles on the treatment of hyperactive children appeared from 1937 to 1941. They marked the beginnings of medication therapy (particularly stimulants) for behaviorally disordered children in particular, as well as the field of child psychopharmacology in general (Bradley, 1937; Bradley & Bowen, 1940; Molitch & Eccles, 1937). Initiated originally to treat the headaches that resulted from conducting pneumoencephalograms during research studies of these disruptive youth, the administration of amphetamine resulted in a noticeable improvement in their behavioral problems and academic performance. Later studies would also confirm such a positive drug response in half or more of hyperactive hospitalized children (Laufer, Denhoff, & Solomons, 1957). As a result, by the 1970s, stimulant medications were gradually becoming the treatment of choice for the behavioral symptoms now associated with ADHD. And so they remain today (see Chapter 27).

The Emergence of a Hyperkinetic Impulse Syndrome

In the 1950s, researchers began a number of investigations into the neurological mechanisms underlying these behavioral symptoms, the most famous of which was probably that by Laufer and colleagues (1957). These writers referred to children with ADHD as having "hyperkinetic impulse disorder," and reasoned that the central nervous system (CNS) deficit occurred in the thalamic area. Here, poor filtering of stimulation occurred, allowing an excess of stimulation to reach the brain. The evidence was based on a study of the effects of the "photo-Metrozol" method, in which the drug metronidazole (Metrozol) is administered while flashes of light are presented to a child. The amount of drug required to induce a muscle jerk of the forearms, along with a spike wave pattern on the electroencephalogram (EEG), serves as the measure of interest. Laufer and colleagues found that inpatient children with hyperactivity required less Metrozol than those without hyperactivity to induce this pattern of response. This finding suggested that the hyperactive children had a lower threshold for stimulation, possibly in the thalamic area. No attempts to replicate this study have been done, and it is unlikely that such research would pass today's standards of ethical conduct in research required by institutional review boards on research with human subjects. Nevertheless, it remains a milestone in the history of the disorder for its delineation of a more specific mechanism that might give rise to hyperactivity (low cortical thresholds or overstimulation). Others at the time also conjectured that the existence of an imbalance between cortical and subcortical areas caused diminished control of subcortical areas responsible for sensory filtering that permitted excess stimulation to reach the cortex (Knobel, Wolman, & Mason, 1959).

By the end of this era, it seemed well accepted that hyperactivity was a brain damage syndrome, even when evidence of damage was lacking. The disorder was thought to be best treated through educational classrooms characterized by reduced stimulation or through residential centers. Its prognosis was considered fair to poor. The possibility that a relatively new class of medications, the stimulants, might hold promise for its treatment was beginning to be appreciated.

THE PERIOD 1960 TO 1969

The Decline of MBD and the Rise of Hyperactivity

In the late 1950s and early 1960s, critical reviews began to question the concept of a unitary syndrome of brain damage in children. They also pointed out the logical fallacy that if brain damage resulted in some of these behavioral symptoms, these symptoms could be pathognomonic of brain damage without any other corroborating evidence of CNS lesions. Chief among these critical reviews were those of Birch (1964), Herbert (1964), and Rapin (1964), who questioned the validity of applying the concept of brain damage to children who had only equivocal signs of neurological involvement, not necessarily damage. A plethora of research followed on children with MBD (see Rie & Rie, 1980, for reviews); in addition, a task force by the National Institute of Neurological Diseases and Blindness (Clements, 1966) recognized at least 99 symptoms for this disorder. The concept of MBD would die a slow death as it eventually was recognized to be vague, overinclusive, of little or no prescriptive value, and without much neurological evidence (Kirk, 1963). Its remaining value was its emphasis on neurological mechanisms over the often excessive, pedantic, and convoluted environmental mechanisms proposed at that time—particularly those etiological hypotheses stemming from psychoanalytical theory, which blamed parental and family factors entirely for these problems (Hertzig, Bortner, & Birch, 1969; Kessler, 1980; Taylor, 1983). The term "MBD" would eventually be replaced by more specific labels applying to somewhat more homogeneous cognitive, learning, and behavioral disorders, such as "dyslexia," "language disorders," "learning disabilities," and "hyperactivity." These new labels were based on children's observable and descriptive deficits rather than on some underlying, unobservable etiological mechanism in the brain.

The Hyperactivity Syndrome

As dissatisfaction with the term "MBD" was occurring, clinical investigators shifted their emphasis to the behavioral symptom thought to most characterize the disorder—that of hyperactivity. And so the concept of a hyperactivity syndrome arose, described in the classic articles by Laufer and Denhoff (1957), Chess (1960), and other reports of this era (Burks, 1960; Ounsted, 1955; Prechtl & Stemmer, 1962). Chess defined "hyperactivity" as follows: "The hyperactive child is one who carries out activities at a higher than normal rate of speed than the average child, or who is constantly in motion, or both" (p. 2379). Chess's article was historically significant for several reasons: (1) It emphasized activity as the defining feature of the disorder rather than speculation about underlying neurological causes, as other scientists of the time would also do; (2) it stressed the need to consider objective evidence of the symptom beyond the subjective reports of parents or teachers; (3) it took the blame for the child's problems away from the parents; and (4) it separated the syndrome of hyperactivity from the concept of a brain damage syndrome. Other scientists of this era would emphasize similar points (Werry & Sprague, 1970). Hyperactivity would now be recognized as a behavioral syndrome that could not only arise from organic pathology but also occur in its absence. Even so, it would continue to be viewed as the result of some biological difficulty rather than as being due solely to environmental causes.

Chess (1960) described the characteristics of 36 children diagnosed with "physiological hyperactivity" from a total of 881 children seen in a private practice. The ratio of males to females was approximately 4:1, and many children were referred prior to 6 years of age, intimating a relatively earlier age of onset than that for other childhood behavioral disorders. Educational difficulties were common in this group, particularly scholastic underachievement, and many displayed oppositional defiant behavior and poor peer relationships. Impulsive and aggressive behaviors, as well as poor attention span, were commonly associated characteristics. Chess believed that the hyperactivity could also be associated with mental retardation, organic brain damage, or serious mental illness (e.g., schizophrenia). Similar findings in later research would lead others to question the specificity and hence the utility of this symptom for the diagnosis of ADHD (Douglas, 1972). As with many of today's prescriptions, a multimodal treatment approach incorporating parent counseling, behavior modification, psychotherapy, medication, and special education was recommended. Unlike Still (1902), Chess and others writing in this era stressed the relatively benign nature of hyperactivity's symptoms and claimed that in most cases they resolved by puberty (Laufer & Denhoff, 1957; Solomons, 1965). Here, then, were the beginnings of a belief that would be widely held among clinicians well into the 1980s—that hyperactivity (ADHD) was outgrown by adolescence.

Also noteworthy in this era was the definition of hyperactivity given in the official diagnostic nomenclature at the time, the second edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-II; American Psychiatric Association, 1968). It employed only a single sentence describing the hyperkinetic reaction of childhood disorder and, following the lead of Chess (1960), stressed the view that the disorder was developmentally benign: "The disorder is characterized by overactivity, restlessness, distractibility, and short attention span, especially in young children; the behavior usually diminishes by adolescence" (p. 50).

Europe and North America Diverge in Viewpoints

It is likely that during this period (or even earlier), the perspective on hyperactivity in North America began to diverge from that in Europe, particularly Great Britain. In North America, hyperactivity would become a behavioral syndrome recognized chiefly by greater-than-normal levels of activity; would be viewed as a relatively common disturbance of childhood; would not necessarily be associated with demonstrable brain pathology or mental retardation; and would be regarded as more of an extreme degree in the normal variation of temperament in children. In Great Britain, the earlier and narrower view of a brain damage syndrome would continue into the 1970s: Hyperactivity or hyperkinesis was seen as an extreme state of excessive activity of an almost driven quality; was viewed as highly uncommon; and was usually thought to occur in conjunction with other signs of brain damage (e.g., epilepsy, hemiplegias, or mental retardation) or a clearer history of brain insult (e.g., trauma or infection) (Taylor, 1988). The divergence in views would lead to large discrepancies between North American and European estimations of the prevalence of the disorder, their diagnostic criteria, and their preferred treatment modalities. A rapprochement between these views would not occur until well into the 1980s (Rutter, 1988, 1989; Taylor, 1986, 1988).

The Prevailing View by 1969

As Ross and Ross (1976) noted in their exhaustive and scholarly review of the era, the perspective on hyperactivity in the 1960s was that it remained a brain dysfunction syndrome, although of a milder magnitude than previously believed. The disorder was no longer ascribed to brain damage; instead, a focus on brain mechanisms prevailed. The disorder was also viewed as having a predominant and relatively homogeneous set of symptoms, chief among which was excessive activity level or hyperactivity. Its prognosis was now felt to be relatively benign because it was believed to be often outgrown by puberty. The recommended treatments now consisted of short-term treatment with stimulant medication and psychotherapy, in addition to the minimum-stimulation types of classrooms recommended in earlier years.

THE PERIOD 1970 TO 1979

Research in the 1970s took a quantum leap forward, with more than 2,000 published studies by the time the decade ended (Weiss & Hechtman, 1979). Numerous clinical and scientific textbooks (Cantwell, 1975; Safer & Allen, 1976; Trites, 1979; Wender, 1971) appeared, along with a most thorough and scholarly review of the literature by Ross and Ross (1976). Special journal issues were devoted to the topic (Barkley, 1978; Douglas, 1976), along with numerous scientific gatherings (Knights & Bakker, 1976, 1980). Clearly, hyperactivity had become a subject that attracted serious professional, scientific, and popular attention.

By the early 1970s, the defining features of hyperactivity or hyperkinesis were broadened to include what investigators previously felt to be only associated characteristics, including impulsivity, short attention span, low frustration tolerance, distractibility, and aggressiveness (Marwitt & Stenner, 1972; Safer & Allen, 1976). Others (Wender, 1971, 1973) persisted with the excessively inclusive concept of MBD, in which even more features (e.g., motor clumsiness, cognitive impairments, and parent–child conflict) were viewed as hallmarks of the syndrome, and in which hyperactivity was unnecessary for the diagnosis. As noted earlier, the diagnostic term "MBD" would fade from clinical and scientific usage by the end of this decade—the result in no small part of the scholarly tome by Rie and Rie (1980) and critical reviews by Rutter (1977, 1982). These writings emphasized the lack of evidence for such a broad syndrome. The symptoms were not well defined, did not correlate significantly among themselves, had no well-specified etiology, and displayed no common course and outcome. The heterogeneity of the disorder was overwhelming, and more than a few commentators took note of the apparent hypocrisy in defin-

ing an MBD syndrome with the statement that there was often little or no evidence of neurological abnormality (Wender, 1971). Moreover, even in cases of well-established cerebral damage, the behavioral sequelae were not uniform across cases, and hyperactivity was seen in only a minority of individuals. Hence, contrary to 25 years of theorizing to this point, hyperactivity was not a common sequela of brain damage; children with true brain damage did not display a uniform pattern of behavioral deficits; and children with hyperactivity rarely had substantiated evidence of neurological damage (Rutter, 1989).

Wender's Theory of MBD

This decade was notable for two different models of the nature of ADHD (see also Barkley, 1998): Wender's theory of MBD (outlined here) and Douglas's model of attention and impulse control in hyperactive children (discussed in a later section). At the start of the decade, Wender (1971) described the essential psychological characteristics of children with MBD as comprising six clusters of symptoms: problems in (1) motor behavior, (2) attentional and perceptual–cognitive functioning, (3) learning, (4) impulse control, (5) interpersonal relations, and (6) emotion. Many of the characteristics first reported by Still (1902) were echoed by Wender (1971) within these six domains of functioning.

1. Within the realm of motor behavior, the essential features were noted to be hyperactivity and poor motor coordination. Excessive speech, colic, and sleeping difficulties were thought to be related to the hyperactivity. Foreshadowing the later official designation of a group of children with attentional problems but without hyperactivity (American Psychiatric Association, 1980), Wender (1971) expressed the opinion that some of these children who were hypoactive and listless still demonstrated attention disturbances. Such cases might now be considered to have the predominantly inattentive type of ADHD. He argued that they should be viewed as having this syndrome because of their manifestation of many of the other difficulties thought to characterize it.

2. Short attention span and poor concentration were described as the most striking deficits in the domain of attention and perceptual–cognitive functioning. Distractibility and daydreaming were also included with these attention disturbances, as was poor organization of ideas or percepts.

3. Learning difficulties were another domain of dysfunction, with most of these children observed to be doing poorly in their academic performance. A large percentage were described as having specific difficulties with learning to read, with handwriting, and with reading comprehension and arithmetic.

4. Impulse control problems, or a decreased ability to inhibit behavior, were identified as a characteristic of most children with MBD. Within this general category, Wender (1971) included low frustration tolerance; an inability to delay gratification; antisocial behavior; lack of planning, forethought, or judgment; and poor sphincter control, leading to enuresis and encopresis. Disorderliness or lack of organization and recklessness (particularly with regard to bodily safety) were also listed within this domain of dysfunction.

5. In the area of interpersonal relations, Wender (1971) singled out the unresponsiveness of these children to social demands as the most serious. Extraversion, excessive independence, obstinence, stubbornness, negativism, disobedience, noncompliance, sassiness, and imperviousness to discipline were some of the characteristics that instantiated the problem with interpersonal relations.

6. Finally, within the domain of emotional difficulties, Wender (1971) included increased lability of mood, altered reactivity, increased anger, aggressiveness, and temper outbursts, as well as dysphoria. The dysphoria of these children involved the specific difficulties of anhedonia, depression, low self-esteem, and anxiety. A diminished sensitivity to both pain and punishment was also felt to typify this area of dysfunction in children with MBD. All these symptoms bear a striking resemblance to the case descriptions that Still (1902) had provided in lectures to support his contention that a defect in moral control and volitional inhibition could exist in children apart from intellectual delay.

Wender (1971) theorized that these six domains of dysfunction could be best accounted for by three primary deficits: (1) a decreased experience of pleasure and pain, (2) a generally high and poorly modulated level of activation, and (3) extraversion. A consequence of the first deficit was that children with MBD would prove less sensitive to both reward and punishment, making them less susceptible to social influence. The generally high and poorly modulated level of activation was thought to be an aspect of poor inhibition. Hyperactivity, of course, was the consummate demon-

stration of this high level of activation. The problems with poor sustained attention and distractibility were conjectured to be secondary aspects of high activation. Emotional overreactivity, low frustration tolerance, quickness to anger, and temper outbursts resulted from the poor modulation of activation. These three primary deficits, then, created a cascade of effects into the larger social ecology of these children, resulting in numerous interpersonal problems and academic performance difficulties.

Like Still (1902), Wender (1971) gave a prominent role to the construct of poor inhibition. He believed that it explained the activation difficulties and the attention problems stemming from them, as well as the excessive emotionality, the low frustration tolerance, and the hot-temperedness of these children. It is therefore quite puzzling why deficient inhibition was not made a primary symptom in this theory, in place of high activation and poor modulation of activation.

Unlike Still (1902), who attempted to devise a theory, however, Wender (1971) did not say much about normal developmental processes with respect to the three primary areas of deficit, and therefore did not clarify more precisely what might be going awry in them to give rise to these characteristics of MBD. The exception was his discussion of a diminished sensitivity to the reasonably well-understood processes of reinforcement and punishment. A higher-than-normal threshold for pleasure and pain, as noted earlier, was thought to create these insensitivities to behavioral consequences.

From a present-day perspective, Wender (1971) is also unclear about a number of issues. For instance, how would the three primary deficits account for the difficulties with motor coordination that occurred alongside hyperactivity in his category of motor control problems? It is doubtful that the high level of activation that was said to cause the hyperactivity would also cause these motor deficits. Nor is it clear just how the academic achievement deficits in reading, math, and handwriting could arise from the three primary deficits in the model. It is also unclear why the construct of extraversion needed to be proposed at all, if what Wender meant by it was reduced social inhibition. This model might be just as parsimoniously explained by the deficit in behavioral inhibition already posited. And the meaning of the term "activation" as used by Wender is not very clearly specified. Did it refer to excessive behavior, in which case hyperactivity would have sufficed? Or did it refer to level of CNS arousal, in which case ample subsequent evidence has not found this to be the case (Hastings & Barkley, 1978; Rosenthal & Allen, 1978)? To his credit, Wender recognized the abstract nature of the term "activation" as he employed it in this theory but retained it because he felt it could be used to incorporate both hyperactivity and hypoactivity in children. It is never made clear just how this could be the case, however. Finally, Wender failed to distinguish symptoms from their consequences (impairments). The former would be the behavioral manifestations directly associated with or stemming from the disorder itself, such as impulsiveness, inattention, distractibility, and hyperactivity. The latter would be the effects of these behaviors on the social environment, such as interpersonal conflict within the family, poor educational performance, peer rejection, and accident proneness, to name just a few.

From the advantage of hindsight and subsequent research over the decades since the formulation of this theory, it is also evident that Wender (1971) was combining the symptoms of ODD (and even CD) with those of ADHD to form a single disorder. Still (1902) did very much the same thing. This was understandable given that clinic-referred cases were the starting point for both theories, and many clinic-referred cases are comorbid for both disorders (ADHD and ODD). However, sufficient accumulated evidence has subsequently shown that ADHD and ODD are not the same disorder (August & Stewart, 1983; Hinshaw, 1987; Stewart, deBlois, & Cummings, 1980).

The Emergence of a Central Place for Attention Deficits

At this time, disenchantment developed over the exclusive focus on hyperactivity as the sine qua non of this disorder (Werry & Sprague, 1970). Significant at this historical juncture would be the article based on the presidential address of Virginia Douglas (1972) to the Canadian Psychological Association. She argued that deficits in sustained attention and impulse control were more likely than just hyperactivity to account for the difficulties seen in these children. These other symptoms were also seen as the major areas on which the stimulant medications used to treat the disorder had their impact. Douglas's article was historically significant in other ways as well. Her extensive and thorough battery of objective measures of various behavioral and cognitive domains, heretofore unused in research on ADHD, allowed her to rule in or out

various characteristics felt to be typical for these children in earlier clinical and scientific lore. For instance, Douglas found that hyperactive children did not necessarily and uniformly have more reading or other learning disabilities than other children, did not perseverate on concept-learning tasks, did not manifest auditory or right–left discrimination problems, and had no difficulties with short-term memory. Most important, she and Susan Campbell (1973) demonstrated that children with hyperactivity were not always more distractible than children without it, and that the sustained attention problems could emerge in conditions in which no significant distractions existed.

The McGill University research team headed by Douglas repeatedly demonstrated that hyperactive children had some of their greatest difficulties on tasks assessing vigilance or sustained attention, such as the continuous-performance test (CPT). These findings would be repeatedly reconfirmed over the next 30 years of research using CPTs (Corkum & Siegel, 1993; Frazier, Demareem, & Youngstrom, 2004). Variations of this test would eventually be standardized and commercially marketed for diagnosis of the disorder (Conners, 1995; Gordon, 1983; Greenberg & Waldman, 1992). Douglas (1972) remarked on the extreme degree of variability demonstrated during task performances by these children—a characteristic that would later be advanced as one of the defining features of the disorder. The McGill team (Freibergs, 1965; Freibergs & Douglas, 1969; Parry & Douglas, 1976) also found that hyperactive children could perform at normal or near-normal levels of sustained attention under conditions of continuous and immediate reinforcement, but that their performance deteriorated dramatically when partial reinforcement was introduced, particularly at schedules below 50% reinforcement. Campbell, Douglas, and Morgenstern (1971) further demonstrated substantial problems with impulse control and field dependence in the cognitive styles of hyperactive children. Like Still (1902), roughly 70 years earlier, Douglas commented on the probable association between deficits in attention–impulse control and deficiencies in moral development that were plaguing her subjects, particularly in their adolescent years. The research of the McGill team showed dramatic improvements in these attention deficiencies during stimulant medication treatment, as did the research at other laboratories at the time (Conners & Rothschild, 1968; Sprague, Barnes, & Werry, 1970).

Finally, of substantial significance were the observations of Douglas's colleague, Gabrielle Weiss, from her follow-up studies (see Weiss & Hechtman, 1986) that although the hyperactivity of these children often diminished by adolescence, their problems with poor sustained attention and impulsivity persisted. This persistence of the disabilities and the risk for greater academic and social maladjustment would be identified by other research teams from their own follow-up investigations (Mendelson, Johnson, & Stewart, 1971), and would be better substantiated by more rigorous studies in the next two decades (see Barkley, Fischer, Edelbrock, & Smallish, 1990; Barkley, Fischer, Smallish, & Fletcher, 2002; Gittelman, Mannuzza, Shenker, & Bonagura, 1985).

Douglas's Model of Attention Deficits

Douglas (1980a, 1980b, 1983; Douglas & Peters, 1979) later elaborated, refined, and further substantiated her model of hyperactivity, which culminated in the view that four major deficits could account for symptoms of ADHD: (1) the investment, organization, and maintenance of attention and effort; (2) the inhibition of impulsive responding; (3) the modulation of arousal levels to meet situational demands; and (4) an unusually strong inclination to seek immediate reinforcement. This perspective initiated or guided a substantial amount of research over the next 15 years, including my own early studies (Barkley, 1977, 1989b; Barkley & Ullman, 1975). It constituted a model as close to a scientific paradigm as the field of hyperactivity was likely to have in its history to that point. Yet over the next 10 years results that emerged were somewhat at odds with this perspective. Scientists began seriously to question both the adequacy of an attention model in accounting for the varied behavioral deficits seen in children with ADHD and the effects of stimulant medications on them (Barkley, 1981, 1984; Draeger, Prior, & Sanson, 1986; Haenlein & Caul, 1987; van der Meere & Sergeant, 1988a, 1988b). Also deserving of mention is that such a description of deficiencies constitutes a pattern and not a theory, given that it stipulates no conditional relations among its parts or how they orchestrate to create the problems seen in the disorder. That is, it makes no testable or falsifiable predictions apart from those contained in the pattern so described.

Douglas's article and the subsequent research published by her team were so influential that they were probably the major reasons the disorder was renamed attention deficit disorder (ADD) in 1980 with the publication of DSM-III (American Psychiatric Associa-

tion, 1980). In this revised official taxonomy, deficits in sustained attention and impulse control were formally recognized as being of greater significance in the diagnosis than hyperactivity. The shift to attention deficits rather than hyperactivity as the major difficulty of these children was useful, at least for a time, because of the growing evidence (1) that hyperactivity was not specific to this particular condition but could be noted in other psychiatric disorders (anxiety, mania, autism, etc.); (2) that there was no clear delineation between "normal" and "abnormal" levels of activity; (3) that activity was in fact a multidimensional construct; and (4) that the symptoms of hyperactivity were quite situational in nature in many children (Rutter, 1989). But this approach corrected the problem of definition for little more than a decade before these same concerns were also raised about the construct of attention (multidimensional, situationally variable, etc.). Yet some research would show that at least deficits in vigilance or sustained attention could be used to discriminate this disorder from other psychiatric disorders (Werry, 1988).

Other Developments of the Era

A number of other historical developments during this period deserve mention.

The Rise of Medication Therapy

The first of these developments was the rapidly increasing use of stimulant medication with school-age hyperactive children. This use was no doubt spawned by the significant increase in research showing that stimulants often had dramatic effects on these children's hyperactive and inattentive behavior. A second development was the use of much more rigorous scientific methodology in drug studies, due in large measure to the early studies by C. Keith Conners (then working with Leon Eisenberg at Harvard University), and somewhat later to the research of Robert Sprague at the University of Illinois, Virginia Douglas at McGill University, and John Werry in New Zealand. This body of literature became voluminous (see Barkley, 1977; Ross & Ross, 1976), with more than 120 studies published through 1976 and more than twice this number by 1995 (Swanson, McBurnett, Christian, & Wigal, 1995), making this treatment approach the most well-studied therapy in child psychiatry.

Despite the proven efficacy of stimulant medication, public and professional misgivings about its increasingly widespread use with children emerged. For example, one news account (Maynard, 1970) reported that in Omaha, Nebraska, as much as 5–10% of the children in grade schools were receiving behavior-modifying drugs. This estimate of drug treatment would later be shown to be grossly exaggerated, as much as 10-fold, due to a misplaced decimal point in the story. And this would certainly not be the last instance of the mass media's penchant for hyperbole, sensation, and scandal in their accounts of stimulant medication treatments for ADHD—a penchant that seems only to have increased over subsequent years. Yet the public interest that was generated by the initial reports led to a congressional review of the use of psychotropic medications for schoolchildren. At this same time, the claim was being advanced that hyperactivity was a "myth" arising from intolerant teachers and parents, and an inadequate educational system (Conrad, 1975; Schrag & Divoky, 1975).

Environment as Etiology

Almost simultaneous with this backlash against "drugging" schoolchildren for behavior problems was another significant development in that decade: a growing belief that hyperactivity was a result of environmental causes. It is not just coincidental that this development occurred at the same time that the United States was experiencing a popular interest in natural foods, health consciousness, the extension of life expectancy via environmental manipulations, psychoanalytic theory, and behaviorism. An extremely popular view was that allergic or toxic reactions to food additives, such as dyes, preservatives, and salicylates (Feingold, 1975), caused hyperactive behavior. It was claimed that more than half of all hyperactive children had developed their difficulties because of diet. Effective treatment could be had if families of these children would buy or make foods that did not contain the offending substances. This view became so widespread that organized parent groups or "Feingold associations," comprised mainly of parents advocating Feingold's diet, were established in almost every U.S. state, and legislation was introduced (although not passed) in California requiring that all school cafeteria foods be prepared without these substances. A sizable number of research investigations was undertaken (see Conners, 1980, for a review), the more rigorous of which found these substances to have little, if any, effect on children's behavior. A National Advisory Committee on Hyperkinesis and Food Addi-

tives (1980) that was convened to review this literature concluded more strongly than Conners that the available evidence clearly refuted Feingold's claims. Nevertheless, it would be more than 10 years before this notion receded in popularity, to be replaced by the equally unsupported hypothesis that refined sugar was more to blame for hyperactivity than were food additives (for reviews, see Milich, Wolraich, & Lindgren, 1986; Wolraich, Wilson, & White, 1995).

The emphasis on environmental causes, however, spread to include possible sources other than diet. Block (1977) advanced the rather vague notion that technological development and more rapid cultural change would result in an increasing societal "tempo," causing growing excitation or environmental stimulation. This excitation or stimulation would interact with a predisposition in some children toward hyperactivity, making it manifest. It was felt that this theory explained the apparently increasing incidence of hyperactivity in developed cultures. Ross and Ross (1982) provided an excellent critique of the theory and concluded that there was insufficient evidence in support of it and some that would contradict it. Little evidence suggested that hyperactivity incidence was increasing, though its identification among children may well have been. Nor was there evidence that its prevalence varied as a function of societal development. Instead, Ross and Ross proposed that cultural effects on hyperactivity have more to do with whether important institutions of enculturation are consistent or inconsistent in the demands made and standards set for child behavior and development. These cultural views were said to determine the threshold for deviance that will be tolerated in children, as well as to exaggerate a predisposition to hyperactivity in some children. Consistent cultures will have fewer children diagnosed with hyperactivity, as they minimize individual differences among children and provide clear and consistent expectations and consequences for behavior that conforms to the expected norms. Inconsistent cultures, by contrast, will have more children diagnosed with hyperactivity, as they maximize or stress individual differences and provide ambiguous expectations and consequences to children regarding appropriate conduct. This intriguing hypothesis remains unstudied. However, on these grounds, an equally compelling case could be made for the opposite effects of cultural influences: In highly consistent, highly conforming cultures, hyperactive behavior may be considerably more obvious in children as they are unable to conform to these societal expectations, whereas inconsistent and low-conforming cultures may tolerate deviant behavior to a greater degree as part of the wider range of behavioral expression they encourage.

A different environmental view—that poor childrearing generally and poor child behavior management specifically lead to hyperactivity—was advanced by schools of psychology/psychiatry at diametrically opposite poles. Both psychoanalysts (Bettelheim, 1973; Harticollis, 1968) and behaviorists (Willis & Lovaas, 1977) promulgated this view, though for very different reasons. The psychoanalysts claimed that parents lacking tolerance for negative or hyperactive temperament in their infants would react with excessively negative, demanding parental responses, giving rise to clinical levels of hyperactivity. The behaviorists stressed poor conditioning of children to stimulus control by commands and instructions that would give rise to noncompliant and hyperactive behavior. Both groups singled out mothers as especially etiologically important in this causal connection, and both could derive some support from studies that found negative mother–child interactions in the preschool years to be associated with the continuation of hyperactivity into the late childhood (Campbell, 1987) and adolescent (Barkley, Fischer, et al., 1990) years.

However, such correlational data cannot prove a cause. They do not prove that poor childrearing or negative parent–child interactions cause hyperactivity; they only show that such factors are associated with its persistence. It could just as easily be that the severity of hyperactivity elicits greater maternal negative reactions, and that this severity is related to persistence of the disorder over time. Supporting this interpretation are the studies of stimulant drug effects on the interactions of mothers and their hyperactive children, which show that mothers' negative and directive behavior is greatly reduced when stimulant medication is used to reduce the hyperactivity in their children (Barkley, 1989b; Barkley & Cunningham, 1979; Barkley, Karlsson, Pollard, & Murphy, 1985; Danforth, Barkley, & Stokes, 1991). Moreover, follow-up studies show that the degree of hyperactivity in childhood is predictive of its own persistence into later childhood and adolescence, apart from its association with maternal behavior (Barkley, Fischer, et al., 1990; Campbell & Ewing, 1990). And given the dramatic hereditary contribution to ADHD, it is also just as likely that the more negative, impulsive, emotional, and inattentive behavior of mothers with their hyperactive children stems in

part from the mothers' own ADHD—a factor that has never been taken into account in the analysis of such data or in interpreting findings in this area. Nevertheless, family context would still prove to be important in predicting the outcome of hyperactive children, even though the mechanism of its action was not yet specified (Weiss & Hechtman, 1986). Parent training in child behavior management, furthermore, would be increasingly recommended as an important therapy in its own right (Dubey & Kaufman, 1978; Pelham, 1977), despite a paucity of studies concerning its actual efficacy at the time (Barkley, 1989c).

The Passage of Public Law 94-142

Another highly significant development was the passage of Public Law 94-142 in 1975, mandating special educational services for children with physical, learning, and behavioral disabilities, in addition to those services already available for mental retardation (see Henker & Whalen, 1980, for a review of the legal precedents leading up to this law). Although many of its recommendations were foreshadowed by Section 504 of the Rehabilitation Act of 1973 (Public Law 93-112), the financial incentives for the states associated with the adoption of Public Law 94-142 probably encouraged its immediate and widespread implementation by them all. Programs for learning disabilities, behavioral–emotional disturbance, language disorders, physical handicaps, and motor disabilities, among others, were now required to be provided to all eligible children in all public schools in the United States.

The full impact of these widely available educational treatment programs for hyperactive children has not yet been completely appreciated, for several reasons. First, hyperactivity, by itself, was overlooked in the initial criteria set forth for behavioral and learning disabilities warranting eligibility for these special classes. Children with such disabilities typically also had to have another condition, such as a learning disability, language delay, or emotional disorder, to receive exceptional educational services. The effects of special educational resources on the outcome of hyperactivity are difficult to assess given this confounding of multiple disorders. It was only after the passage of IDEA in 1990 (and a subsequent 1991 memorandum) that the U.S. Department of Education and its Office of Special Education chose to reinterpret these regulations, thereby allowing children with ADHD to receive special educational services for ADHD per se under the "Other Health Impaired" category of IDEA. Second, the mandated services had been in existence for only a little more than a decade when the long-term outcome studies begun in the late 1970s began to be reported. Those studies (e.g., Barkley, Fischer, et al., 1990) suggested that over 35% of children with ADHD received some type of special educational placement. Although the availability of these services seems to have reduced the percentage of children with ADHD retained in grade for their academic problems, compared to earlier follow-up studies, the rates of school suspensions and expulsions did not decline appreciably from pre-1977 rates. A more careful analysis of the effects of Public Law 94-142, and especially of its more recent reauthorization as the IDEA, is in order before its efficacy for children with ADHD can be judged.

The Rise of Behavior Modification

This growing emphasis on educational intervention for children with behavioral and learning disorders was accompanied by a plethora of research on the use of behavior modification techniques in the management of disruptive classroom behavior, particularly as an alternative to stimulant medication (Allyon, Layman, & Kandel, 1975; O'Leary, Pelham, Rosenbaum, & Price, 1976). Supported in large part by their successful use for children with mental retardation, behavioral technologies were now being extended to myriad childhood disorders—not only as potential treatments of symptoms but also as theoretical statements of their origins. Although the studies demonstrated considerable efficacy of these techniques in the management of inattentive and hyperactive behavior, they were not found to achieve the same degree of behavioral improvement as the stimulants (Gittelman-Klein et al., 1976), and so did not replace them as a treatment of choice. Nevertheless, opinion was growing that the stimulant drugs should never be used as a sole intervention, but should be combined with parent training and behavioral interventions in the classroom to provide the most comprehensive management approach for the disorder.

Developments in Assessment

Another hallmark of this era was the widespread adoption of the parent and teacher rating scales developed by C. Keith Conners (1969) for the assessment of symptoms of hyperactivity, particularly during trials on stimulant medication. For at least 20 years, these simply constructed ratings of behavioral items would be the "gold standard" for rating children's hyperac-

tivity for both research purposes and treatment with medication. The scales would also come to be used for monitoring treatment responses during clinical trials. Large-scale normative data were collected, particularly for the teacher scale, and epidemiological studies throughout the world relied on both scales for assessing the prevalence of hyperactivity in their populations. Their use moved the practice of diagnosis and the assessment of treatment effects from that of clinical impression alone to one in which at least some structured, semi-objective, and quantitative measure of behavioral deviance was employed. These scales would later be criticized for their confounding of hyperactivity with aggression. This confounding called into question whether the findings of research that relied on the scales were the result of oppositional, defiant, and hostile (aggressive) features of the population or of their hyperactivity (Ullmann, Sleator, & Sprague, 1984). Nevertheless, the widespread adoption of these rating scales in this era marks a historical turning point toward the use of quantitative assessment methods that can be empirically tested and assist in determining developmental patterns and deviance from norms.

Also significant during this decade was the effort to study the social-ecological impact of hyperactive–inattentive behavior. This line of research set about evaluating the effects on family interactions produced by a child with hyperactivity. Originally initiated by Campbell (1973, 1975), this line of inquiry dominated my own research over the next decade (Barkley & Cunningham, 1979; Cunningham & Barkley, 1978, 1979; Danforth et al., 1991), particularly evaluations of the effects of stimulant medication on these social exchanges. These studies showed that children with hyperactivity were much less compliant and more oppositional during parent–child exchanges than children without it, and that their mothers were more directive, commanding, and negative than mothers of nonhyperactive children. These difficulties would increase substantially when the situation changed from free-play to task-oriented demands. Studies also demonstrated that stimulant medication resulted in significant improvements in child compliance and decreases in maternal control and directiveness. Simultaneously, Humphries, Kinsbourne, and Swanson (1978) reported similar effects of stimulant medication, all of which suggested that much of parents' controlling and negative behavior toward hyperactive children was the result rather than the cause of the children's poor self-control and inattention. At the same time, Carol Whalen and Barbara Henker at the University of California–Irvine demonstrated similar interaction conflicts between hyperactive children and their teachers and peers, as well as similar effects of stimulant medication on these social interactions (Whalen & Henker, 1980; Whalen, Henker, & Dotemoto, 1980). This line of research would increase substantially in the next decade, and would be expanded by Charles Cunningham and others to include studies of peer interactions and the effects of stimulants on them (Cunningham, Siegel, & Offord, 1985).

A Focus on Psychophysiology

The decade of the 1970s was also noteworthy for a marked increase in the number of research studies on the psychophysiology of hyperactivity in children. There were numerous published studies measuring galvanic skin response, heart rate acceleration and deceleration, various parameters of the EEG, electropupillography, averaged evoked responses, and other aspects of electrophysiology. Many researchers were investigating the evidence for theories of over- or underarousal of the CNS in hyperactivity—theories that grew out of the speculations in the 1950s on cortical overstimulation and the ideas of both Wender (1971) and Douglas (1972; both discussed earlier) regarding abnormal arousal in the disorder. Most of these studies were seriously methodologically flawed, difficult to interpret, and often contradictory in their findings. Two influential reviews at the time (Hastings & Barkley, 1978; Rosenthal & Allen, 1978) were highly critical of most investigations but concluded that if there was any consistency across findings, it might be that hyperactive children showed a sluggish or underreactive electrophysiological response to stimulation. These reviews laid to rest the belief in an overstimulated cerebral cortex as the cause of the symptoms in hyperactivity, but they did little to suggest a specific neurophysiological mechanism for the observed underreactivity. Further advances in the contributions of psychophysiology to understanding hyperactivity would await further refinements in instrumentation and in definition and diagnosis of the disorder, along with advances in computer-assisted analysis of electrophysiological measures.

An Emerging Interest in Adult MBD or Hyperactivity

Finally, the 1970s should be credited with the emergence of clinical and research interests in the existence of MBD or hyperactivity in adult clinical patients. Ini-

tial interest in adult MBD can be traced to the latter part of the 1960s, seemingly arising as a result of two events. The first of these was the publication of several early follow-up studies demonstrating persistence of symptoms of hyperactivity or MBD into adulthood in many cases (Mendelson et al., 1971; Menkes, Rowe, & Menkes, 1967). The second was the publication by Harticollis (1968) of the results of neuropsychological and psychiatric assessments of 15 adolescent and young adult patients (ages 15–25) seen at the Menninger Clinic. The neuropsychological performance of these patients suggested evidence of moderate brain damage. Their behavioral profile suggested many of the symptoms that Still (1902) initially identified in the children he studied, particularly impulsiveness, overactivity, concreteness, mood lability, and proneness to aggressive behavior and depression. Some of the patients appeared to have demonstrated this behavior uniformly since childhood. Using psychoanalytic theory, Harticollis speculated that this condition arose from an early and possibly congenital defect in the ego apparatus, in interaction with busy, action-oriented, successful parents.

The following year, Quitkin and Klein (1969) reported on two behavioral syndromes in adults that might be related to MBD. The authors studied 105 patients at the Hillside Hospital in Glen Oaks, New York, for behavioral signs of "organicity" (brain damage); behavioral syndromes that might be considered neurological "soft signs" of CNS impairment; and any EEG findings, psychological testing results, or aspects of clinical presentation and history that might differentiate these patients from patients with other types of adult psychopathology. From the initial group of 105 patients, the authors selected those having a childhood history that suggested CNS damage, including early hyperactive and impulsive behavior. These subjects were further sorted into three groups based on current behavioral profiles: those having socially awkward and withdrawn behavior (n = 12), those having impulsive and destructive behavior (n = 19), and a "borderline" group that did not fit neatly into these other two groups (n = 11). The results indicated that nearly twice as many of the patients in these three "organic" groups as those in the control group had EEG abnormalities and impairments on psychological testing indicating organicity. Furthermore, early history of hyperactive–impulsive–inattentive behavior was highly predictive of placement in the adult impulsive–destructive group, implying a persistent course of this behavioral pattern from childhood to adulthood. Of the 19 patients in the impulsive–destructive group, 17 had received clinical diagnoses of character disorders (primarily emotionally unstable types), as compared to only five patients in the socially awkward group (who received diagnoses of the schizoid and passive–dependent types).

The results were interpreted as being in conflict with the beliefs widely held at the time that hyperactive–impulsive behavior tends to wane in adolescence. Instead, the authors argued that some of these children continued into young adulthood with this specific behavioral syndrome. Quitkin and Klein (1969) also took issue with Harticollis's (1968) psychoanalytic hypothesis that demanding and perfectionistic childrearing by parents caused or contributed to this syndrome given that their impulsive–destructive patients did not uniformly experience such an upbringing. In keeping with Still's (1902) original belief that family environment could not account for this syndrome, these authors hypothesized "that such parents would intensify the difficulty, but are not necessary to the formation of the impulsive–destructive syndrome" (Quitkin & Klein, 1969, p. 140) and that the "illness shaping role of the psycho-social environment may have been overemphasized by other authors" (p. 141). Treatment with a well-structured set of demands and educational procedures, as well as with phenothiazine medication, was thought to be indicated.

Later in this decade, Morrison and Minkoff (1975) similarly argued that explosive personality disorder or episodic dyscontrol syndrome in adulthood might well be the adult sequel to the hyperactivity syndrome in childhood. They also suggested that antidepressant medications might be useful in their management; this echoed a suggestion made earlier by Huessy (1974) in a letter to the editor of a journal that both antidepressants and stimulants might be the most useful medications for the treatment of adults with hyperkinesis or MBD. But the first truly scientific evaluation of the efficacy of stimulants for adults with MBD must be credited to Wood, Reimherr, Wender, and Johnson (1976), who used a double-blind, placebo-controlled method to assess response to methylphenidate in 11 of 15 adults with MBD, followed by an open trial of pemoline (another stimulant) and the antidepressants imipramine and amitriptyline. The authors found that eight of the 11 individuals tested on methylphenidate had a favorable response, whereas 10 of the 15 individuals tested in the open trial showed a positive response to either the stimulants or the antidepressants. Others in the 1970s and into the 1980s would also make the case for the existence of an adult equivalent of childhood hyperki-

nesis or MBD and the efficacy of using stimulants and antidepressants for its management (Gomez, Janowsky, Zetin, Huey, & Clopton, 1981; Mann & Greenspan, 1976; Packer, 1978; Pontius, 1973; Rybak, 1977; Shelley & Riester, 1972). Yet not until the 1990s would both the lay public and the professional field of adult psychiatry seriously begin to recognize the adult equivalent of childhood ADHD on a more widespread basis and to recommend stimulant or antidepressant treatment in these cases (Spencer et al., 1995; Wender, 1995) and even then the view was not without its critics (Shaffer, 1994).

The work of Pontius (1973) in this decade is historically notable for her proposition that many cases of MBD in adults demonstrating hyperactive and impulsive behavior may arise from frontal lobe and caudate dysfunction. Such dysfunction would lead to "an inability to construct plans of action ahead of the act, to sketch out a goal of action, to keep it in mind for some time (as an overriding idea) and to follow it through in actions under the constructive guidance of such planning" (p. 286). Moreover, if adult MBD arises from dysfunction in this frontal–caudate network, it should also be associated with an inability "to re-program an ongoing activity and to shift within *principles* of action whenever necessary" (p. 286, original emphasis). Pontius went on to show that, indeed, adults with MBD demonstrated deficits indicative of dysfunction in this brain network. Such observations would prove quite prophetic over 20 years later, when research demonstrated reduced size in the prefrontal–caudate network in children with ADHD (Castellanos et al., 1996; Filipek et al., 1997), and when ADHD theorists argued that the neuropsychological deficits associated with it involved the executive functions, such as planning; and the control of behavior by mentally represented information, rule-governed behavior, and response fluency and flexibility; among other deficits (Barkley, 1997a, 1997c).

The Prevailing View by 1979

The 1970s closed with the prevailing view that hyperactivity was not the only or most important behavioral deficit seen in hyperactive children, and that poor attention span and impulse control were equally (if not more) important in explaining their problems. Brain damage was relegated to an extremely minor role as a cause of the disorder, at least in the realm of childhood hyperactivity or MBD; however, other brain mechanisms, such as under-arousal or under-reactivity, brain neurotransmitter deficiencies (Wender, 1971), or neurological immaturity (Kinsbourne, 1977), were viewed as promising. Greater speculation about potential environmental causes or irritants emerged, particularly diet and childrearing. Thus, the most frequently recommended therapies for hyperactivity were not only stimulant medication but also widely available special education programs, classroom behavior modification, dietary management, and parent training in child management skills. A greater appreciation for the effects of hyperactive children on their immediate social ecology, and for the impact of stimulant medication in altering these social conflicts, was beginning to emerge.

However, the sizable discrepancy between North American and European views of the disorder remained: North American professionals continued to recognize the disorder as more common, in need of medication, and more likely to be an attention deficit, while those in Europe continued to view it as uncommon, defined by severe overactivity, and associated with brain damage. Those children in North America diagnosed as having hyperactivity or attention deficits would in Europe likely be diagnosed as having CD and be treated with psychotherapy, family therapy, and parent training in child management. Medication would be disparaged and little used. Nevertheless, the view that attention deficits were as important in the disorder as hyperactivity was beginning to make its way into European taxonomies (e.g., the *International Classification of Diseases*, ninth revision [ICD-9]; World Health Organization, 1978). Finally, in the 1970s there was some recognition that there were adult equivalents of childhood hyperactivity or MBD, that they might be indicative of frontal–caudate dysfunction, and that these cases responded to the same medication treatments suggested earlier for childhood ADHD (the stimulants and antidepressants).

THE PERIOD 1980 TO 1989

The exponential increase in research on hyperactivity characteristic of the 1970s continued unabated into the 1980s, making hyperactivity the most well-studied childhood psychiatric disorder in existence. More books were written, conferences convened, and scientific articles presented during this decade than in any previous historical period. This decade would become known for its emphasis on attempts to develop more specific diagnostic criteria; the differential conceptualization and diagnosis of hyperactivity versus other

psychiatric disorders; and, later in the decade, critical attacks on the notion that an inability to sustain attention was the core behavioral deficit in ADHD.

The Creation of an ADD Syndrome

Marking the beginning of this decade was the publication of DSM-III (American Psychiatric Association, 1980) and its radical reconceptualization (from that in DSM-II) of the hyperkinetic reaction of childhood diagnosis to that of ADD (with or without hyperactivity). The new diagnostic criteria were noteworthy for not only their greater emphasis on inattention and impulsivity as defining features of the disorder but also their creation of much more specific symptom lists, an explicit numerical cutoff score for symptoms, specific guidelines for age of onset and duration of symptoms, and the requirement of exclusion of other childhood psychiatric conditions as better explanations of the presenting symptoms. This was also a radical departure from the ICD-9 criteria set forth by the World Health Organization (1978) in its own taxonomy of child psychiatric disorders, which continued to emphasize pervasive hyperactivity as a hallmark of this disorder.

Even more controversial was the creation of subtypes of ADD, based on the presence or absence of hyperactivity (+ H/– H), in the DSM-III criteria. Little, if any, empirical research on this issue existed at the time these subtypes were formulated. Their creation in the official nomenclature of psychiatric disorders would, by the end of the 1980s, initiate numerous research studies into their existence, validity, and utility, along with a search for other potentially useful ways of subtyping ADD (situational pervasiveness, presence of aggression, stimulant drug response, etc.). Although the findings were at times conflicting, the trend in these studies was that children with ADD – H differed from those with ADD + H in some important domains of current adjustment. Those with ADD – H were characterized as more prone to daydreaming, hypoactive, lethargic, and disabled in academic achievement, but as substantially less aggressive and less rejected by their peers (Barkley, Grodzinsky, & DuPaul, 1992; Carlson, 1986; Goodyear & Hynd, 1992; Lahey & Carlson, 1992). Unfortunately, this research came too late to be considered in the subsequent revision of DSM-III.

In that revision (DSM-III-R; American Psychiatric Association, 1987), only the diagnostic criteria for ADD + H (now renamed ADHD; see "ADD Becomes ADHD," below) were stipulated. ADD – H was no longer officially recognized as a subtype of ADD, but was relegated to a minimally defined category, undifferentiated ADD. This reorganization was associated with an admonition that far more research on the utility of this subtyping approach was necessary before its place in this taxonomy could be identified. Despite the controversy that arose over the demotion of ADD – H in this fashion, it was actually a prudent gesture on the part of the committee asked to formulate these criteria. At the time, the committee (on which I served) had little available research to guide its deliberations in this matter. There was simply no indication whether ADD – H had a similar or qualitatively different type of attention deficit, which would make it a separate childhood psychiatric disorder in its own right. Rather than continue merely to conjecture about the nature of the subtype and how it should be diagnosed, the committee essentially placed the concept in abeyance until more research was available to its successor committee to guide its definition. Notable in the construction of DSM-III-R was its emphasis on the empirical validation of its diagnostic criteria through a field trial, which guided the selection of items for the symptom list and the recommended cutoff score on that list (Spitzer, Davies, & Barkley, 1990).

The Development of Research Diagnostic Criteria

At the same time that the DSM-III criteria for ADD + H and ADD – H were gaining recognition, others attempted to specify research diagnostic criteria (Barkley, 1982; Loney, 1983). My own efforts in this endeavor were motivated by the rather idiosyncratic and highly variable approach to diagnosis being used in clinical practice up to that time, the vague or often unspecified criteria used in published research studies, and the lack of specificity in current theoretical writings on the disorder up to 1980. There was also the more pragmatic consideration that, as a young scientist attempting to select hyperactive children for research studies, I had no operational or consensus-based criteria available for doing so. Therefore, I set forth a more operational definition of hyperactivity, or ADD + H. This definition not only required the usual parent and/or teacher complaints of inattention, impulsivity, and overactivity, but it also stipulated that these symptoms had to (1) be deviant for the child's mental age, as measured by well-standardized child behavior rating scales; (2) be relatively pervasive within the jurisdiction of the major

caregivers in the child's life (parent/home and teacher/school); (3) have developed by 6 years of age; and (4) have lasted at least 12 months (Barkley, 1982).

Concurrently, Loney (1983) and her colleagues had been engaged in a series of historically important studies that would differentiate the symptoms of hyperactivity or ADD + H from those of aggression or conduct problems (Loney, Langhorne, & Paternite, 1978; Loney & Milich, 1982). Following an empirical/statistical approach to developing research diagnostic criteria, Loney demonstrated that a relatively short list of symptoms of hyperactivity could be empirically separated from a similarly short list of aggression symptoms. Empirically derived cutoff scores on these symptom ratings by teachers could create these two semi-independent constructs. These constructs would prove highly useful in accounting for much of the heterogeneity and disagreement across studies. Among other things, it would become well established that many of the negative outcomes of hyperactivity in adolescence and young adulthood were actually due to the presence and degree of aggression coexisting with the hyperactivity. Purely hyperactive children would be shown to display substantial cognitive problems with attention and overactivity, whereas purely aggressive children would not. Previous findings of greater family psychopathology in hyperactive children would also be shown to be primarily a function of the degree of coexisting aggression or CD in the children (August & Stewart, 1983; Lahey et al., 1988). Furthermore, hyperactivity would be found to be associated with signs of developmental and neurological delay or immaturity, whereas aggression was more likely to be associated with environmental disadvantage and family dysfunction (Hinshaw, 1987; Milich & Loney, 1979; Paternite & Loney, 1980; Rutter, 1989; Werry, 1988; Weiss & Hechtman, 1986). The need for future studies to specify clearly the makeup of their samples along these two dimensions was now obvious. And the raging debate as to whether hyperactivity was separate from or merely synonymous with conduct problems would be settled by the important research discovery of the semi-independence of these two behavioral dimensions and their differing correlates (Ross & Ross, 1982). These findings would also lead to the demise of the commonplace use of the Conners's 10-item Hyperactivity Index to select children as hyperactive. It would now be shown that many of these items actually assessed aggression rather than hyperactivity, resulting in samples of children with mixed disorders (Ullmann et al., 1984).

The laudable drive toward greater clarity, specificity, and operational defining of diagnostic criteria would continue throughout this decade. Pressure would now be exerted from experts within the field (Quay, 1988b; Rutter, 1983, 1989; Werry, 1988) to demonstrate that the symptoms of ADHD could distinguish it from other childhood psychiatric disorders—a crucial test for the validity of a diagnostic entity—rather than continuing simply to demonstrate differences from nondisordered populations. The challenge would not be easily met. Eric Taylor (1986) and colleagues in Great Britain made notable advances in further refining the criteria and their measurement along more empirical lines. Taylor's (1989) statistical approach to studying clusters of behavioral disorders resulted in the recommendation that a syndrome of hyperactivity could be valid and distinct from other disorders, particularly conduct problems. This distinction required that the symptoms of hyperactivity and inattention be excessive and handicapping to the children; occur in two of three broadly defined settings (e.g., home, school, and clinic); be objectively measured rather than subjectively rated by parents and teachers; develop before age 6; last at least 6 months; and exclude children with autism, psychosis, anxiety, or affective/mood disorders (depression, mania, etc.).

Efforts to develop research diagnostic criteria for ADHD eventually led to an international symposium on the subject (Sergeant, 1988) and a general consensus that subjects selected for research on ADHD should at least meet the following criteria: (1) reports of problems with activity and attention by adults in at least two of three independent settings (home, school, clinic); (2) endorsement of at least three of four difficulties with activity and three of four with attention; (3) onset before 7 years of age; (4) duration of 2 years; (5) significantly elevated scores on parent–teacher ratings of these ADHD symptoms; and (6) exclusion of autism and psychosis. These proposed criteria were quite similar to others developed earlier in the decade (Barkley, 1982) but provided for greater specificity of symptoms of overactivity and inattention, and a longer duration of symptoms.

Subtyping of ADD

Also important in this era was the attempt to identify useful approaches to subtyping other than those just based on the degree of hyperactivity (+ H/– H) or aggression associated with ADD. A significant though underappreciated line of research by Roscoe Dykman

and Peggy Ackerman at the University of Arkansas distinguished between ADD with and ADD without learning disabilities, particularly reading impairments. Their research (Ackerman, Dykman, & Oglesby, 1983; Dykman, Ackerman, & Holcomb, 1985) and that of others (e.g., McGee, Williams, Moffit, & Anderson, 1989) showed that some of the cognitive deficits (verbal memory, intelligence, etc.) formerly attributed to ADHD were actually more a function of the presence and degree of language/reading difficulties than of ADHD. And although some studies showed that ADHD with reading disabilities is not a distinct subtype of ADHD (Halperin, Gittelman, Klein, & Rudel, 1984), the differential contributions of reading disorders to the cognitive test performance of children with ADHD required that subsequent researchers carefully select subjects with pure ADHD not associated with reading disability. If they did not, then they at least should identify the degree to which reading disorders exist in the sample and partial out the effects of these disorders on the cognitive test results.

Others in this era attempted to distinguish between "pervasive" and "situational" hyperactivity; the former was determined by the presence of hyperactivity at home and school, and the latter referred to hyperactivity in only one of these settings (Schachar, Rutter, & Smith, 1981). It would be shown that children with pervasive hyperactivity were likely to have more severe behavioral symptoms, greater aggression and peer relationship problems, and poor academic achievement. DSM-III-R (American Psychiatric Association, 1987) incorporated this concept into an index of severity of ADHD (see the last portion of Table 2.1). British scientists even viewed pervasiveness as an essential criterion for the diagnosis of a distinct syndrome of hyperactivity (as noted earlier). However, research appearing at the end of the decade (Costello, Loeber, & Stouthamer-Loeber, 1991) demonstrated that such group differences were more likely to be the results of differences in the source of the information used to classify the children (parents vs. teachers) than of actual behavioral differences between the situational and pervasive subgroups. This did not mean that symptom pervasiveness might not be a useful means of subtyping or diagnosing ADHD, but that more objective means of establishing it were needed than just comparing parent and teacher ratings on a questionnaire.

A different and relatively understudied approach to subtyping was created by the presence or absence of significant anxiety or affective disturbance. Several studies demonstrated that children with both ADHD and significant problems with anxiety or affective disturbance were likely to show poor or adverse responses to stimulant medication (Taylor, 1983; Voelker, Lachar, & Gdowski, 1983) and would perhaps respond better to antidepressant medications (Pliszka, 1987). The utility of this latter subtyping approach would be investigated and supported further in the next decade (DuPaul, Barkley, & McMurray, 1994; Tannock, 2000).

ADD Becomes ADHD

Later in the 1980s, in an effort to improve further the criteria for defining this disorder, the DSM was revised (DSM-III-R; American Psychiatric Association, 1987) as noted earlier, resulting in the renaming of the disorder to ADHD. The revisions were significant in several respects. First, a single list of symptoms and a single cutoff score replaced the three separate lists (inattention, impulsivity, and hyperactivity) and cutoff score in DSM-III. Second, the item list was now based more on empirically derived dimensions of child behavior from behavior rating scales, and the items and cutoff score underwent a large field trial to determine their sensitivity, specificity, and power to distinguish ADHD from other psychiatric disorders and from the absence of disorder (Spitzer et al., 1990). Third, the need was stressed that one had to establish the symptoms as developmentally inappropriate for the child's mental age. Fourth, the coexistence of mood disorders with ADHD no longer excluded the diagnosis of ADHD. And, more controversially, the subtype of ADD – H was removed as a subtype and relegated to a vaguely defined category, undifferentiated ADD, which was in need of greater research on its merits. ADHD was now classified with two other behavioral disorders (ODD and CD) in a supraordinate family or category known as the "disruptive behavior disorders," in view of their substantial overlap or comorbidity in clinic-referred populations of children.

ADHD as a Motivation Deficit Disorder

One of the more interesting conceptual developments only began to emerge in the latter half of the decade. This was the nascent and almost heretical view that ADHD was not actually a disorder of attention. Doubt about the central importance of attention to the disorder crept in late in the 1970s, as some researchers more fully plumbed the depths of the attention construct

with objective measures, while others took note of the striking situational variability of the symptoms (Douglas & Peters, 1979; Rosenthal & Allen, 1978; Routh, 1978; Sroufe, 1975). As more rigorous and technical studies of attention in children with ADHD appeared in the 1980s, an increasing number failed to find evidence of problems with attention under some experimental conditions, while observing them under others (for reviews, see Douglas, 1983, 1988; also see Barkley, 1984; Draeger et al., 1986; Sergeant, 1988; Sergeant & van der Meere, 1989; van der Meere & Sergeant, 1988a, 1988b). Moreover, if attention was conceptualized as involving the perception, filtering, and processing of information, no substantial evidence could be found in these studies for any such deficits. These findings, coupled with the realization that both instructional and motivational factors in an experiment played a strong role in determining the presence and degree of ADHD symptoms, led some investigators to hypothesize that deficits in motivation might be a better model for explaining the symptoms seen in ADHD (Glow & Glow, 1979; Rosenthal & Allen, 1978; Sroufe, 1975). Following this line of reasoning, others pursued a behavioral or functional analysis of these symptoms, resulting in hypothesized deficits in the stimulus control over behavior, particularly by rules and instructions. I argued that such deficits arose from neurological factors (Barkley, 1988a), whereas others argued that they arose from poor training of the child by parents (Willis & Lovaas, 1977).

I initially raised the possibility that rule-governed behavior might account for many of the deficits in ADHD but later amended this view to include the strong probability that response to behavioral consequences might also be impaired and could conceivably account for the problems with following rules (Barkley, 1981, 1984, 1990). Others independently advanced the notion that a deficit in responding to behavioral consequences, not attention, might be the difficulty in ADHD (Benninger, 1989; Haenlein & Caul, 1987; Quay, 1988a; Sagvolden, Wultz, Moser, Moser, & Morkrid, 1989; Sergeant, 1988; van der Meere & Sergeant, 1988a). That is, ADHD might arise out of an insensitivity to consequences (reinforcement, punishment, or both). This insensitivity was viewed as being neurological in origin. Yet this idea was not new, having been advanced some 10–20 years earlier by investigators in Australia (Glow & Glow, 1979), by those studying children with conduct problems (see Patterson, 1982, for a review), and by Wender (1971) in his classic text on MBD (discussed earlier). What was original in these more recent ideas is the greater specificity of their hypotheses and increasing evidence supporting them. Others continued to argue against the merits of a Skinnerian or functional analysis of the deficits in ADHD (Douglas, 1989), and for the continued explanatory value of cognitive models of attention in accounting for the deficits in ADHD.

The appeal of the motivational model came from several different sources: (1) its greater explanatory value in accounting for the more recent research findings on situational variability in attention in ADHD; (2) its consistency with neuroanatomical studies suggesting decreased activation of brain reward centers and their cortical–limbic regulating circuits (Lou et al., 1984, 1989); (3) its consistency with studies of the functions of dopamine pathways in regulating locomotor behavior and incentive or operant learning (Benninger, 1989); and (4) its greater prescriptive power in suggesting potential treatments for the ADHD symptoms. Whether or not ADHD would be labeled a motivational deficit, there was little doubt that these new theories based on the construct of motivation required altering the way in which this disorder was to be conceptualized. From here on, any attempts at theory construction would need to incorporate some components and processes dealing with motivation or effort.

Other Developments of the Era

The Increasing Importance of Social Ecology

The 1980s also witnessed considerably greater research into the social-ecological impact of ADHD symptoms on the children, their parents (Barkley, 1989b; Barkley, Karlsson, & Pollard, 1985; Mash & Johnston, 1982), teachers (Whalen, Henker, & Dotemoto, 1980, 1981), siblings (Mash & Johnston, 1983), and peers (Cunningham et al., 1985; Henker & Whalen, 1980). These investigations further explored the effects of stimulant medications on these social systems; they buttressed the conclusion that children with ADHD elicit significant negative, controlling, and hostile or rejecting interactions from others, which can be greatly reduced by stimulant medication. From these studies emerged the view that the disabilities associated with ADHD do not rest solely in a child, but in the interface between the child's capabilities and the environmental demands made within the social-ecological context in which that child must perform (Whalen & Henker, 1980). Changing the attitudes, behaviors, and expecta-

tions of caregivers, as well as the demands they make on children with ADHD in their care, should result in changes in the degree to which such children are disabled by their behavioral deficits.

Theoretical Advances

During this decade, Herbert Quay adopted the neuropsychological model of anxiety by Jeffrey Gray (1982, 1987, 1994) to explain the origin of the poor inhibition evident in ADHD (Quay, 1988a, 1988b, 1997). Gray identified both a behavioral inhibition system (BIS) and a behavioral activation system (BAS) as being critical to understanding emotion. He also stipulated mechanisms for basic nonspecific arousal and for the appraisal of incoming information that must be critical elements of any attempt to model the emotional functions of the brain. According to this theory, signals of reward serve to increase activity in the BAS, thus giving rise to approach behavior and the maintenance of such behavior. Active avoidance and escape from aversive consequences (negative reinforcement) likewise activate this system. Signals of impending punishment (particularly conditioned punishment), as well as frustrative nonreward (an absence of previously predictable reward), increase activity in the BIS. Another system is the fight–flight system, which reacts to unconditioned punitive stimuli.

Quay's use of this model for ADHD indicated that the impulsiveness characterizing the disorder could arise from diminished activity in the brain's BIS. This model predicted that those with ADHD should prove less sensitive to such signals, particularly in passive avoidance paradigms (Quay, 1988b). The theory also specifies predictions that can be used to test and even falsify the model as it applies to ADHD. For instance, Quay (1988a, 1988b) predicted that there should be greater resistance to extinction following periods of continuous reinforcement in those with ADHD, but less resistance when training conditions involve partial reward. They should also demonstrate a decreased ability to inhibit behavior in passive avoidance paradigms in which avoidance of the punishment is achieved through the inhibition of responding. And those with ADHD should also demonstrate diminished inhibition to signals of pain and novelty, as well as to conditioned signals of punishment. Finally, Quay predicted increased rates of responding by those with ADHD under fixed-interval or fixed-ratio schedules of consequences. Some of these predictions were supported by subsequent research; others either remain to be investigated more fully and rigorously, or have not been completely supported by the available evidence (see Milich, Hartung, Martin, & Haigler, 1994; Quay, 1997). Nevertheless, the theory remains a viable one for explaining the origin of the inhibitory deficits in ADHD and continues to deserve further research.

Further Developments in Nature, Etiology, and Course

Another noteworthy development in this decade was the greater sophistication of research designs in the attempt to explore the unique features of ADHD relative to other psychiatric conditions, rather than just in comparison to the absence of disorder. As Rutter (1983, 1989) noted repeatedly, the true test of the validity of a syndrome of ADHD is the ability to differentiate its features from other psychiatric disorders of children, such as mood or anxiety disorders, learning disorders, and particularly CD. Those studies that undertook such comparisons indicated that situational hyperactivity was not consistent in discriminating among psychiatric populations, but that difficulties with attention and pervasive (home and school) hyperactivity were more reliable in doing so and were often associated with patterns of neuropsychological immaturity (Firestone & Martin, 1979; Gittelman, 1988; McGee, Williams, & Silva, 1984a, 1984b; Rutter, 1989; Taylor, 1988; Werry, 1988).

The emerging interest in comparing children with ADD + H to those with ADD – H furthered this line of inquiry by demonstrating relatively unique features of each group in contrast to each other (see Chapters 2 and 17) and to groups of children with learning disabilities and no disability (Barkley, DuPaul, & McMurray, 1990, 1991). Further strengthening the position of ADHD as a psychiatric syndrome is evidence from family aggregation studies that relatives of children with ADHD have a different pattern of psychiatric disturbance than that of children with CD or mixed ADHD and CD (Biederman, Munir, & Knee, 1987; Lahey et al., 1988). Children with pure ADHD were more likely to have relatives with ADHD, academic achievement problems, and dysthymia, whereas those children with CD had a greater prevalence of relatives with CD, antisocial behavior, substance abuse, depression, and marital dysfunction. This finding led to speculation that ADHD has a different etiology than CD. The former was said to arise out of a biologically based disorder of

temperament or a neuropsychological delay; the latter, from inconsistent, coercive, and the dysfunctional childrearing and management frequently associated with parental psychiatric impairment (Hinshaw, 1987; Loeber, 1990; Patterson, 1982, 1986).

Equally elegant research examined potential etiologies of ADHD. Several studies on cerebral blood flow revealed patterns of underactivity in the prefrontal areas of the CNS and their rich connections to the limbic system via the striatum (Lou et al., 1984, 1989). Other studies (Hunt, Cohen, Anderson, & Minderaa, 1988; Rapoport & Zametkin, 1988; Shaywitz, Shaywitz, Cohen, & Young, 1983; Shekim, Glaser, Horwitz, Javaid, & Dylund, 1988; Zametkin & Rapoport, 1986) of brain neurotransmitters provided further evidence that deficiencies in dopamine, norepinephrine, or both, may be involved in explaining these patterns of brain underactivity—patterns arising in precisely those brain areas in which dopamine and norepinephrine are most involved. Drawing these lines of evidence together even further is the fact that these brain areas are critically involved in response inhibition, motivational learning, and response to reinforcement. More rigorous published studies on the hereditary transmission of ADHD (Goodman & Stevenson, 1989) indicated a strong heritability for ADHD symptoms.

Follow-up studies appearing in this decade were also more methodologically sophisticated, and hence more revealing of not only widespread maladjustment in children with ADHD as they reached adolescence and adulthood but also potential mechanisms involved in the differential courses shown within this population (Barkley, Fischer, et al., 1990, 1991; Fischer, Barkley, Edelbrock, & Smallish, 1990; Gittelman et al., 1985; Lambert, 1988; Weiss & Hechtman, 1993). These findings are discussed in Chapter 9. Again, neuropsychological delays, the presence and pervasiveness of early aggression, and mother–child conflict were associated with a different, and more negative, outcome in later childhood and adolescence than was ADHD alone (Campbell, 1987; Paternite & Loney, 1980).

There was also a movement during this decade away from the strict reliance on clinic-referred samples of children with ADHD toward the use of community-derived samples. This change was prompted by the widely acknowledged bias that occurs among clinic-referred samples of children with ADHD as a result of the process of referral itself. It is well known that children who are referred are often more (though not always the most) impaired, have more numerous comorbid conditions, are likely to have associated family difficulties, and are skewed toward those socioeconomic classes that value the utilization of mental health care resources. Such biases can create findings that are not representative of the nature of the disorder in its natural state. For instance, it has been shown that the ratio of boys to girls within clinic-referred samples of children with ADHD may range from 5:1 to 9:1, and that girls with ADHD within these samples are as likely to be as aggressive or oppositional as boys (see Chapter 2). By contrast, in samples of children with ADHD derived from community- or school-based samples, the ratio of boys to girls is only 2.5:1, and girls with ADHD are considerably less likely than boys to be aggressive. For these and other reasons, a greater emphasis on studying epidemiological samples of children, and the rates and nature of ADHD within them (Offord et al., 1987), arose toward the latter half of the 1980s.

Developments in Assessment

The 1980s also witnessed some advances in the tools of assessment, in addition to those for treatment. The Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1983, 1986) emerged as a more comprehensive, more rigorously developed and better-normed alternative to the Conners Rating Scales (Barkley, 1988c). It would become widely adopted in research on child psychopathology in general, not just in ADHD, by the end of the decade. Other rating scales more specific to ADHD were also developed, such as the ADD – H Comprehensive Teacher's Rating Scale (ACTeRS; Ullmann et al., 1984), the Home and School Situations Questionnaires (Barkley & Edelbrock, 1987; DuPaul & Barkley, 1992), the Child Attention Profile (see Barkley, 1988c), and the ADHD Rating Scale (DuPaul, 1991).

Gordon (1983) developed, normed, and commercially marketed a small, portable, computerized device that administered two tests believed to be sensitive to the deficits in ADHD. One was a CPT measuring vigilance and impulsivity, and the other was a direct reinforcement of low rates (DRL) test assessing impulse control. This device became the first commercially available objective means of assessment for children with ADHD. Although the DRL test showed some promise in early research (Gordon, 1979), it was subsequently shown to be insensitive to stimulant medication effects (Barkley, Fischer, Newby, & Breen, 1988) and was eventually deemphasized as useful in the diagnosis in ADHD. The

CPT, by contrast, showed satisfactory discrimination of children with ADHD from nondisabled groups and was sensitive to medication effects (Barkley et al., 1988; Gordon & Mettelman, 1988). Although cautionary statements indicated that more research evidence was needed to evaluate the utility of the instrument (Milich, Pelham, & Hinshaw, 1985), and that its false-negative rate (misses of children with legitimate ADHD) might be greater than that desired in a diagnostic tool, the device and others like it (Conners, 1995; Greenberg & Waldman, 1992) found a wide clinical following by the next decade.

Greater emphasis was also given to developing direct behavioral observation measures of ADHD symptoms that could be taken in the classroom or clinic, and that would be more objective and useful adjuncts to the parent and teacher rating scales in the diagnostic process. Abikoff, Gittelman-Klein, and Klein (1977) and O'Leary (1981) developed classroom observation codes with some promise for discriminating between children with ADHD and those with other or no disabilities (Gittelman, 1988). Roberts (1979), drawing on the earlier work of Routh and Schroeder (1976) and Kalverboer (1988), refined a laboratory playroom observation procedure that discriminated not only between children with ADHD and nondisabled children, but also between children with aggression or mixed aggression and ADHD. This coding system had excellent 2-year stability coefficients. Somewhat later, I streamlined the system (Barkley, 1988b) for more convenient clinical or classroom use and found it to be sensitive to stimulant medication effects (Barkley et al., 1988), to differentiate between children with ADD + H and ADD – H (Barkley, DuPaul, et al., 1991), and to correlate well with parent and teacher ratings of ADHD symptoms (Barkley, 1991). Nevertheless, problems with developing normative data and the practical implementation of such a procedure in busy clinic practices remained hindrances to its widespread adoption.

Developments in Treatment

Developments also continued in the realm of treatments for ADHD. Comparisons of single versus combined treatments were more common during the decade (Barkley, 1989a), as was the use of more sophisticated experimental designs (Hinshaw, Henker, & Whalen, 1984; Pelham, Schnedler, Bologna, & Contreras, 1980) and mixed interventions (Satterfield, Satterfield, & Cantwell, 1981). Several of these historical developments in treatment require mention. The first was the emergence of a new approach to the treatment of ADHD: cognitive-behavioral therapy, or CBT (Camp, 1980; Douglas, 1980a; Kendall & Braswell, 1985; Meichenbaum, 1988). Founded on the work of Russian neuropsychologists (Vygotsky and Luria), North American developmental and cognitive psychologists (Flavell, Beach, & Chinsky, 1966), and early cognitive-behavioral theories (Meichenbaum, 1977), the CBT approach stressed the need to develop self-directed speech in impulsive children to guide their definition of and attention to immediate problem situations, to generate solutions to these problems, and to guide their behavior as the solutions were performed. Self-evaluation, self-correction, and self-directed use of consequences were also viewed as important (Douglas, 1980a, 1980b). Although the first reports of the efficacy of this approach appeared in the late 1960s and the 1970s (Bornstein & Quevillon, 1976; Meichenbaum & Goodman, 1971), it was not until the 1980s that the initial claims of success with nonclinical populations of impulsive children were more fully tested in clinical populations of children with ADHD. The initial results were disappointing (Abikoff, 1987; Gittelman & Abikoff, 1989). Generally, they indicated some degree of improvement in impulsiveness on cognitive laboratory tasks; however, the improvement was insufficient to be detected in teacher or parent ratings of school and home ADHD behaviors, and CBT was certainly not as effective as stimulant medication (Brown, Wynne, & Medenis, 1985). Many continued to see some promise in these techniques (Barkley, 1981, 1989b; Meichenbaum, 1988; Whalen, Henker, & Hinshaw, 1985), particularly when they were implemented in natural environments by important caregivers (parents and teachers); others ended the decade with a challenge to those who persisted in their support of the CBT approach to provide further evidence for its efficacy (Gittelman & Abikoff, 1989). Such evidence would not be forthcoming. Later, even the conceptual basis for the treatment came under attack as being inconsistent with Vygotsky's theory of the internalization of language (Diaz & Berk, 1995).

A second development in treatment was the publication of a specific parent training format for families of children with ADHD and oppositional behavior. A specific set of steps for training parents of children with ADHD in child behavior management skills was developed (Barkley, 1981) and refined (Barkley, 1997b). The approach was founded on a substantial research literature (Barkley, 1997b; Forehand & McMahon,

1981; Patterson, 1982) demonstrating the efficacy of differential attention and time-out procedures for treating oppositional behavior in children—a behavior frequently associated with ADHD. These two procedures were coupled with additional components based on a theoretical formulation of ADHD as a developmental disorder that is typically chronic and associated with decreased rule-governed behavior and an insensitivity to certain consequences, particularly mild or social reinforcement. These components included counseling parents to conceptualize ADHD as a developmentally disabling condition; implementing more powerful home token economies to reinforce behavior, rather than relying on attention alone; using shaping techniques to develop nondisruptive, independent play; and training parents in cognitive-behavioral skills to teach their children during daily management encounters, particularly in managing disruptive behavior in public places (see Chapter 21). Because of the demonstrated impact of parental and family dysfunction on the severity of children's ADHD symptoms, on the children's risk for developing ODD and CD, and on the parents' responsiveness to treatments for the children, clinicians began to pay closer attention to intervention in family systems rather than just in child management skills. Noteworthy among these attempts were the modifications to the previously described parent training program by Charles Cunningham at McMaster University Medical Center (Cunningham, 1990, 2006). Arthur Robin at Wayne State University and the Children's Hospital of Michigan, and Sharon Foster at West Virginia University (Robin & Foster, 1989) also emphasized the need for work on family systems, as well as on problem-solving and communication skills in treating the parent–adolescent conflicts so common in families of teenagers with ADHD (see Chapter 22 for a discussion of this approach).

A similar increase in more sophisticated approaches to the classroom management of children with ADHD occurred in this era (Barkley, Copeland, & Sivage, 1980; Pelham et al., 1980; Pfiffner & O'Leary, 1987; Whalen & Henker, 1980). These developments were based on earlier promising studies in the 1970s of contingency management methods in hyperactive children (Allyon et al., 1975; see Chapter 24 for the details of such an approach). Although these methods may not produce the degree of behavioral change seen with the stimulant medications (Gittelman et al., 1980), they provide a more socially desirable intervention that can be a useful alternative when children have mild ADHD and cannot take stimulants or when their parents decline the prescription. More often, these methods serve as an adjunct to medication therapy to further enhance academic achievement.

The fourth area of treatment development was in social skills training for children with ADHD (see Chapter 23). Hinshaw and colleagues (1984) developed a program for training children with ADHD in anger control techniques. This program demonstrated some initial short-term effectiveness in assisting these children to deal with this common deficit in their social skills and emotional control (Barkley et al., 2000). Related approaches to social skills training for children with ADHD also showed initially promising results (Pfiffner & McBurnett, 1997), but subsequent research did not bear out this promise and suggested that some children with ADHD may even become more aggressive after participation in such group training formats (see Chapter 23).

Finally, medication treatments for children with ADHD expanded to include the use of the tricyclic antidepressants, particularly for those children with characteristics that contraindicated use of a stimulant medication (e.g., Tourette syndrome or other tic disorders) or for those with anxiety/depression (Pliszka, 1987). The work of Joseph Biederman and his colleagues at Massachusetts General Hospital (Biederman, Baldessarini, Wright, Knee, & Harmatz, 1989; Biederman, Gastfriend, & Jellinek, 1986) on the safety and efficacy of tricyclic antidepressants encouraged the rapid adoption of these drugs by many practitioners (see Ryan, 1990), particularly when the stimulants, such as methylphenidate (Ritalin), were receiving such negative publicity in the popular media (see the next section). There simultaneously appeared initially positive research reports on the use of the antihypertensive drug clonidine in the treatment of children with ADHD, particularly those with very high levels of hyperactive–impulsive behavior and aggression (Hunt, Caper, & O'Connell, 1990; Hunt, Minderaa, & Cohen, 1985; see Chapter 27).

Developments in Public Awareness

Several noteworthy developments also occurred in the public forum during this decade. Chief and most constructive among these was the blossoming of numerous parent support associations for families with ADHD. Although less than a handful of these existed in the early 1980s, within 9 years there would be well over

100 such associations throughout the United States alone. By the end of the decade, these would begin to organize into national networks and political action organizations known respectively as CHADD (originally Children with ADD, now Children and Adults with ADHD) and the Attention Deficit Disorder Association (ADDA). With this greater public/parent activism, initiatives were taken to have state and federal laws reevaluated and, it was hoped, changed to include ADHD as an educational disability in need of special educational services in public schools.

When it was passed in 1975, Public Law 94-142 included the concept of MBD under the category of learning disabilities that would be eligible for special educational services. But it did not include hyperactivity, ADD, or ADHD in its description of learning or behavioral disorders eligible for mandated special services in public school. This oversight would lead many public schools to deny access for children with ADD or ADHD to such services, and would cause much parental and teacher exasperation in trying to get educational recognition and assistance for this clearly academically disabling disorder. Other parents would initiate lawsuits against private schools for learning-disabled students for educational malpractice in failing to provide special services for children with ADHD (Skinner, 1988). By the early 1990s, these lobbying efforts would be partially successful in getting the U.S. Department of Education to reinterpret Public Law 94-142—and its 1990 reauthorization as IDEA—to include children with ADHD under the category of "Other Health Impaired" because of their difficulties in alertness and attention. Upon this reinterpretation, children with ADHD could now be considered eligible for special educational services, provided that the ADHD resulted in significant impairment in academic performance. Such efforts to obtain special educational resources for children and adolescents with ADHD stemmed from their tremendous risk for academic underachievement, failure, retention, suspension, and expulsion, not to mention negative social and occupational outcomes (Barkley, Fischer, et al., 1990, 1991; Cantwell & Satterfield, 1978; Weiss & Hechtman, 1986).

The Church of Scientology Campaign

Yet with this increased public activism also came a tremendously destructive trend in the United States, primarily fueled by the Church of Scientology and its Citizens Commission on Human Rights (CCHR). This campaign capitalized on the mass media's general tendency to publish alarming or sensational anecdotes uncritically, as well as the public's gullibility for such anecdotes. Drawing on evidence of an increase in stimulant medication use with schoolchildren, as well as the extant public concern over drug abuse, CCHR members effectively linked these events together to play on the public's general concern about using behavior-modifying drugs with children. In a campaign reminiscent of the gross overstatement seen in the earlier "Reefer Madness" campaign by the U.S. government against marijuana, members of CCHR selectively focused on the rare cases of adverse reactions to stimulants and greatly exaggerated both their number and degree to persuade the public that these reactions were commonplace. They also argued that massive overprescribing was posing a serious threat to schoolchildren, though actual evidence of such overprescribing was never presented. By picketing scientific and public conferences on ADHD, actively distributing leaflets to parents and students in many North American cities, seeking out appearances on many national television talk shows, and placing numerous letters to newspapers decrying the evils of Ritalin and the myth of ADHD (Bass, 1988; CCHR, 1987; Cowart, 1988; Dockx, 1988), CCHR members and others took this propaganda directly to the public. Ritalin, they claimed, was a dangerous and addictive drug often used by intolerant educators and parents and by money-hungry psychiatrists as a chemical straitjacket to subdue normally exuberant children (Clark, 1988; CCHR, 1987; Dockx, 1988). Dramatic, exaggerated, or unfounded claims were made that Ritalin use could frequently result in violence or murder, suicide, Tourette syndrome, permanent brain damage or emotional disturbance, seizures, high blood pressure, confusion, agitation, and depression (CCHR, 1987; Clark, 1988; Dockx, 1988; Laccetti, 1988; "Ritalin Linked," 1988; Toufexis, 1989; Williams, 1988). They also claimed that the increasing production and prescription of Ritalin were leading to increased abuse of such drugs by the general public (Associated Press, 1988; Cowart, 1988; "Rise in Ritalin Use," 1987). Great controversy was said to exist among the scientific and professional practice communities relative to this disorder and the use of medication. No evidence presented in these articles, however, demonstrated a rise in Ritalin abuse or linked it with the increased prescribing of the medication. Moreover, close inspection of professional journals and conferences revealed that no major or widespread controversy

ever existed within the professional or scientific fields over the nature of the disorder or the effectiveness of stimulant medication. Yet lawsuits were threatened, initiated, or assisted by the CCHR against practitioners for medical negligence and malpractice, and against schools for complicity in "pressuring" parents to have their children placed on these medicines (Bass, 1988; Cowart, 1988; Henig, 1988; Twyman, 1988; see also the 1988 segment on ABC's *Nightline*). A major lawsuit ($125 million) was also filed by the CCHR against the American Psychiatric Association for fraud in developing the criteria for ADHD (Henig, 1988; "Psychiatrist Sued," 1987), though the suit would later be dismissed.

So effective was this national campaign by the CCHR, so widespread were newspaper and television stories on adverse Ritalin reactions, and so easily could public sentiment be misled about a disorder and its treatment by a fringe political–religious group and overzealous, scandal-mongering journalists that within 1 year the public attitude toward Ritalin was dramatically altered. Ritalin was seen as a dangerous and overprescribed drug, and the public believed that there was tremendous professional controversy over its use. The minor benefits to come out of this distorted reporting were that some practitioners would become more rigorous in their assessments and more cautious in prescribing medication. Schools also became highly sensitized to the percentage of their enrollment receiving stimulant medication, and in some cases encouraged exploration of alternative behavioral means of managing children.

Yet even the few modestly positive effects of this campaign were greatly outweighed by the damaging effects on parents and children. Many parents were scared into unilaterally discontinuing the medication for their children without consulting their treating physicians. Others rigidly refused to consider the treatment, if recommended, as one part of their child's treatment plan or were harassed into such refusal by well-meaning relatives misled by the distorted church propaganda and media reports. Some adolescents with ADHD began refusing the treatment, even if it had been beneficial to them, after being alarmed by these stories. Some physicians stopped prescribing the medications altogether out of concern for the threats of litigation, thereby depriving many children in their care of the clear benefits of this treatment approach. Most frustrating to watch was the unnecessary anguish created for parents whose children were already on the medication or who were contemplating its use. The psychological damage done to those children whose lives could have been improved by this treatment was incalculable. The meager, poorly organized, and sporadically disseminated response of the mental health professions was primarily defensive in nature (Weiner, 1988) and (as usual) too little, too late to change the tide of public opinion. It would take years even to partially reverse this regression in public opinion toward ADHD and its treatment by medication, and the chilling effect all this had on physicians' prescribing of the medication. Public suspicion and concern over medication use for ADHD remains even today.

The Prevailing View at the End of the 1980s

This decade closed with the professional view of ADHD as a developmentally disabling condition with a generally chronic nature, a strong biological or hereditary predisposition, and a significant negative impact on academic and social outcomes for many children. However, its severity, comorbidity, and outcome were viewed as significantly affected by environmental (particularly familial) factors. Growing doubts about the central role of attention deficits in the disorder arose late in the decade, while increasing interest focused on possible motivational factors or reinforcement mechanisms as the core difficulty in ADHD. Effective treatment was now viewed as requiring multiple methods and professional disciplines working in concert over longer time intervals, with periodic reintervention as required, to improve the long-term prognosis for ADHD. The view that environmental causes were involved in the genesis of the disorder was weakened by increasing evidence for the heritability of the condition and its neuroanatomical localization. Even so, evidence that familial–environmental factors were associated with outcome was further strengthened. Developments in treatment would expand the focus of interventions to parental disturbances and family dysfunction, as well as to the children's anger control and social skills. A potentially effective role for the use of tricyclic antidepressants and antihypertensive medications was also demonstrated, expanding the armamentarium of symptomatic interventions for helping children with ADHD.

Despite these tremendous developments in the scientific and professional fields, the general public became overly sensitized to and excessively alarmed by the increasing use of stimulant medication as a treatment for this disorder. Fortunately, the explosive growth of parent support–political action associations

for ADHD arose almost simultaneously with this public controversy over Ritalin and held the promise of partially counteracting its effects and making the education of children with ADHD a national political priority at the start of the 1990s. These associations also offered the best hope that the general public could be provided with a more accurate depiction of ADHD and its treatment. Perhaps now the public could be made to understand that hyperactive, disruptive child behaviors could arise out of a biologically based disability that could be diminished or amplified by the social environment, rather than being entirely due to bad parenting and diet, as the simplistic yet pervasive societal view maintained.

THE PERIOD 1990 TO 1999

During the 1990s, a number of noteworthy developments occurred in the history of ADHD, chief among them being the increase in research on the neurological and genetic basis of the disorder and on ADHD as it occurs in clinic-referred adults.

Neuroimaging Research

Researchers had long suspected that ADHD was associated in some way with abnormalities or developmental delays in brain functioning. Supporting such an interpretation in the 1990s were numerous neuropsychological studies showing deficits in performance by children with ADHD on tests that were presumed to assess frontal lobe or executive functions (for reviews, see Barkley, 1997a; Barkley et al., 1992; Goodyear & Hynd, 1992). Moreover, psychophysiological research in earlier decades had suggested brain underactivity, particularly in functioning related to the frontal lobes (Hastings & Barkley, 1978; Klorman, 1992). Thus, there was good reason to suspect that delayed or disturbed functioning in the brain, and particularly the frontal lobes, might be involved in this disorder.

In 1990, Alan Zametkin and his colleagues at the National Institute of Mental Health (NIMH) published a landmark study. They evaluated brain metabolic activity in 25 adults with ADHD who had both a childhood history of the disorder and children with the disorder. The authors used positron emission tomography (PET), an exceptionally sensitive technique for detecting states of brain activity and its localization within the cerebral hemispheres. The results of this study indicated significantly reduced brain metabolic activity in adults with ADHD relative to a control group, primarily in frontal and striatal regions. Such results were certainly consistent in many, though not all, respects with the earlier demonstrations of reduced cerebral blood flow in the frontal and striatal regions in children with ADHD (Lou et al., 1984, 1989). Significant in the Zametkin and colleagues (1990) study, however, was its use of a much better defined sample of patients with ADHD and its focus on adults with ADHD. Although later attempts by this research team to replicate their original results with teenagers were consistent with these initial results for girls with ADHD, no differences were found in boys with ADHD (see Ernst, 1996, for a review). Sample sizes in these studies were quite small, however, almost ensuring some difficulties with the reliable demonstration of the original findings. Despite these difficulties, the original report stands out as one of the clearest demonstrations to date of reduced brain activity, particularly in the frontal regions, in ADHD.

At the same time as the NIMH research using PET scans appeared, other researchers were employing magnetic resonance imaging (MRI) to evaluate brain structures in children with ADHD. Hynd, Semrud-Clikeman, Lorys, Novey, and Eliopulos (1990) were the first to use this method, and they focused on total brain volume, as well as specific regions in the anterior and posterior brain sections. Children with ADHD were found to have abnormally smaller anterior cortical regions, especially on the right side, and they lacked the normal right–left frontal asymmetry. In subsequent research that focused on the size of the corpus callosum, this team found that both the anterior and posterior portions were smaller in children with ADHD (Hynd et al., 1991); however, in a later study, only the posterior region was found to be significantly smaller (Semrud-Clikeman et al., 1994). Additional studies were reported by Hynd and colleagues (1993), who found a smaller left caudate region in children with ADHD, and Giedd and colleagues (1994), who found smaller anterior regions of the corpus callosum (rostrum and rostral body).

More recently, two research teams published MRI studies with considerably larger samples of children with ADHD (Castellanos et al., 1994, 1996; Filipek et al., 1997). These studies documented significantly smaller right prefrontal lobe and striatal regions in these children. Castellanos and colleagues (1996) also found smaller right-sided regions of structures in the basal ganglia, such as the striatum, as well as the right

cerebellum. Filipek and colleagues (1997) observed the left striatal region to be smaller than the right. Despite some inconsistencies across these studies, most have implicated the prefrontal–striatal network as being smaller in children with ADHD, with the right prefrontal region being smaller than the left. Such studies have placed on a considerably firmer foundation the view that ADHD does indeed involve impairments in the development of the brain, particularly in the prefrontal–striatal regions, and that these impairments are likely to have originated in embryological development (Castellanos et al., 1996). Advances in neuroimaging technology continue to provide exciting and revealing new developments in the search for the structural differences in the brain that underlie this disorder (see Chapter 14). For instance, the advent of functional MRI (fMRI), with its greater sensitivity for localization of activity, has already resulted in a number of newly initiated investigations into possible impairments in these brain regions in children and adults with ADHD.

Genetic Research

Since the 1970s, studies have indicated that the parents of children with hyperactivity, ADD, or ADHD seem to have a greater frequency of psychiatric disorders, including ADHD. Cantwell (1975) and Morrison and Stewart (1973) both reported higher rates of hyperactivity in the biological parents of hyperactive children than in adoptive parents of such children. Yet both studies were retrospective, and both failed to study the biological parents of the adopted hyperactive children as a comparison group (Pauls, 1991). In the 1990s, a number of studies, particularly those by Biederman and colleagues, clarified and strengthened this evidence of the familial nature of ADHD. Between 10 and 35% of the immediate family members of children with ADHD were found to have the disorder; the risk to siblings of these children was approximately 32% (Biederman, Faraone, & Lapey, 1992; Biederman, Keenan, & Faraone, 1990; Pauls, 1991; Welner, Welner, Stewart, Palkes, & Wish, 1977). Even more striking, research has shown that if a parent has ADHD, the risk to the offspring is 57% (Biederman et al., 1995). Thus, family aggregation studies reveal that ADHD clusters among biological relatives of children or adults with the disorder, strongly implying a hereditary basis to this condition.

At the same time that these studies were appearing, several studies of twins were focusing on the heritability of the dimensions of behavior underlying ADHD (i.e., hyperactive–impulsive and inattentive) behavior, or on the clinical diagnosis of ADHD itself. Large-scale twin studies on this issue have quite consistently found a high heritability for ADHD symptoms or for the clinical diagnosis, with minimal or no contribution made by the shared environment (Edelbrock, Rende, Plomin, & Thompson, 1995; Levy & Hay, 1992). For instance, Gilger, Pennington, and DeFries (1992) found that if one twin was diagnosed with ADHD, the concordance for the disorder was 81% in monozygotic twins and 29% in dizygotic twins. Stevenson (1994) summarized the status of twin studies on symptoms of ADHD by stating that the average heritability is .80 for symptoms of this disorder (range .50–.98). More recent large-scale twin studies are remarkably consistent with this conclusion, demonstrating that the majority of variance (70–90%) in the trait of hyperactivity–impulsivity is due to genetic factors (averaging approximately 80%), and that such a genetic contribution may increase as scores for this trait become more extreme, although this latter point is debatable (Faraone, 1996; Gjone, Stevenson, & Sundet, 1996; Gjone, Stevenson, Sundet, & Eilertsen, 1996; Rhee, Waldman, Hay, & Levy, 1999; Silberg et al., 1996; Thapar, Hervas, & McGuffin, 1995; van den Oord, Verhulst, & Boomsma, 1996). Thus, twin studies added substantially more evidence to that already found in family aggregation studies supporting a strong genetic basis to ADHD and its behavioral symptoms. More recent twin studies have further buttressed the strong genetic contribution to ADHD (see Chapter 14). Equally important is the consistent evidence in such research that whatever environmental contributions may be made to ADHD symptoms fall more within the realm of unique (nonshared) environmental effects than within that of common or shared effects.

Also in this decade, a few researchers began using molecular genetic techniques to analyze DNA taken from children with ADHD and their family members to identify genes that may be associated with the disorder. The initial focus of this research was on the dopamine type 2 gene, given findings of its increased association with alcoholism, Tourette syndrome, and ADHD (Blum, Cull, Braverman, & Comings, 1996; Comings et al., 1991), but others failed to replicate this finding (Gelernter et al., 1991; Kelsoe et al., 1989). More recently, the dopamine transporter gene was implicated in ADHD (Cook et al., 1995; Cook, Stein, & Leventhal, 1997). Another gene related to dopamine,

the *D4RD* (repeater gene) was found to be overrepresented in the seven-repetition form of the gene in children with ADHD (LaHoste et al., 1996). The latter finding has been replicated in a number of additional studies and indicates that the presence of this allele increases the risk for ADHD by 1.5. Clearly, research into the molecular genetics involved in the transmission of ADHD across generations continues to be an exciting and fruitful area of research endeavor. Such research offers promise for the eventual development of not only genetic tests for ADHD and subtyping of ADHD into potentially more homogeneous and useful genotypes but also more specific pharmacological agents for treating ADHD.

ADHD in Adults

Although articles dealing with the adult equivalents of childhood hyperactivity or MBD date back to the late 1960s and 1970s (discussed earlier), they did not initiate widespread acceptance of these adult equivalents in the field of adult psychiatry and clinical psychology. It was not until the 1990s that the professional fields and the general public recognized ADHD in adults as a legitimate disorder. This was due in large part to a best-selling book by Edward Hallowell and John Ratey (1994), *Driven to Distraction*, which brought the disorder to the public's attention. More serious and more rigorous scientific research was also conducted on adults with ADHD across this decade. In addition, the greater clinical professional community began to consider the disorder a legitimate clinical condition worthy of differential diagnosis and treatment (Goldstein, 1997; Nadeau, 1995; Wender, 1995).

This broadening acceptance of ADHD in adults continues to the present time and is likely to increase further in the decades ahead. It seems to have been strengthened in some part throughout the 1990s by the repeated publication of follow-up studies that documented the persistence of the disorder into adolescence in up to 70% of cases, and into adulthood in up to as many as 66% of childhood cases (Barkley, Fischer, et al., 1990, 2002; Mannuzza, Gittelman-Klein, Bessler, Malloy, & LaPadula, 1993; Weiss & Hechtman, 1993). And it can be attributed as well to published studies on clinically referred adults diagnosed with the disorder (Biederman et al., 1993; Murphy & Barkley, 1996; Shekim, Asarnow, Hess, Zaucha, & Wheeler, 1990; Spencer, Biederman, Wilens, & Faraone, 1994). But it is probably in part a result of pressure from the general public, which was made more cognizant of this disorder in adults through various media, including the publication of other best-selling, popular books on the subject (Kelly & Ramundo, 1992; Murphy & LeVert, 1994; Weiss, 1992); numerous media accounts of the condition in adults; the efforts of large-scale parent support groups discussed earlier (e.g., CHADD) to promote greater public awareness of this issue; and the advent of Internet chat rooms, Web pages, and bulletin boards devoted to this topic (Gordon, 1997). Adults who obtain such information and seek out evaluation and treatment for their condition are simply not satisfied any longer with outdated opinions of some adult mental health specialists that the disorder does not exist in adults and is commonly outgrown by adolescence, a belief that was widespread in the 1960s.

Also notable in the 1990s was the publication of more rigorous studies demonstrating the efficacy of stimulants (Spencer et al., 1995) and antidepressants (Wilens et al., 1996) in the management of adult ADHD. Such studies confirmed the initial clinical speculations in the 1970s, as well as the conclusions from earlier, smaller studies by Paul Wender and his colleagues in the 1970s and 1980s (described earlier), that such medications were efficacious for this disorder in adults (Wender, Reimherr, & Wood, 1981; Wender, Reimherr, Wood, & Ward, 1985). Thus, the adult form of ADHD was found not only to share many patterns of symptoms and comorbid disorders with the childhood form, but also to respond just as well to the same medications that proved so useful in the management of childhood ADHD (see Chapter 35).

Other Developments of the Era

The 1990s were marked by other significant developments in the field of ADHD. In 1994, new diagnostic criteria for the disorder set forth in DSM-IV (American Psychiatric Association, 1994) included several improvements over those in the earlier DSM-III-R. But suffice it to say here that they reintroduced criteria for the diagnosis of a purely inattentive form of ADHD, similar to ADD – H in DSM-III. The diagnostic criteria also now require evidence of symptoms' pervasiveness across settings, as well as the demonstration of impairment in a major domain of life functioning (home, school, work). Based on a much larger field trial than any of their predecessors, DSM-IV contained the most empirically based criteria for ADHD in the history of this disorder (see Chapter 2).

A further development during this decade was the NIMH multisite study of ADHD that focused on various combinations of long-term treatments (Arnold et al., 1997; MTA Cooperative Group, 1999; see Chapter 28). This study (the Multimodal Treatment Study of ADHD [MTA]) determined what combinations of treatments were most effective for what subgroups of ADHD, based on those treatment strategies with the greatest empirical support in the prior treatment literature. Another long-term treatment study reported findings of great significance to the field: The Swedish government commissioned the longest treatment study of stimulant medication ever undertaken, the results of which indicated that amphetamine treatment remained effective for the entire 15 months of the investigation (see Gillberg et al., 1997). More sobering was the report that an intensive, yearlong treatment program using primarily CBT strategies produced no substantial treatment effects either at posttreatment or at follow-up (Braswell et al., 1997). Similarly, a yearlong, intensive early intervention program for hyperactive–aggressive children found no significant impact of parent training either at posttreatment or at 2-year follow-up (Barkley et al., 2000; Barkley, Fischer, et al., 2002); the school-based portion of this multimethod program produced some immediate treatment gains, but by 2-year follow-up, these had dissipated (Shelton et al., 2000). Finally, a multisite study of stimulant medication with and without intensive behavioral and psychosocial interventions revealed that the psychosocial interventions added little or nothing to treatment outcome beyond that achieved by stimulant medication alone (Abikoff & Hechtman, 1995). Its final results, not reported until 2004 (see Chapter 28), were in keeping with the findings of the MTA that the combined treatments were generally no substantially better than medication treatment alone. Although these studies do not entirely undermine earlier studies on the effectiveness of behavioral interventions for children with ADHD, they do suggest that some of those interventions produce minimal or no improvement when used on a large-scale basis; that the extent of improvement is difficult to detect when adjunctive stimulant medication is also used; and that treatment effects may not be maintained over time following treatment termination.

The 1990s also witnessed the emergence of trends that would be developed further over the next decade. These trends included a renewed interest in theory development related to ADHD (Barkley, 1997a, 1997c; Quay, 1988a, 1997; Sergeant & van der Meere, 1994), as well as an expanding recognition and treatment of the disorder in countries outside the United States and Canada (Fonseca et al., 1995; Shalev, Hartman, Stavsky, & Sergeant, 1995; Toone & van der Linden, 1997; Vermeersch & Fombonne, 1995). A new stimulant combination, Adderall, which appeared on the market in this decade, showed promise as being as effective for ADHD as the other stimulants (Swanson et al., 1998), and at least three new nonstimulant medications and an additional stimulant were in development or in Phase II clinical trials by several pharmaceutical companies during this decade. There also appeared to be an increasing interest in the use of peers as treatment agents in several new behavioral intervention programs for academic performance and peer conflict in school settings (DuPaul & Henningson, 1993; see Chapters 23 and 24).

The Prevailing View at the End of the 1990s

It seems clear that during the 1990s there was a shift back to viewing ADHD as far more influenced by neurological and genetic factors than by social or environmental ones. Clearly, the interaction of these sources of influence is generally well accepted by professionals at this time, but greater emphasis is now being placed on the former than on the latter in understanding the potential causation of the disorder. Moreover, evidence began accruing that the influence of the environment on the symptoms of the disorder fall chiefly in the realm of unique or nonshared factors rather than among the more frequently considered but now weakly supported common or shared family factors.

Over this decade, there was also a discernible shift toward the recognition that a deficit in behavioral inhibition may be the characteristic that most clearly distinguishes ADHD from other mental and developmental disorders (Barkley, 1997a; Nigg, 2001; Pennington & Ozonoff, 1996; Schachar, Tannock, & Logan, 1993), and that this deficit is associated with a significant disruption in the development of typical self-regulation. It is also noteworthy that the subtype of ADHD comprising chiefly inattention without hyperactive–impulsive behavior may possibly be a qualitatively distinct disorder from the subtype with hyperactive–impulsive behavior or the subtype with combined behavior (Barkley et al., 1992; Goodyear & Hynd, 1992; Lahey & Carlson, 1992). The issue of comorbidity became increasingly important in subgrouping children with ADHD, leading to greater understanding of the way disorders

that coexist with ADHD may influence family functioning, academic success, developmental course and outcome, and even treatment response. In contrast to the attitudes apparent in the middle of the 20th century, the view of ADHD at the close of the century was a less developmentally benign one, owing in large part to multiple follow-up studies that documented the pervasiveness of difficulties with adaptive functioning in the adult lives of many (though by no means all) persons clinically diagnosed with ADHD in childhood.

There is little doubt that the use of pharmacology in the management of the disorder continued its dramatic rise in popularity, owing in no small part to repeated demonstration of the efficacy of stimulants in the treatment of the disorder; the greater recognition of subtypes of ADHD, as well as girls and adults with ADHD; and the rather sobering results of multimethod intensive psychosocial intervention programs. Even so, combinations of medication with psychosocial and educational treatment programs remained the norm in recommendations for the management of the disorder across the 1990s, much as they were in the 1980s.

Across this decade, the expansion, solidification, and increased political activity and power of the patient and family support organizations, such as CHADD, were indeed a marvel to behold. They clearly led to far wider public recognition of the disorder, as well as to controversies over its existence, definition, and treatment with stimulant medications; still, the general trend toward greater public acceptance of ADHD as a developmental disability remained a largely optimistic one. Moreover, such political activity resulted in increased eligibility of those with ADHD for entitlements, under the IDEA, and legal protections, under the Americans with Disabilities Act of 1990 (Public Law 101-336).

THE NEW CENTURY: 2000 TO THE PRESENT

A number of developments arising in this period are covered in detail throughout the remaining chapters in this volume, so they receive only brief topical mention here because of their importance to the history of the disorder. For instance, the recently published DSM-5 diagnostic manual contains a few important adjustments to criteria for the clinical diagnosis of ADHD (American Psychiatric Association, 2013), as discussed in the next chapter. Those adjustments, and others that were recommended but not included, have spawned recent controversy that is also addressed in that chapter.

Trends from the 1990s have certainly continued into the 21st century, with far more published research on heredity, molecular genetics, and neuroimaging, along with some initial efforts to link these fields together. The result has been an explosion in the size of the ADHD literature, which has nearly doubled in 2013 alone, along with the publication of numerous meta-analyses of various segments of it, as referenced in various chapters in this volume. Not only has the hereditary basis of ADHD become firmly established by hundreds of recent studies, but numerous candidate genes for the disorder are also being identified, and new chromosomal regions deserving of greater investigation via scans of the entire human genome that involve hundreds, and soon thousands, of affected families. This area of research has revealed that not only are genes involved in the regulation of dopamine and norepinephrine networks in the brain involved in ADHD but also other, far evolutionary older genes involved in brain cell growth, endpoint termination, and neuronal sprouting may also be implicated (see Chapter 14).

Although no entirely new theories of ADHD have been proposed, existing theories have been expanded and clarified (Barkley, 2012b). There have also been tremendous advances in establishing the underlying neurological nature and mechanisms involved in ADHD in the field of neuroimaging, along with findings from developmental (longitudinal) neuroimaging studies documenting the delayed brain growth and altered growth trajectories associated with the disorder (see Chapter 14). There continues to be abundant research on the neuropsychology of ADHD and potential endophenotypes for use in genetic and neuroimaging investigations, discussed in several chapters in this volume.

Research efforts at subtyping ADHD have also increased since 2000 (see Chapters 2 and 17; also see Milich, Balentine, & Lynam, 2001). This research seems to suggest that the prior DSM-IV subtyping approach to ADHD has not proven useful, and that the disorder is likely to be a single condition varying in severity within the population while having two highly intercorrelated yet partially distinct symptom dimensions. Yet other research is leading to the possibility that perhaps a new attention disorder has been unearthed (Barkley, 2012a, 2012b, 2013). Known as SCT, this subset may have accounted for approximately 30–50% of those children previously placed in the DSM-IV predominantly inattentive type of ADHD, what clinicians began calling ADD. It is characterized

by a cognitive sluggishness and social passivity, in sharp contrast to the distractible, impulsive, overactive, and emotional difficulties so characteristic of the combined type of the disorder. Because of its derogatory and potentially offensive name, and the implication that the underlying cognitive dysfunction is known, I have suggested that the term SCT be renamed "concentration deficit disorder" (CDD; Barkley, 2014).

Although ADHD is a single and dimensional disorder in the human population, advances in molecular genetics may offer the possibility of genetically subtyping samples of individuals with ADHD into those who do and do not possess a particular candidate allele, so as to study over time the impact of the allele on the psychological and social phenotype of the disorder, and its developmental course and risk for future impairments. Such longitudinal studies are now under way.

Further work has also examined those disorders likely to be comorbid with ADHD in both children (see Angold, Costello, & Erkanli, 1999) and adults (Barkley, Murphy, & Fischer, 2008), and the impact they may have on risk for impairments, life course, and even treatment response in ADHD. It now appears, for instance, that the overlap of ADHD with the learning disorders (reading, spelling, math) may stem from both separate, distinct etiologies that arise together in particular cases and some small shared genetic contribution to both disorders, in contrast to the earlier, more simplistic view that one type of disorder may be causing the other. For now, existing evidence suggests that although the two sets of disorders are genetically linked to each other to a small extent, they also have a greater proportion of unshared etiologies. ADHD, however, may be a direct contributor to a progressive increase in problems with reading (and even story and video) comprehension, perhaps through its detrimental effects on working memory. The case for major depressive disorder gives us fairly substantial evidence that ADHD may create a genetic susceptibility to this disorder, albeit one that may require exposure to stress, social disruption, or traumatic events to become fully manifest. And the link of ADHD to ODD and CD, as well as later substance use and antisocial activity, continues to be supported by ongoing research.

The domain of treatment has seen several advances, not the least of which has been the continued reporting of results from the MTA (see Chapter 28), although there is controversy about how initial and especially follow-up results should be interpreted. No one doubts that this monumental study indicated that medication treatment is superior to psychosocial treatment or community care as usual in the initial results. Continuing disagreement appears to concern whether the combination of medication and psychosocial components resulted in important benefits that were not as evident in the medication-only condition. Although many professionals continue to adhere to the view that many cases require combined therapy and that it offers advantages for especially comorbid cases, some certainly concede the point that some cases may do sufficiently well on medications and require little additional psychosocial care.

Another advance in treatment was the development of sustained-release delivery systems for the previously extant stimulant medications (see Chapter 27). These new delivery systems are chemical engineering marvels (sustained-release pellets, osmotic pumps, skin patches, prodrugs, etc.); within the few years of their initial introduction to the marketplace, these extended-release formulations have become the standard of care for medication management, at least in the United States. Such delivery systems allow single doses of medication to manage ADHD symptoms effectively for periods of 8–12 hours. This has eliminated the need for school dosing and its numerous associated problems, not the least of which is stigmatization of children who required midday doses.

This decade also saw U.S. Food and Drug Administration (FDA) approval of two new nonstimulants for treating ADHD. The first of these new medications was the norepinephrine reuptake inhibitor, atomoxetine (Strattera). First approved for use in the United States in January 2003 by the FDA, atomoxetine was the first drug approved for management of ADHD in adults, along with use in children and teens. Over the next several years, the drug received approval for use in numerous other countries and is now prescribed for more than 4 million individuals worldwide. Attractive to many is the fact that this medication has no abuse potential and is therefore not a scheduled drug in the United States, which makes it far easier to prescribe than stimulants, which are Schedule II medications. As one of the most studied medications ever submitted for FDA approval for a neuroscience indication, atomoxetine has become the second-choice medication behind stimulants for management of ADHD in many professional association guidelines for ADHD management.

The second nonstimulant approved by the FDA in the United States was guanfacine XR (Intuniv) in

2009. For more on this medication, see Chapter 27. Guanfacine is an alpha$_2$ agonist that was originally used in the treatment of hypertension. A similar drug, clonidine, had been investigated for use in ADHD over the past 30 years with some success and was even used clinically off-label by some physicians in efforts to better manage the impulsive, hyperactive, and emotionally excitable aspects of ADHD apart from any benefits relative to attention. Some even combined clonidine with stimulants in an attempt to gain greater coverage of ADHD or some symptoms of comorbid disorders associated with anger or other emotion dysregulation. But its significant risks for cardiotoxicity limited its adoption on a more widespread basis, along with its lack of FDA approval for the management of ADHD. In contrast, guanfacine has been shown to present less risk for adverse cardiotoxic events. By reformulating the medication into an extended-release delivery system, guanfacine XR can be taken just once daily, providing treatment coverage of ADHD symptoms across much of the waking day. Like atomoxetine, guanfacine XR does not appear to improve ADHD symptoms as much as the stimulants, but both nonstimulants appear to benefit approximately 75% of individuals taking either medication. They may also be first-choice drugs when ADHD co-occurs with certain other psychiatric disorders or health conditions that might preclude the use of stimulants, or when stimulants may arguably produce some exacerbation, such as anxiety or tic disorders.

Few new psychosocial treatments for ADHD have been identified in nearly a decade since publication of the previous edition of this volume. Research continues to show that various formats of behavioral parent training can help parents manage children and teens with ADHD (see Chapters 21 and 22, this volume), as can training teachers in various behavior management strategies (see Chapter 24). But exciting developments in the alteration or combination of existing treatments may make them more effective for managing various impairments in ADHD. For instance, new CBTs for adults that focus on the executive function deficits that are so impairing in ADHD have been developed and evaluated in randomized trials with considerable success (see Chapter 32). A new variation in social skills training, known as Friendship Coaching, developed by Mikami and colleagues (Chapter 23, this volume) may offer a successful intervention for the social problems of children with ADHD. Prior studies have suggested that social skills training, at least as traditionally delivered in clinics by professionals, has not been especially effective. Mikami's approach uses parents as therapists (friendship coaches) to deliver the appropriate methods throughout the natural stream of social interactions with children in the natural social ecology. Initial promise was evident in the development of cognitive rehabilitation training programs relying on computer software game technology, such as those for working memory training. Yet subsequent efforts to replicate these initially promising findings have shown more limited, if any, positive effects (see Chapter 26). And controversy continues to surround the issue of the effectiveness of EEG biofeedback training (neurofeedback) developed more than 20 years ago (as discussed briefly in Chapter 11), with less rigorous studies showing clinical benefits, while more rigorous ones are less beneficial, if at all.

The international recognition of ADHD has grown sharply since 2000, owing in part to the emergence and expansion of parent support groups in many countries; the dramatic increase in research articles on ADHD in journals, especially from developing or non-Western countries; and the emergence of foreign professional societies dedicated to ADHD, such as Eunythydis in Europe and the World Federation for ADHD, both of which hold annual meetings that comprise numerous presentations on topics related to ADHD. Certainly, educational and advertising efforts by the pharmaceutical industry associated with the increasing number of countries approving the use of these medications has also contributed to greater international recognition of the disorder. But substantial credit must also be given to the increasing access people have to the Internet and the increasing amount of information on ADHD existing there. The Internet allows anyone with a computer, iPad, or smartphone to have nearly instantaneous access to websites such as those sponsored by CHADD (*www.chadd.org*) and ADDA (*www.add.org*) in the United States, the Center for ADD Awareness of Canada (*www.caddac.ca*) and its partner, the Canadian ADHD Resource Alliance (*www.caddra.ca*), among others. It also permits access to numerous videos on YouTube and similar forums.

There was a time when each country had its own view of mental disorders, their causes, and their management. Hence, the United States might view ADHD one way, Sweden in another, and Italy, France, Germany, or Spain might each view it in a different way. Such walls between different countries' understand-

ings of ADHD have now figuratively come crashing down, with the democratizing spread of information via the Internet and the scientific (and nonscientific!) information it can bring to any user. This means that there is no longer an Italian view of ADHD or a U.S. view, but an international view, founded on the most recent scientific advances as they become available on the Internet. Professionals, for instance, who may still practice a psychoanalytic view of childhood disorders as arising from early upbringing, can no longer count on this view going unchallenged by parents of children or adults with ADHD in their practices. These patients and families can readily discover on the Internet that such views have no scientific credibility; that long-term, analytically focused psychotherapy is not effective for ADHD; and that medications and more empirically based psychosocial accommodations are the cutting edge treatments. If they cannot obtain them in their country, they can quickly locate a neighboring one that is better informed and where such therapies may be accessible. We should expect to see more such developments on the international scene in the coming years. But as a consequence, we also continue to expect the same sort of media sensationalism and misrepresentation, baseless social criticism, and even Scientology's propaganda efforts periodically to erupt alongside this expanding international recognition of ADHD as a legitimate mental health and public health disorder.

ADHD has undoubtedly become a valid disorder and frequent topic of scientific study, widely accepted throughout the mental health and medical professions as a legitimate neurodevelopmental disability. At this time, it is unmistakably one of the most well-studied childhood disorders. That it is also the object of healthy, sustained research initiatives into its adult counterparts has led to far greater acceptance of adult ADHD than what occurred two decades earlier for the childhood version of the disorder. Further discoveries concerning the nature, causes, and developmental course of ADHD promise tremendous advances in our insight into not only this disorder but also the very nature and development of human self-regulation more generally, and its rather substantial neurological, genetic, and unique environmental underpinnings. Along with these advances will undoubtedly come new treatments and their combinations. These, let us hope, will greatly limit the impairments experienced by many who suffer from ADHD across their lifespan.

KEY CLINICAL POINTS

- ✓ ADHD has a long and exceptionally rich history of clinical and scientific publications, more than 10,000 since the initial descriptions of clinical patients by Weikard in 1775.
- ✓ Early conceptualizations of ADHD focused on inattention, impulsive behavior, and excessive activity, as well as defective moral control of behavior. Proponents of these views recognized that ADHD-like behavior could arise from brain injuries yet might also develop from flawed social environments. Later views emphasized ADHD's association with brain damage, particularly to the frontal lobes, followed by an emphasis on brain dysfunction, then hyperactivity. Current views of the etiologies of ADHD now emphasize its neurodevelopmental nature and the prominent roles played by genetic, as well as nongenetic, neurological factors.
- ✓ Advances in developing diagnostic criteria have resulted in more precise specification of symptoms, along with two symptom lists; an emphasis on childhood or early-adolescent onset of the disorder in most cases; and a requirement for both cross-setting pervasiveness of symptoms and evidence of impairment in one or more major life activities.
- ✓ More recent theories of ADHD have viewed deficits in self-regulation as central to the disorder, while also suggesting that deficits in executive functioning and biologically based motivational difficulties that undergird self-regulation are likely to account for most or all of the symptoms associated with the disorder.
- ✓ Efforts at subtyping ADHD, such as in the DSM-IV, did not prove successful. But a subset of inattentive children manifesting SCT or CDD, along with social passivity and other distinguishing clinical features, may yet come to be recognized as a second attention disorder that is distinct from yet partially overlaps ADHD.
- ✓ Research using neuroimaging techniques has served to isolate particular brain regions (especially the frontal–striatal–cerebellar network, and possibly other regions) as underlying the disorder, and particularly as involved in the difficulties with inhibition and executive functioning.
- ✓ Increasing research on heredity and genetics has clearly shown a striking hereditary basis to ADHD, along with the identification of numerous candidate

genes or chromosomal locations that hold some promise in explaining the disorder.

- ✓ Research into the neuropsychology of ADHD has also increased substantially in the past decade; it supports the view that ADHD is not only an inhibitory disorder but also one associated with deficits in the major executive functions that underlie self-regulation.
- ✓ Further research, especially on prenatal neurological hazards and postnatal injuries and environmental toxins, suggests that some cases of ADHD may arise from brain injury rather than, or in interaction with, genetic mechanisms.
- ✓ Numerous longitudinal studies now support the conclusion that ADHD is a relatively chronic disorder affecting many domains of major life activities from childhood through adolescence and into adulthood.
- ✓ Within the past decade, new medications and new delivery systems for older medications have been developed that both broaden the range of treatment options for managing the heterogeneity of clinical cases and sustain medication effects for longer periods across the day (with less need for in-school dosing).
- ✓ Advances in psychosocial treatment research have revealed specific subsets of individuals with ADHD who may be more or less likely to benefit from these empirically proven interventions. They have also revealed the limitations of these approaches for generalization and maintenance of treatment effects if they are not specifically programmed into the treatment protocol.
- ✓ ADHD is now recognized as a universal disorder, with an ever-growing international acceptance of both its existence and its status as a chronic disabling condition, for which combinations of medications and psychosocial treatments and accommodations may offer the most effective approach to management.

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