

HOSPITAL PHYSICIAN®

PEDIATRIC MEDICINE BOARD REVIEW MANUAL

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STATEMENT OF EDITORIAL PURPOSE

The *Hospital Physician Pediatric Medicine Board Review Manual* is a study guide for residents and practicing physicians preparing for board examinations in pediatrics. Each quarterly manual reviews a topic essential to the current practice of pediatric medicine.

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The Challenge of Attention-Deficit/Hyperactivity Disorder

Mark L. Wolraich, MD

INTRODUCTION

The condition referred to as attention-deficit/hyperactivity disorder (ADHD) appears to be a relatively new diagnosis. However, the cluster of symptoms associated with ADHD has long been recognized by physicians as a cause of dysfunction in children, although the etiology of the disorder has been somewhat controversial. One of the earliest descriptions of ADHD was made in the mid-nineteenth century by Heinrich Hoffman, a German physician who personified the characteristics of ADHD as 2 characters in a children's book, *Fidgety Phil and Harry Who Looks in the Air*.¹ The first reference to the disorder in the medical literature was made in 1902 by George Still, who described a disease that he characterized as resulting from a defect in moral character.² He noted that the problem resulted in a child's inability to internalize rules and limits and manifested itself in patterns of restless, inattentive, and overaroused behaviors. He suggested that the children had likely experienced brain damage but that the behavior could arise from hereditary and environmental factors.

The connection between brain damage and ADHD was strengthened after the 1917–1918 influenza epidemic when it was observed that the development of encephalitis in some recovering children resulted in symptoms of restlessness, inattention, impulsivity, easy arousability, and hyperactivity.³ By the 1960s, many cases with similar behavioral manifestations but without clear evidence of brain damage were documented, and the name of the disorder was changed to minimal cerebral/brain dysfunction/damage.⁴ Because it was not possible to establish a clear relationship between brain damage and the condition in most cases, the diagnostic label was again revised to more behaviorally descriptive terms, such as hyperkinetic impulse reaction disorder in the psychiatric classification system (DSM-II)⁵ and hyperactive child syndrome in the pediatric literature.⁶ In 1980, thanks to the work of Virginia Douglas and others, the focus shifted from hyperactivity to inattention,⁷ as reflected in the diagnostic label attention deficit dis-

order.⁸ The 3 core behavioral dimensions of inattention, impulsivity, and hyperactivity were defined at this time and remain the core symptoms of the disorder in the current diagnostic criteria.⁹

ETIOLOGY

ADHD is a heterogeneous disorder with 3 subtypes (inattentive, hyperactivity/impulsivity, and combined subtypes) and multiple etiologies that manifest similar behavioral symptoms. The most common cause of ADHD is genetic transmission. The role of genetic factors was demonstrated by a twins study that found a heritability of 0.75 (75% of the variance in the phenotype can be attributed to genetic factors).¹⁰ In addition, family studies have shown that adoptive relatives of children with ADHD are less likely to have the disorder compared with biologic relatives; biologic siblings of children with ADHD have a 2 to 3 times greater risk for being diagnosed with the disorder, and first-degree relatives have a greater risk compared with controls.¹¹ Most recently, several gene associations have been identified in a portion of patients with ADHD. In particular, genes that regulate dopamine have been associated with ADHD, including the dopamine transporter gene (DAT1) and the dopamine D4 receptor gene (DRD4).¹²

Progress has been made in identifying some of the central nervous system characteristics of individuals with ADHD. Studies of brain anatomy have demonstrated that on average individuals with ADHD have smaller prefrontal cortex, basal ganglia, or cerebellar vermix. Functional studies with positron emission tomography, single photon emission computed tomography, and functional magnetic resonance imaging have shown striatal hypoperfusion in individuals with ADHD compared with control individuals. On a neurotransmitter level, it is clear that the behavioral activities associated with ADHD relate to the dopamine and norepinephrine systems in the same areas of the brain identified by anatomical studies.^{13–15} As our ability to understand and measure brain activity at a molecular