Attention Deficit Hyperactivity Disorder

DESCRIPTION

Attention deficit hyperactivity disorder (ADHD) is a developmental disorder that emerges in childhood that sometimes continues through adulthood. ADHD is the most common diagnosed child psychological disorder (Cherkasova & Hechtman, 2009), with an estimated occurrence in 2% to 7% (Hervey, Epstein, & Curry, 2004) of the childhood population, with boys being overrepresented 3:1 (Barkley, 1997). According to the Centers for Disease Control and Prevention (CDC), 4.5 million children were diagnosed with ADHD as of 2006. The frequency of diagnosis has increased 3% per year from 1997 to 2006, again with boys being diagnosed more frequently than girls. However, some individuals are not diagnosed until adulthood. The prevalence of these disorders is not as well understood nor documented.

The *Diagnostic and Statistical Manual for Mental Disorders* (*DSM-IV-TR;* American Psychiatric Association [APA], 2000) notes that the frequent descriptive patterns in individuals with ADHD are inattentiveness, impulsivity, and/or hyperactivity. A person with a deficit in attention may become easily distracted, dissociate from reality, make careless mistakes and/or have difficulty listening, sustaining attention, and organizing tasks. Individuals with impulsivity are characterized by interrupting, acting without regard for consequences, being impatient, and/or having difficulty waiting. People with ADHD demonstrate hyperactivity by fidgeting, talking excessively, demonstrating difficulty while doing quiet tasks and/or activities, and having trouble staying still. Based on predominant symptoms, the *DSM-IV-TR* (APA, 1994) divided ADHD into three subtypes: predominantly inattentive type, predominantly hyperactive-impulsive type, and the combined type.

The prevalence of these disorders has increased exponentially. In 1981, almost no articles were written on ADHD according to Lopez-Munoz, Alamo, Quintero Gutierrez, and Garcia (2008). By 2005, over 850 articles were being published each year. The top five journals where publications on ADHD were found were psychiatric ones. Interestingly, none of the top 20 journals were neuropsychological, though over 40% were from U.S. journals.

NEUROPATHOLOGY/PATHOPHYSIOLOGY

The cognitive deficits associated with ADHD can be attributed to dysfunction in the frontostriatal circuitry, along with regions outside of this area including the cerebellum and the temporoparietal lobes (Cherkasova & Hechtman, 2009; Schmahmann, 2010; Vaidya & Stollstorff, 2008). These areas interconnect to mediate aspects of attention (Arnsten, 2009). The cognitive deficits extend beyond executive functioning to include spatial, temporal, and lower-level “nonexecutive” functions. In addition

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to differences in selective regions, there are global brain differences in ADHD (Vaidya & Stollstorff, 2008). The pathophysiology of ADHD has also been shown to involve dysfunction of the catecholaminergic neurotransmitters, dopamine (DA) and norepinephrine (NE), especially in the prefrontal cortex (PFC) (Arnsten, 2009; Vaidya & Stollstorff, 2008).

The frontostriatal circuitry, including the dorsolateral prefrontal cortex, ventrolateral prefrontal cortex, dorsal anterior cingulate cortex, as well as the caudate nucleus and putamen, is the region of brain circuitry that has attracted the most attention from neuroimaging researchers in ADHD (Cherkasova & Hechtman, 2009). This circuitry regulates attention based on relevance (i.e., top-down attention) and mediates task-relevant response selection both with and without affective value (Arnsten, 2009; Vaidya & Stollstorff, 2008). Much research has shown evidence suggesting structural abnormalities in the frontal lobes and striatal structures of people with ADHD (Arnsten, 2009; Cherkasova & Hechtman, 2009; Vaidya & Stollstorff, 2008). Such studies have reported prefrontal volume and cortical thickness reductions as well in the caudate nucleus and pallidum, significant regional gray matter reduction, and hypoactivation of the network as a whole (Cherkasova & Hechtman, 2009; Vaidya & Stollstorff, 2008). The right hemisphere of the PFC is especially important for “top-down” attentional processes and for impulse control. Imaging studies have shown reduced size and functional activity of the right PFC in patients with ADHD (Arnsten, 2009). Research involving the use of stimulant medications resulted in heightened activity in these areas after usage (Cherkasova & Hechtman, 2009).

Recent evidence has implicated that the cerebellum has many cognitive and affective functions and demonstrates cerebellar-cortical connections with regions involved in higher order cognitive operations including those of the cerebellum with the PFC (Cherkasova & Hechtman, 2009). The cerebellum is involved in several executive and nonexecutive functions affected in ADHD. Studies have documented structural and functional abnormalities of the cerebellum, including reduced volumes and cortical thinning, as well as decreased activity during certain cognitive tasks (Cherkasova & Hechtman, 2009; Vaidya & Stollstorff, 2008). Cerebellar input into prefrontal regions that are involved in ADHD related functions are also suggested to be weakened or impaired (Cherkasova & Hechtman, 2009).

Parietal regions are relevant to attentional functioning, and executive functions are thought to be subserved by a network of regions including parietal structures. ADHD-related dysfunction may be found in many of the parietal lobe's functional units (Cherkasova & Hechtman, 2009). Neuroimaging studies have shown structural and functional abnormalities in the parietal lobes of people with ADHD (Arnsten, 2009; Cherkasova & Hechtman, 2009; Vaidya & Stollstorff, 2008). Overall parietal volume reductions have been noted, as well as reductions in gray matter, white matter, and cortical thickness (Cherkasova & Hechtman, 2009). Cherkasova and Hechtman (2009) and Vaidya and Stollstorff (2008) report reduced activity in many parietal regions, especially in the right hemisphere. This altered parietal activity in ADHD has also been seen in the context of numerous cognitive functions. Conversely, there is some evidence of compensatory recruitment of parietal structures in ADHD in the face of frontal-anterior cingulated deficits (Cherkasova & Hechtman, 2009).

The temporal lobe may be of interest in ADHD research because of its role in the auditory processing of linguistic information, as linguistic skill impairments have been reported in ADHD (Cherkasova & Hechtman, 2009). Several structural imaging studies have documented decreases in temporal lobe total volume as well as decreases in gray matter volume, and cortical thickness (Cherkasova & Hechtman, 2009; Vaidya & Stollstorff, 2008). These structural abnormalities are thought to be attributed to the potential delayed maturation of temporal lobes in children with ADHD (Cherkasova & Hechtman, 2009). Although there is some evidence of temporal lobe abnormalities in ADHD, the evidence is not as strong as that of cerebellar or parietal dysfunction (Vaidya & Stollstorff, 2008).

The catecholamines DA and NE have recently been recognized as playing a role in the pathophysiology of ADHD. In 2009, Arnsten stated that “DA and NE are so critical to PFC function that depleting them is as detrimental as removing the cortex itself. Small changes in these catecholamines can have marked effects on PFC function” (p. 36). The dopaminergic and norepinephrinergic systems modulate and determine the balance between sensory/reactive and control processes (Vaidya & Stollstorff, 2008). Neuroreceptor imaging has noted weaker transmission of DA in the striatum, PFC, and limbic structures, which likely reflects global reductions in DA release throughout the brain (Arnsten, 2009; Vaidya & Stollstorff, 2009). Weaker production of NE has also been seen in the PFC of people with ADHD (Arnsten, 2009).

NEUROPSYCHOLOGICAL/CLINICAL PRESENTATION

Deficits in executive functions such as working memory, planning, and inhibitory control have been reported to be neuropsychological factors that contribute to ADHD symptoms (Wahlstedt, 2009). In 1997, Barkley stated that executive functions shift control from one to another context by internal representations regarding possible outcomes. Among many factors, executive function involves the acts of planning, decision making, implementing strategies, and working memory (Wahlstedt, 2009). More specifically, inhibitory control has been reported to be a fundamental aspect of the later emergence of other domains in executive function (Wiersema, van der Meere, Antrop, & Roeyers, 2006). Other neuropsychological factors such as temporal information processing and reinforcement sensitivity have been associated with individuals with ADHD (Luman et al., 2009). Interestingly, other possible neuropsychological deficits may also be playing a role as well (Wahlstedt, 2009).

Research studies have shown that several neuropsychological deficits have been widely reported in adults with ADHD. In a meta-analysis conducted by Hervey et al. (2004), neuropsychological deficits in attention, response inhibition, memory, processing speed, motor speed, and overall intelligence were widely noted among an adult sample with ADHD, with attention and response inhibition being the most notably affected domains. Although individuals generally report these deficits, neuropsychological tests such as Conners' Continuous Performance Test, Wechsler Adult Intelligence Scale, Test of Auditory Discrimination (TOAD), and Rey-Osterrieth are commonly used to address these deficits. It was noted that adults with ADHD performed worse in the following domains of function: behavioral inhibition, attention, and memory. Overall, some of the

general findings in the meta-analysis showed that adults with ADHD performed poorly compared with the adults in the control group when there was a verbal presentation of stimuli rather than a visual presentation and when the test required higher cognitive demands or increased complexity (Hervey et al., 2004).

Other studies have aimed to look at the neuropsychological factors associated in children with ADHD. Barkley (1997) explains that the behavioral deficits associated with ADHD (i.e., poor sustained attention, impulsiveness, and hyperactivity) arise relatively early, typically before the age of seven, and are fairly persistent over development. In a study conducted by Wahlstedt (2009), neuropsychological factors such as delay aversion, inhibitory control, and reaction time were investigated to see if they played significant roles in ADHD. Results showed that (1) inhibitory control and reaction time variability were mainly associated with symptoms of inattention and that (2) significant interaction effects of delay aversion and reaction time were associated with inattention and hyperactivity/impulsivity symptoms. In a study that looked at children with ADHD, children with ADHD+ODD (Oppositional Defiant Disorder), and children without ADHD, Luman et al. (2009) found that inhibition problems were only seen among those children diagnosed with ADHD. It was also noted that children with ADHD only showed neurocognitive deficits in (1) temporal information: children were more likely to show time underestimations and deficits (2) reinforcement sensitivity: children were more likely to profit less from penalty (Luman et al., 2009).

Overall, there seems to be numerous studies indicating that neuropsychological deficits such as response inhibition, delay aversion, temporal information, reinforcement sensitivity, as well as neuropsychological deficits seem to be significant in individuals with ADHD.

DIAGNOSIS

The *Diagnostic and Statistical Manual for Mental Disorders* (*DSM-IV-TR;* APA, 2000) provides the widely accepted criteria for diagnosis of ADHD. Patients must present with six or more of the following symptoms of inattention, which have been present for at least 6 months to a point that is disruptive and inappropriate for the developmental level:

> *Often does not give close attention to details or makes careless mistakes in schoolwork, work, or other activities; often has trouble keeping attention on tasks or play activities; often does not seem to listen when spoken to directly; often does not follow instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behavior or failure to understand instructions); often has trouble organizing activities; often avoids, dislikes, or does not want to do things that take a lot of mental effort for a long period of time (such as schoolwork or homework); often loses things needed for tasks and activities; is often easily distracted; is often forgetful in daily activities*

If the patient does not present with symptoms of inattention, the individual must present six or more of the following symptoms of hyperactivity-impulsivity that have been present for at least 6 months to an extent that is disruptive and inappropriate for developmental level:

> *Often fidgets with hands or feet or squirms in seat; often gets up from seat when remaining in seat is expected; often runs about or climbs when and where it is not appropriate (adolescents or adults may feel very restless); often has trouble playing or enjoying leisure activities quietly; is often "on the go" or often acts as if "driven by a motor"; often talks excessively; often blurts out answers before questions have been finished; often has trouble waiting one's turn; often interrupts or intrudes on others (e.g., butts into conversations or games)*

The symptoms presented that cause impairment must be seen before the age of 7, and must be displayed in two or more settings (i.e., home, school, work, etc.); however, it is not uncommon for impairments to present with different severity across settings. For example, the more sedentary and continuous a situation the more likely symptoms will be noticeable. There must be clear evidence of impairment in social, school, and work functioning. These symptoms must not happen during a psychotic episode, and they must not be better accounted for by another mental disorder.

The World Health Organization (WHO) has determined ADHD diagnosis occurs when an individual presents several of the following symptoms and if these symptoms have been prominently seen for several months:

> *Being fidgety, restless, and hyperactive most of the time; having poor concentration in activities, leaving tasks unfinished, and frequently shifting from one activity to another; impulsive behavior such as often interrupting others or doing dangerous things; being distracted from activities by minor events and happenings; easy excitability, overtalkativeness, and aggressive behavior*

The diagnosis of ADHD is dependent upon documenting a childhood history of the disorder, along with a document of the patient's early adult history and current functioning (Resnick, 2000; Weiss, Hechtman, & Weiss, 1999). ADHD is associated with executive dysfunction; therefore, the task of describing one's childhood presents a challenge that may be more revealing of deficits in these areas than any other aspect of the assessment (Holmes et al., 2010; Weiss et al., 1999). Triolo (1999) states that it is important to have the patient undergo a routine medical exam in order to rule out any medical problems that may mimic the symptoms of ADHD. The medical assessment should also include the patient's documented medical history

(Newill, Goyette, & Fogarty, 1984). Comorbidity of ADHD with other psychiatric disorders is as high as 77% (Biederman et al., 1993), and many of these other disorders are associated with attention deficits in their own right. It is essential to do a complete screen for other psychiatric disorders (Weiss et al., 1999).

Cognitive measures of executive function may be used to help identify those with ADHD (Holmes et al., 2010). By using psychological testing, information such as the patient's intellectual potential, academic achievement, and possible comorbid learning disabilities, will be provided. The components of such testing will vary according to what areas the psychologist feels may be impaired (Weiss et al., 1999). It is possible to enhance clinical diagnosis of ADHD by employing neuropsychological/cognitive tests of executive functioning, brief tests of response inhibition, and working memory. Such neuropsychological testing can provide high levels of discrimination between individuals with and without ADHD (Holmes et al., 2010).

TREATMENT

Although medication is the main approach to treating patients with ADHD, education, psychotherapy, and environmental modification are also used as treatments. Educating patients with ADHD about their condition should include the latest information available regarding each patient's diagnosis, treatment, prognosis, and outcome (Resnick, 2000; Wender, 1995).

The drug treatment of ADHD is of both practical and theoretical importance. The use of medications must always be carefully considered (Resnick, 2000; Wender, 1995). Stimulant medications are the best-researched and most effective drugs available to specifically target symptoms related to ADHD. Interestingly, methylphenidate was prescribed three times more frequently than any other medication for the treatment of ADHD (Lopez Munoz et al., 2008). Medication for ADHD normalizes the under-arousal of the frontal lobe and permits some increased inhibition of the problem behavior (Resnick, 2000). However, Weiss et al. (1999) explain that if these drugs are ineffective or give rise to more serious somatic or psychiatric side effects, second-line drugs should be considered. Generally speaking, in adults as in children, stimulants are the first line of pharmacological treatment. Although 70% to 80% of school-aged children will respond positively to stimulant medication, the percentage drops somewhat to between 50% and 78% in adults (Wilens, Biederman, & Spencer, 1998). Antidepressants are effective and may be the pharmacological treatment of choice when there is a comorbid finding of depression or anhedonia, when stimulants are ineffective, or when there are unpleasant side effects from stimulants (Resnick, 2000). In Wender's experience (1995), drug treatment frequently produces a near complete remission of symptoms. Wender's drug-responsive ADHD patients often claim that they function better than they ever have in their lives.

Psychotherapy helps patients deal with personal, family, interpersonal, and work problems that tend to develop because of ADHD-related behaviors (Resnick, 2000). A significant part of the impairment from ADHD comes from its secondary impact on self-esteem, self-comfort, quality of life, hopefulness, and family burden. Psychotherapy is the treatment of choice to address these secondary effects of psychosocial disability (Weiss et al., 1999). Often, the amount of psychotherapy needed will be brief but intermittent. Common types of psychotherapy generally used include individual, focused psychoeducational, marital, and group therapies (Resnick, 2000). For some patients improvement in actual functioning is accentuated when pharmacotherapy of core symptoms is combined with psychotherapy. Given that adults with ADHD experience considerable psychosocial impairment and given that these patterns are embedded in lifetime histories of dysfunction, addressing a form of psychological therapy as a therapeutic option is crucial (Weiss et al., 1999).

Research by Resnick (2000) suggests that an important component of the treatment plan for adults with ADHD will necessarily focus on a review of needed environmental changes and adjustments that can reduce the expression of symptoms and their impact on the individual. In order to do this, the patient must understand what constitutes an optimal environment and lifestyle for individuals with ADHD. Practitioners often help identify behavioral problem areas, and restrict their current environment and lifestyle to minimize and control ADHD symptoms.

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