ADHD, Memory, and Executive Function

Louise I. Keeton

University of North Texas

Abstract

ADHD is a neurobiological disorder. The anatomy of the brain in patients with ADHD is different from non-ADHD patients. In addition to attention, working memory and executive function are also negatively affected.

ADHD, Memory, and Executive Function

Maybe it should be called Memory-Deficient/Hyperactive Disorder instead of Attention-Deficit/Hyperactivity Disorder (ADHD). Or even Executive Function-Deficient/Hyperactivity Disorder. Strong statements? Perhaps. However, as researchers continue to investigate ADHD, they are learning that memory and executive function problems play a major role in this disorder.

What is ADHD and how is it diagnosed?

ADHD is a neurobiological disorder (CHADD, 2001). ADHD, in this and its previous designations (Minimal Brain Dysfunction, Hyperkinetic Reaction of Children, and Attention-Deficit Disorder With or Without Hyperactivity), has been identified for over a century and occurs in about three to five percent of children (Fine, 2001; CHADD, 2001). In adults, the prevalence is approximately two to four percent (CHADD, 2001). From 10-60% of ADHD cases identified in children persist into adolescence and young adulthood (CHADD, 2001; Seidman, 1998).

Currently, the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (4th ed.) (1994) identifies the following four types of ADHD:

- ADHD, predominantly inattentive type
- ADHD, predominantly hyperactive-impulsive type
- ADHD, combined type, and
- ADHD, not otherwise specified.

The first two types of ADHD are diagnosed by determining if a patient has six or more symptoms from a list of nine for each type. These symptoms must be present for at least six months in two or more different settings, such as school, work, and home. In addition, the symptoms must be present before the patient has reached the age of seven and must show "clear

evidence of clinically significant impairment in social, academic, or occupational functioning" (*DSM-IV*, 1994). The combined type of ADHD is diagnosed by meeting the criteria for both the inattentive and hyperactive/impulsive types of ADHD. The last type of ADHD, not otherwise specified, does not appear in the literature outside the *DSM-IV*.

What causes ADHD?

More is known about the apparent neurobiological causes of ADHD (in all its forms) with every study done. Edward Hallowell, the author of *Driven to Distraction* and himself an ADHD-diagnosed adult, sees himself in terms of having "unique brain biology" (1997). There is a heritable component to ADHD. It is known that for a child with ADHD, there is a "forty percent chance that one of his parents has ADHD" (Kutscher, 2003). Also, there is probably a "transgenerational transmission of core symptom dimensions" revealed through studies of fraternal and identical twins (Vance, 2000). Moreover, fewer girls have ADHD than boys (anywhere from 3:1 to 9:1 cited by Gershon in 2002) and this fact, too, points to a genetic factor to the disorder (Vance, 2000). Work is progressing on identifying the specific genes involved. The "dopamine transporter gene and the dopamine D4 receptor gene," that work in the basal ganglia and prefrontal cortex, have been named as likely "candidate genes" and "have begun to be systematically investigated" (Fine, 2001; Vance, 2000).

The ADHD Brain

The brains of children are anatomically different from non-ADHD children. The brains of ADHD children are approximately eight percent smaller than the brains of normal children (Talan, 1998). Additionally, the brains of ADHD patients are symmetrical in size, but the left side of the brain of non-ADHD patients is normally larger than the right brain. This normal asymmetrical anatomy takes place because of the complex functions related to language centered

in the left side of the brain. The left side of the brain in ADHD patients fails to grow larger than the right brain, thus remaining symmetrical. The left side of the brain is more vulnerable during embryonic development (personal notes from seminar taught by Nikita Katz, spring 2003). Consequently, research focusing on the genetic causes of ADHD may be able to target their studies to this developmental time period in the future.

The front regions of the brain affect attention, among other things (Tripp, 2002; Sergeant 2002). From structural and functional Magnetic Resonance Imaging, "subtle abnormalities in the frontal networks of children with ADHD" have been found and this points to "frontal lobe dysfunction") in children with ADHD (Tripp, 2002). Moreover, Sergeant also concludes that ADHD cannot only be limited to the "frontal lobes but must be extended to both the basal ganglia and cerebellum" (2002). In short, the brains of children with ADHD differ in several ways from non-ADHD children and these differences biologically affect their ability to process information.

Other regions of the brain, including the hippocampus (responsible for formation of the short-term memory stores and the catalog of memory storage) and the corpus callosum (responsible for connecting the two hemispheres of the brain) are also different in ADHD individuals when compared to non-ADHD individuals (personal notes from seminar taught by Nikita Katz, spring 2003; Hallowell, 1997).

Memory Dysfunction

The deficits in attention involved in ADHD have long been documented, hence the name of the disorder. Researchers are now identifying how these attention deficits also profoundly affect memory in ADHD patients, especially in short-term memory (STM) or what is sometimes called working memory (WM) in some memory models. STM falls between the sensory store

and the long-term memory (LTM) store in the traditional Shiffrin/Atkinson memory model (Mealer, 1996). Information in STM decays rapidly and cannot be transferred to LTM unless further processes involving attention and executive function occur (Mealer, 1996). WM is "the process of holding new visual or auditory information in mind as you retrieve older knowledge or procedures to apply to the new material" (Packer, 1998). These memory problems also appear to be physiologic in nature and may also account for some of the underlying causes of the disorder. A fact to note is that "ADHD children generally show more problems on tasks requiring immediate memory [STM or WM] and recall than non-ADHD children" (Mealer, 1996). Barkley also reports that ADHD patients have deficient working memory (1994). Conners, who developed rating scales used extensively by parents and teachers to note symptoms in ADHD children, says, "Without working memory there is only automatic reaction to sensory events and fixed memories of past events which are inflexible in the face of changing reality" (1996). As a result, ADHD patients cannot adapt to their world as easily as non-ADHD patients.

The areas of the brains that are active during memory function are also different in ADHD children from those used in memory function of non-ADHD children. Positron Emission Tomography has shown ADHD patients use parts of the brain associated with visual tasks (the occipital regions) when WM is being used, rather than the prefrontal cortex that non-ADHD patients use (Fine, 2001).

Another problem that might affect memory function in ADHD patients is related to sleep. Neimark says, "Sleep improves memory in humans—and specifically, the sleep associated with dreaming [and] rapid eye movement" (1995). It is during rapid eye movement sleep, it appears, that stimuli are consolidated into LTM. That is, the networks of neurons responsible for memory are reinforced during REM sleep. Sleep disturbance is usually a temporary, but common,

problem for ADHD patients (Kutscher, 2003), especially when first taking the stimulant medication, methylphenidate, which is commonly prescribed for ADHD. As a result, one of the easiest ways to move stimuli through the memory consolidation process (from STM/WM to LTM) is disrupted, at least temporarily.

Executive Dysfunction

In addition to problems with attention and memory, executive function (EF) problems are now being identified in ADHD patients. Executive function is difficult to define. Currently, there are 33 definitions of executive function (Sergeant, 2002). One way to think of EF is as central processing or self monitoring (Packer, 1998). EF encompasses the following:

- Planning for the future
- Inhibiting and delaying response
- Initiating behavior
- Switching between activities flexibly (Packer, 1998)

EF is generally investigated using neuropsychological testing of various kinds. In fact, some of the diagnostic symptom criteria for inattention that are listed in the *DSM-IV* can also be called executive dysfunctions. Dysfunction in these areas manifests prominently in ADHD patients. Murphy has found that "ADHD subjects performed more poorly…on a number of executive control tasks" (2002).

Conclusion

Unfortunately, most children do not outgrow ADHD. In fact, "up to 67 percent of children [diagnosed with ADHD] will continue to experience symptoms of ADHD in adulthood" (CHADD, 2001; Seidman, 1998). Since the anatomy of the brains of ADHD patients is different, that fact will not change as they mature. Because ADHD patients use different parts of their

brains to process information into memory, the resulting problems with memory may also persist as ADHD children grow up. In addition, executive dysfunction is not something that is outgrown. Furthermore, in today's world dysfunctional genes cannot simply be exchanged for functional ones. Thus, ADHD children, as well as the adolescents and adults who will continue to experience problems related to ADHD as they mature, must compensate for the differences in their brain size, brain development level, brain use areas, and genetic predisposition to ADHD and the dysfunction in memory and executive function that result.

References

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Barkley, R. A., Murphy, K. & Kwasnik, D. (1996, April). Psychological adjustment and adaptive impairments in young adults with ADHD. *Journal of Attention Disorders (1)* 1, 41-54.

Children and Adults with Attention-Deficit Hyperactivity Disorder. (2001, July). The Disorder Named AD/HD – CHADD Fact Sheet #1. Retrieved June 13, 2003, from: http://www.chadd.org/fs/fs1.htm

Conners, C. K. (1996, October). Editorial - Attention, memory, and time. *Journal of Attention Disorders (1)* 3, 130-131.

Fine, Lisa. (2001, May 9). Research: Paying Attention. *Ed Week*. Retrieved June 23, 2003, from http://www.edweek.org/ew/newstory.cfm?slug=34adhd.h20

Gershon, J. A meta-analytic review of gender differences in ADHD. *Journal of Attention Disorders (5)* 3, 143-154.

Hallowell, Edward M. (1997, May/June). What I've Learned from ADD. *Psychology Today*. Retrieved June 12, 2003, from: http://www.psychologytoday.com/htdocs/prod/ptoarticle/pto-19970501-00030.asp

Kutscher, M. L. (2003). *ADHD: The Tip of the Iceberg*. [E-book]. Retrieved June 20, 2003, from: http://www.pediatricneurology.com/adhd.htm

Mealer, C., Morgan, S., & Luscomb, R. Cognitive functioning of ADHD and non-ADHD boys on the WISC-III and WRAML: an analysis within a memory model. *Journal of Attention Disorders (1)* 3, 133-145.

Murphy, J. (2002, March). Cognitive functioning in adults with Attention-Deficit Hyperactivity Disorder. *Journal of Attention Disorders* (5) 4, 203-209.

Neimark, Jill. (1995, January/February). It's Magical. It's Malleable. It's…memory… *Psychology Today.* Retrieved June 12, 2003, from: http://www.psychologytoday.com/htdocs/prod/ptoarticle/pto19950101000021.asp

Packer, L. E. (1998). Overview of Executive Dysfunction. Retrieved June 14, 2003, from: http://www.tourettesyndrome.net/ef_overview.htm#FOOTNOTES.

Seidman, L. J., Biederman, J., Weber, Wendy, Hatch, M., & Faraone, S. V. Neuropsychological Function in Adults with Attention-Deficit Hyperactivity Disorder. *Biological Psychiatry (44)*, 260-268.

Sergeant, J. A. Geurts, H., & Oosterlaan, J. (2003). How specific is a deficit of executive functioning for AD/HD? *Behavioral Brain Research 130*, 3-28.

Talan, Jamie. (1998, July/August). The Brain Belongs to Daddy. *Psychology Today*. Retrieved June 12, 2003, from: http://www.psychologytoday.com/htdocs/prod/ptoarticle/pto-19980701- 000013.asp

Tripp, G., Ryan, J., & Peace, K. (2002). Neuropsychological functioning in children with DSM-IV combined typed ADHD. *Australian and New Zealand Journal of Psychiatry 36.* 771-779.

Vance, A. L. A. & Luk, E. S. L. (2000). Attention deficit hyperactivity disorder: current progress and controversies. *Australian and New Zealand Journal of Psychiatry(34)*, 719-730.