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Advances in Understanding and Managing ADHD

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In the short space that I have been provided I would like to highlight several important developments in the understanding and management of attention deficit hyperactivity disorder (ADHD) in children and adults. Since 1902, and certainly since the 1980s, ADHD has been conceptualized as a disorder involving hyperactive behavior, inattention, and poor impulse control. The disorder has been known to affect 3-8% of school-age children. Once thought to be just a childhood disorder outgrown by adolescence, research in the past twenty years has documented persistence of the disorder into

adulthood in at least 50-66% of cases or more, and studies of adult populations have found a prevalence of ADHD of at least 4-5%. Several thousand studies have been published on ADHD, indeed nearly one thousand of them since I published the prior edition of my clinical handbook on ADHD in 1978. I know because I had to read most of them in order to update this volume for its third edition. Among the many developments that have occurred in the past decade in clinical and basic research on this disorder, I have selected three to highlight here. I do so only to highlight the extraordinary vibrancy of research on this disorder and not to elevate these developments over any I may have to ignore given the space constraints for this article.

The first development worth noting has been an evolution in the way we conceptualize the disorder itself. Whereas we once thought of the disorder as largely one of restless and hyper-

active-impulsive behavior combined with inattention, we are broadening this view considerably. Indeed, this old view now seems to be a relatively shallow one capturing only its most obvious yet superficial elements. Our new view stems from numerous studies into the neuropsychology of the disorder along with efforts to develop theoretical models of ADHD. The latter is a sure sign that a field of science is maturing – when it moves into developing theories about an area of science. This view holds that ADHD is a disorder of behavioral inhibition and self-regulation that is associated with deficits in executive functioning that permits self-regulation. Executive functioning and self-regulation are social adaptations in humans that seem to exist to permit social cooperation and the organization of behavior toward social goals to achieve a net long-term maximization of social consequences. If ADHD disrupts these functions, then it creates impairments in the ability to cooperate with others and to organize behavior across time toward the social future. The individual is left stuck in the social “now” and less able to direct behavior toward later consequences or deferred gratification. Seen another way, ADHD is the consummate disorder of time management as the individual with it can not organize his or her behavior relative to time and the social future that likely lies ahead of him or her. There seem to be at least four executive abilities besides behavioral inhibition, each of which is a form of self-control used to guide behavior toward a goal: (1) nonverbal working memory, or sensing-to-the-self, chiefly visual imagery and private audition [rehearing]; (2) verbal working memory, or private speech to the self; (3) emotional self-regulation, or emotion and motivation to the self that creates intrinsic motivation or willpower; and (4) planning and generativity, or private self-directed play, that permits problem-solving and innovation in goal directed behavior. If ADHD disrupts executive functioning, then these are the mental actions with which it is likely interfering and these may

account for many of the symptoms we see in those with the disorder. This broader conceptualization of ADHD has numerous testable predictions as well as implications for its management relative to the old attention deficit perspective of ADHD.

A second development worth highlighting is the advances in understanding of the etiology of ADHD coming out of neurology, specifically neuro-imaging, and genetics. These fields are often considered separate from each other but researchers of ADHD are beginning to combine them to link specific candidate genes associated with ADHD with their neuro-imaging and neuropsychological “signatures” or distinct patterns of structure and function. We have had evidence for more than fifteen years that ADHD is associated with reduced brain volume and especially reduced psychophysiological activity in the frontal lobes, basal ganglia, and cerebellum and that these structures involve a network responsible for behavioral inhibition. A fourth region, the anterior cingulate, may also be involved in ADHD and, along with the dorsolateral aspects of the frontal lobe, may assist with working memory, problem-solving and conflict resolution, and the executive aspects of attention. Research has also shown for more than thirty years that ADHD runs in families, suggesting a strong pattern of inheritance to the disorder. More recent studies involving large samples of twins have repeatedly documented the striking contribution of genetics to this disorder and its associated traits, making it among the three most genetically influenced psychiatric disorders currently known (the others appear to be bipolar disorder and autistic spectrum disorders). On average, 80% or more of individual differences in the traits underlying ADHD are the result of genetic effects, with there being minimal or no evidence of any contribution from shared-within-family influences, while there is a small degree of influence for unique, non-shared events. The latter could easily be the result of biological haz-

ards the individual encounters during development that have deleterious effects on the brain, such as maternal smoking and alcohol use during pregnancy, premature delivery and associated bleeding into the brain, and numerous post-natal hazards such as traumatic brain injuries. Researchers have linked particular candidate gene polymorphisms to differences in patterns of EEG activity and even results of neuropsychological tests in samples of ADHD children. Others have begun to show that response to stimulant medications may be partially determined by which version of these gene variants the individual possesses. Other investigators (including myself) have sorted these samples of ADHD children and adults into groups based on the version of the gene they possess so as to study the psychological phenotype or life course events that may be associated with that particular gene variant within those having the disorder. Such research will eventually permit the subtyping of ADHD, not based on crude behavioral indicators as we do now in the DSM-IV, but on specific genetic variants that will likely reveal differences that are clinically important among these genetic subtypes, possibly including predicting medication response. It is my hope that before my career is over we may have genetic testing to supplement our diagnostic procedures to provide us with a more accurate means for identifying and subtyping those with ADHD.

Yet a third development in the field has been in the area of treatment. While we have seen no new psychological treatments for ADHD in the past twenty years, researchers have examined the combinations of those we have, including combining them with medications to evaluate any added benefits such combinations may provide. Such research suggests that while medications, particularly the stimulants, may be the most effective treatments that we have for the disorder, the combination of medications with behavioral and psychosocial treatments and accommodations are useful for some subsets of

ADHD, depending on the comorbid disorders and demographic factors. One noteworthy development in the area of treatment has been the advent of once-daily delivery systems for the stimulant medications. No new stimulant medications have been identified or approved by the Food and Drug Administration. The original immediate-release versions of the stimulants, such as methylphenidate and the amphetamines, were helpful but problematic because of their short time course, often providing therapeutic benefit from just three to five hours. This resulted in the need for dosing of patients several times per day and especially at mid-day in school. That, of course, was associated with the potential for increased humiliation and stigma of child patients but also increased alarm over schools storing and dispensing Schedule II potentially addictive drugs. Two technologies were developed and eventually FDA approved that have permitted once-daily dosing such that the medication remains in the body for much longer periods than the original drugs. One invention, Concerta, was of a miniature osmotically driven pump that looks like a small capsule but in essence is a device that squeezes out a liquid methylphenidate sludge over a period of eight to twelve hours providing greater management of ADHD symptoms across the day. Another invention, used in Medadate CD and Adderall XR, among others, was a pellet, time-release technology in which small pellets of the drug were coated with varying time-release coatings that dissolved at different times of the day with some dissolving immediately, others in an hour, still others in two hours, and so on. This technology can also provide symptomatic control for eight to twelve hours, thus eliminating mid-day dosing at school. A skin patch for methylphenidate has also been invented that eliminates the need to swallow the medication. Undoubtedly, other ingenious technologies will follow to create a wider selection of medication and delivery system options that can tailor treatment better to the individual needs of patients.

In the area of treatment, we have also witnessed the development and FDA-approval of the first new medication for ADHD in twenty-five years. That medication is atomoxetine, or Strattera, invented by the Eli Lilly Co. Strattera was also the first drug FDA-approved for treatment of adult ADHD besides being used for child and adolescent ADHD. Atomoxetine is not a stimulant. It is a highly selective norepinephrine re-uptake inhibitor that increases the availability of norepinephrine outside the nerve cell. Interestingly, it does have a secondary result of increasing dopamine in the prefrontal cortex, but not in the striatum or nucleus accumbens that undoubtedly accounts for its lack of addiction potential. Research demonstrates significant improvement in ADHD symptoms and related difficulties similar to, though not always identical to the effects seen in the stimulant medications. In particular, the medication may be of use in cases of comorbid anxiety disorders and nervous tics, given that the medication seems to actually treat anxiety and does not exacerbate tics as stimulants may do, though in a minority of cases.

These and many other developments in the field of ADHD demonstrate the exceptional vibrancy, creativity, and productivity in the science of ADHD and the broader domain of clinical psychology and serve to showcase the clinical value of a scientifically grounded approach to the understanding and management of disorders such as ADHD.

Further Reading:

Barkley, R. A. (2006). *Attention Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment (3rd ed.)*. New York: Guilford Publications (www.guilford.com).

Barkley, R. A. & Murphy, K. R. (2006). *Attention Deficit Hyperactivity Disorder: A Clinical Workbook (3rd ed.)*. New York: Guilford Publications.

The ADHD Report. Founding editor: Barkley, R.A. New York: Guilford Publications.

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