

Growing into successful adults with adhd



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Attention-deficit/hyperactivity disorder (ADHD), one of the most commonly diagnosed neurobiological and behavioral disorders in children, continues through adolescence and adulthood. ADHD affects 3 to 5% of school-aged children in the United States. This means that in a classroom of 20 to 30 children, at least one will have the disorder. ADHD can be a lifelong disorder displaying four primary symptoms: inattention, hyperactivity, stubbornness, and impulsiveness. Usually, the first symptoms of ADHD that appear are impulsiveness and hyperactivity. Inattention may not appear for a year or more after the other two symptoms. When any of these symptoms in combination begin to affect school performance, parents or teachers may begin to suspect ADHD (Meyers, 2010).

Hyperactive children are described by others as “ constantly on the go.” They run around touching everything they see and talk excessively. They may fidget and squirm in their seats, have trouble sitting still during dinner, school and story time, be constantly in motion, and have difficulty doing quiet tasks or activities (NIMH, 2009). Daily tasks may become unattainable with the symptom of impulsivity.

Children who are impulsive may say inappropriate things or wear their emotions on their sleeves because they cannot think before speaking. Many ADHD children find that waiting for what they want is hard to do. They often lose friends, even as toddlers, because of grabbing toys and hitting other children. Older children sometimes get into trouble because they do not think about the consequences of their actions (Meyers, 2010; NIMH, 2009).

Children with symptoms of inattention can be easily distracted, miss details, forget things, frequently switch from one activity to another, and have difficulty focusing on one thing. They become bored with a task after only a few minutes, have difficulty organizing and completing a task such as homework assignments or learning something new. Also, they may lose things needed to complete tasks or activities, do not seem to listen when spoken to, daydream, become easily confused, and move slowly. Lastly, they may have difficulty processing information as quickly and accurately as others, and struggle to follow instructions (NIMH, 2009).

Many parents who have children with ADHD have difficulty understanding that their children can concentrate and focus extremely well as long as they are doing something they enjoy. When the task is difficult or involves something they do not find interesting, it is hard for the child to pay attention. “ Children who are inattentive but not hyperactive or impulsive may never be diagnosed and go through their lives labeled as lazy, spacey, or daydreamers” (Meyers, 2010, p. 14).

Obviously, not everyone who is active, inattentive, or impulsive has ADHD; however, according to the Diagnostic and Statistical Manual of Mental Disorders-IV-TR (DSM-IV-TR) (2000),

The diagnosis of ADHD requires that the behaviors exist to a degree that is inappropriate for the child’s age. The behaviors must appear before the age of seven, continue for at least six months, and interfere substantially in two areas of the child’s life, such as home and classroom (p. 65).

Professionals who are qualified to diagnose ADHD include child psychologists, psychiatrists, developmental pediatricians, and behavioral neurologists.

The doctor usually begins his investigation by gathering information that can rule out other reasons for the child's behavior. Some conditions can cause behavior that mimics ADHD, including traumatic events that have taken place suddenly, such as a death in the family. " Seizure activity; middle ear infections, that are severe enough to cause hearing problems; medical disorders that affect the brain; some learning disabilities; and anxiety or depression" can all be linked to ADHD behavior (Meyers, 2010, p. 17).

Doctors will also check medical records and try to get a sense of what the child's home life is like. Several interviews may be conducted to form part of the diagnosis by gathering information from parents, teachers, and the child. A series of tests may be used to evaluate the child's intelligence and learning achievement. Finally, the doctor will compare all the evidence and criteria specified by the DSM and form the diagnosis of ADHD (Meyers, 2010).

According to The National Institute of Mental Health (2009), ADHD has three subtypes: Predominantly Hyperactive-Impulsive, Predominantly Inattentive, and Combined Hyperactive-Impulsive and Inattentive. " ADHD can be mistaken for other problems because parents and teachers can miss the fact that children with symptoms of inattention have the disorder because they are often quiet and less likely to act out. They may sit quietly, seeming to work, but they are often not paying attention to what they are doing. They may get along well with other children, compared with those with the other

subtypes, who tend to have social problems. But children with the inattentive kind of ADHD are not the only ones whose disorders can be missed. For example, adults may think that children with the hyperactive and impulsive subtypes just have emotional or disciplinary problems”(NIMH, 2009).

People with ADHD are constantly criticized through much of their lives. “ They are not spoiled, lazy, self-indulgent, inconsiderate people. They are individuals with a condition that they cannot control” (Meyers, 2010, p. 11). While there is no cure for ADHD, treatments can relieve many of the disorder’s symptoms. This will allow the ADHD child/adult to be successful in school and lead productive lives.

Historical Perspective

Heinrich Hoffman, a physician, in 1863 was one of the first to observe the symptoms of ADHD and wrote a poem entitled “ Fidgety Phil” (Buttross, 2007). ADHD is the current diagnostic label used for children manifesting symptoms of inattention, impulsiveness, and excessive activity, but it hasn’t always been so (Barkley, 2006). Some of the names ADHD has been known by include encephalitis lethargica, minimal brain damage, minimal cerebral palsy, mild retardation, minimal brain dysfunction, hyperkinesis, and atypical ego development (Rafalovich, 2004).

Rafalovich, a sociologist, frames ADHD from a uniquely sociological perspective. He examines how social forces have impacted historical and contemporary discourses on the disorder of ADHD. Rafalovich helps us see how social forces bear on medical problems, and he gives us a distinctive view of how deviant behavior became medicalized. The terms idiocy and

imbecility are examples. In the late 1870's, both words represented someone who was socially a misfit, but the medical literature of the day sought to confine and define both words in strictly medical terms. Eventually, imbecility became linked to a person's inability to show moral restraint and/or lawful behavior. George F. Still, an English physician, is known by many as the founding father of present-day ADHD. Still's discussion about the moral control in his patients as a medical problem shifted the focus from adults to children precisely because he studied the disorder in children.

The end of World War I saw an outbreak of "sleepy sickness" that became known in the 1920's as encephalitis lethargica (EL). Until this time, it was a disease unknown to medicine of the day, but quickly became the centerpiece of the medical establishment as it reached epidemic proportions. More often than not, EL was a fatal disease characterized by great sluggishness, hallucinations and fever, but occasionally there were periods of remission followed by full-blown relapses resulting in death. Those who survived were never the same. Rafalovich (2004) cites Kessler (1980) as listing twenty-seven different symptoms which included: "sleep reversals, emotional instability, irritability, obstinacy, lying, thieving, impaired memory and attention, personal untidiness, tics, depression, poor motor control, and general hyperactivity (p 30)." If some of the symptoms look familiar, it is because some of them are associated with present-day ADHD (Barkley, 2006). Furthermore, Rafalovich cites physicians of the day, Kennedy (1924) and Strecker (1929) as viewing these post-encephalitic children's problems being the result of impairment of the neurological processes. This

neurological impairment Strecker saw as affecting either motor behaviors and/or types of conduct.

Emerging from this period after EL, theories and research into the possibility of brain damage or dysfunction resulting from encephalitis, pre-/perinatal trauma, or birth trauma were predominant. From them emerged the hypothesis for the disorder that became known as minimal brain dysfunction (MBD). The term MBD would fade due to its vagueness, over-inclusiveness, little prescriptive value, and no neurologic evidence (Barkley, 2006). Also MBD was rejected because today's diagnostic system is based on observable criteria for the disorder (D'Alonzo, 1996).

Research by Bradley (1937), Bradley and Bowen (1940), Moltich and Eccles (1937) marked the beginning of psychopharmacology as we know it today for the treatment of ADHD (Barkley, 2006). The accidental discovery by researchers administering amphetamine after running pneumoencephalograms on subjects to treat headaches produced marked improvements in behavioral problems and in academic performance (Barkley, 2006).

Virginia Douglas's research in Canada epitomized much of the research which occurred during the 1970's and 1980's. Her models and research elaborated, refined and substantiated the disorder we know as ADHD. The focus shifted from hyperactivity to a focus on attention deficits and impulse control due in part to her research. The shift in focus occurred because hyperactivity could not be isolated as specific to ADHD alone. The DSM-III

(American Psychiatric Association, 1980) would rename the disorder ADD, due in part to her research (Barkley, 2006).

The 1970's and 1980's saw a rise in prescription medication to treat hyperactive, school-age children because research continued to show a marked improvement in behavior due to medication. In addition, the rise in prescriptions was tied to a more rigorous methodology employed in drug studies. Also, a growing interest in the influence of environmental causation of the disorder was noted. People, too, became more interested in their personal environment by consuming natural foods and a general rise in health consciousness; however, it was a campaign by the Church of Scientology and its Citizens Commission on Human Rights (CCHR) that reverberates still today (Barkley, 2006).

In the 1980's, the Church of Scientology sued the American Psychiatric Association for fraud in their development of criteria for determining ADHD. The case was later dismissed, but the Church's persistent warning of the dangers of stimulant medications to treat ADHD left a lasting mark. Today people in general, are more hesitant about the use of stimulant medications to treat the disorder. One positive that grew out of the Church's campaign was the utilization of more multimodal means to treat ADHD (Barkley, 2006).

Theoretical Foundations

In popular usage, a theory may be considered a weakly supported thought, but in scientific usage theory is a stronger concept. A theory in scientific usage must withstand the test of time by being proposed, tested, retested, and ultimately accepted or rejected. ADHD is not a mere theory. ADHD is not

a temporary state that a child will outgrow. ADHD is not due to parental failure to discipline or childhood willfulness caused by a bad temperament, but a real disorder (DeRuvo, Lougy, and Rosenthal, 2009). Barkley (2006) sheds light on the bad-parenting theory by observing it may simply be the fact of an un-medicated ADHD adult parent interacting with their un-medicated ADHD child making a bad situation worse.

ADHD is not some “phantom disorder” because scientific research over 95 years has consistently identified a group manifesting attention problems, impulse control, and sometimes hyperactivity. It is not caused by family problems, too much television or video games, food allergies, excess sugar, or poor teachers or schools (Jaksa, 1998). Those who do not believe in this disorder claim that teachers, doctors, and parents are using medication to keep their children in line, instead of “good old fashioned discipline.” However, most medical doctors, psychologists, psychiatrists, and educators are convinced that ADHD is a real illness. Even the Social Security Administration recognizes ADHD as a disability, and “children who have measurable functional deficits, in the context of school performance are eligible for disability benefits” (Meyers, 2010, p. 11). ADHD is also recognized by the court system, United States Department of Education, the Office for Civil Rights, the National Institutes of Health, the American Psychiatric Association, and the American Medical Association as a disorder (Barker, 2006; Booth, Fellman, Greenbaum, Matlen, Markel, Morris, Robin, and Tzelepis, 2009).

ADHD is not an excuse for misbehavior. Quite the opposite! Professionals, including physicians, educators, and therapists, routinely teach ADHD

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children that living with the disorder is a challenge, not an excuse.

Accommodations for the disabled, mandated by federal and state law, are not meant to excuse anyone from their responsibilities to society; rather, they are society's effort to ensure a level playing field for all, including those living with ADHD (Booth, et al, 2009).

Etiology

People who have ADHD are considered a heterogeneous population with variations in the degree of their symptoms, age of onset, cross-situational pervasiveness, and the presence of comorbid disorders (Barkley, 2006).

Time and on-going studies continue to narrow the search for the causation of ADHD. More and more, research and subsequent studies seem to point in the direction of and support a biochemical and/or organic basis (Pledge, 2002).

Fowler (2002) confirms this biochemical basis by observing that when neurotransmitters do not work the way they are supposed to the entire brain works inefficiently and ADHD behavior is manifested.

Recent studies have focused on abnormal brain anatomy as a cause of ADHD. Through the use of brain imaging technology, including functional magnetic resonance imaging (fMRI), positron emission tomography (PET), and single photon emission computed tomography (SPECT), certain regions of the brain have been identified as different in those with ADHD (Butross, 2007). Through the use of brain imaging, scientists have discovered a developmental delay in some regions of the brain where the ability to suppress inappropriate actions and thoughts, focus attention, remember things moment to moment, work for rewards and plan. The region of the brain that controls movement matures faster than normal, which causes the

two regions to be different. This difference causes the fidgety symptoms associated with ADHD (NIMH, 2008). All of these studies increasingly support the idea that ADHD causation is neurodevelopmental in its origin (Barkley, 2006).

Executive function (EF) impairment is a prominent manifestation of the cognitive dysfunction associated with ADHD among adults. According to Barkley (2008), EF refers to brain functions that activate, organize, integrate and manage other functions. It enables individuals to account for short and long term consequences of their actions and to plan for those results. It also allows individuals to make real time evaluations of their actions and make necessary adjustments if those actions are not achieving the desired result. Impairment in executive skills may appear as impulsive, inattentive and unfocused. In addition, they also appear as immature cognitive and behavior patterns that impair functioning in multiple environments. (Barkley, 2008; Harper & Wadsworth, 2007).

The mounting scientific evidence suggests that the attention system of the brain is a complex process involving multiple regions of the brain. This system uses neurotransmitters to carry messages to various parts of the brain from the brain stem. Certain medications, namely stimulants, have a beneficial affect upon neurotransmitters in the brain resulting in more efficient performance in the attention system (Goldstein, S. & M., 2000). Yet, within the last two decades, research has made significant steps forward due to technology. Knowledge continues to grow based upon cerebral blood flow studies, studies regarding the electrical activity of the brain in those who

have ADHD, and neuropsychological tests focused on frontal lobe dysfunction (Barkley, 2006).

Genetics

Numerous studies have shown that in most cases of ADHD there is a substantial genetic component. These studies include twins, adoptions, genetics and family history (Butross, 2007; Rutledge, 2008). Genetics account for 80% of the causes of ADHD. This genetic predisposition to having ADHD manifests in early childhood (NIMH, 2009; DeRuvo, Lougy, & Rosenthal, 2009).

Scientists have discovered that ADHD is associated with certain gene mutations. Human cells contain about 25, 000 genes that are made up of the material deoxyribonucleic acid (DNA). DNA is made up of four base compounds (adenine, thymine, cytosine, and guanine). Mutations occur when there are alterations in the bases. These alterations consist of the substitution of one for another or the addition or deletion of one or more (Butross, 2007).

The D4 receptor, which is located on chromosome 11 binds to the neurotransmitters dopamine, epinephrine, and norepinephrine. These neurotransmitters perform with each other to control attention, inhibition and motor planning. A mutation in the D4 dopamine receptor gene would cause abnormal brain signals to go to the brain. (Butross, 2007). This imbalance contributes to the symptoms associated with ADHD (DeRuvo, Lougy, & Rosenthal, 2009). A mutation on chromosome 16 has also been linked to ADHD. Mutations in chromosome 16 have been implicated in

Autism, which has led to speculations that changes in this chromosome region contribute to the common deficits of inattention and hyperactivity that are found in both ADHD and Autism. Siblings have a strong chance of sharing these mutations (Butross, 2007).

NIMH (2008) suggested research has also shown children with ADHD, who carry a particular version of a certain gene, have thinner brain tissue in the part of the brain that is linked to inattention. However, as children with this gene grew up, the brain developed to a normal level of thickness and their ADHD symptoms improved (NIMH, 2009).

While genetic predisposition for ADHD has been demonstrated through family, adoption and twin studies; the child of a parent with ADHD will not necessarily develop this disorder. Studies of family histories of children with behavioral disorders have not always shown a clear pattern of inheritance. Researchers are looking at several genes that will cause people to be more likely to develop this disorder. Research currently continues to identify the genes responsible for ADHD. Knowing the genes involved will help researchers prevent the disorder before symptoms develop. Learning about specific genes could also lead to better treatments. (Butross, 2007; NIMH, 2009).

Environment

Environmental factors are linked to the cause of ADHD. A common theme across biomedical sciences is that exposure to adverse environments during prenatal and postnatal developmental periods, increase the risk of disease later in life. Examples of specific prenatal risk factors associated with ADHD

symptoms include exposure to nicotine, alcohol and/or recreational drugs (Butross, 2007).

Delivery complications such as breech presentation, prolonged labor, or nuchal cord have been implicated as possible causes of ADHD. Nuchal cord is what happens when the umbilical cord becomes wrapped around an infant's neck during delivery. Low birth weight also indicates a potential risk. Risk factors such as meningitis, maternal stress during pregnancy, and poor maternal diet also play a part in the development of this disorder (Mill & Petronis, 2008). Injury while in the uterus due to high blood pressure or infection in the mother as well as infection or injury after birth, have all been implicated as potential causes of ADHD. Children who have suffered a brain injury show signs of ADHD; this type of injury does not occur often in children. Other suggested environmental factors include low parental education level, poverty and parenting styles. These factors have not yet been proven (Butross, 2007; NIMH, 2009).

Prenatal exposure to chemical toxins such as polychlorinated biphenyl's (PCBs) and glucocorticoids early in development can increase the risk of childhood ADHD. PCBs were used as coolants and insulating fluids for transformers and capacitors. Glucocorticoids are a class of steroid hormones involved in the regulation of glucose metabolism. NIMH (2008) also found research on the effects of chemical toxins involving preschoolers who were exposed to high levels of lead. Lead can be found in plumbing fixtures or paint found in older structures. The results of this study showed preschoolers were at high risk for developing ADHD due to high levels of lead (Butross, 2007; NIMH, 2009).

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Researchers have proposed that vitamin deficiency, allergic reactions to food additives and watching too much television may also be related to ADHD (Butross, 2007). Schmidle (2010) suggested that continued research has shown excessive sugars, dyes, preservatives and possibly even milk and wheat products can affect activity level, attention, interactions, confidence and sense of well being. In addition, Schmidle (2010), found research explaining how eating high fat and high calorie foods increases brain stimulation (Schmidle, 2010). According to the NIMH (2008), British research suggests that consuming certain foods with additives such as artificial colors are linked to hyperactivity. Research is in progress to confirm these findings (Butross, 2007; NIMH, 2009). On the other hand, Ballard, Hall, & Kaufmann (2010) found no sufficient research that currently exists suggesting sugars or fats cause or increase ADHD symptoms.

Differential Diagnosis

The medical approach versus the psychosocial approach causes a large amount of variance in determining diagnosis, prevalence and treatment in individuals with ADHD. This disorder is the core of many mental disorders; lack of recognizing the issues involved with this diagnosis has led to an increase of incorrect results or findings in clinical practice (Schlatcher, 2008).

Any combination of problematic behaviors can be diagnosed as ADHD.

Excluding all other possibilities during assessment makes diagnosing less complicated (Butross, 2007; Rutledge, 2008). Morrison (2006) explains how to differentiate ADHD from other conditions during childhood. Children with ADHD learn slowly which may cause them to appear mentally retarded (MR); however, once their attention is retained, they are able to learn at a normal

level. Children with ADHD are differentiated from Autism by communication. ADHD children communicate normally, unlike those with Autistic Disorder (AD). Children who have ADHD could appear as having Depression. Both of these disorders have poor attention span and/or agitation; however, the duration of depression does not last as long as the duration of ADHD.

Individuals with Tourette's Disorder (TD) display hyperactivity; however, those individuals who have only ADHD will not show motor and vocal tics normally displayed by individuals with TD. Other disorders such as Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) also appear similar to ADHD; however, children who have these disorders show a lack of remorse and their behaviors are purposeful unlike ADHD (Morrison, 2006).

Controversy exists regarding ADHD and its similarities to Bipolar Disorder (BD). BD overlaps significantly with ADHD; however, when irritability is severe in youth with ADHD, an episode of mania can differentiate the two disorders. During an episode, the child's symptoms should be compared to his or her usual behavior. In addition, adolescents with ADHD can develop severe behavior problems, similar to BD, in response to the combined stress of social pressures, academic demands, and puberty (Baroni, Leibenluft, Luckenbaugh, Lunsford, & Towbin, 2009).

Rutlege (2008) proposed that behavior problems can be linked to ADHD. Behavior problems that are limited to home are more likely to stem from family stress, poor parenting or difficult family dynamics. If students have problems at school but get along well in other environments, this usually suggests they are struggling with teaching or learning difficulties and not ADHD. Problems getting along with peers are usually due to poor social

skills. Problems that are confined to the playground, including unsupervised playtime in the neighborhood, suggest problems coping with unstructured situations or having a personality trait known as risk-taking or thrill seeking. People with this trait require more stimulation to avoid boredom, and they are drawn to activities that most youth would view as overly dangerous or frightening. Problems getting along at work can develop when children are old enough to hold down jobs. Teenagers who have difficulties at work but get along in other settings, may be able to solve this problem simply by changing jobs or employers. Therefore, a standard ADHD diagnosis should not be made when a child only has significant problems in one setting (Rutledge, 2008).

According to Butross (2007), if a child is reared in a chaotic environment, they may show signs of ADHD. A recent move, family separation, divorce, death, or other significant events could affect concentration. A child who is experiencing ongoing violence in the home, such as witnessing spousal abuse or experiencing some form of abuse, will likely have problems paying attention in school due to the mental and possibly physical injuries that he or she is dealing with (Butross, 2007).

A school setting has factors that can cause a child who may not have ADHD to display ADHD-like behaviors. A lack of classroom structure and routine, unclear teacher expectations, lack of engaging lessons, and poor behavior management may create disruptive behaviors in children who do not have ADHD. These children may be misinterpreted as having ADHD. A child in this environment may be unclear about behavioral, academic, and social expectations. In the absence of clear expectations, children find themselves

lost, anxious and out of control. This environment is often accompanied by clutter, too much downtime, unstructured free time, and boring tasks.

Children will create their own structure, including attention seeking behaviors, acting out behaviors, and uncontrolled movement inside the classroom (DeRuvo, Lougy, & Rosenthal, 2009).

To eliminate disorders and conditions that can mimic ADHD, in addition to hearing or vision problems, health problems, sleep disorders, family issues, or other behavioral disorders; an extensive medical history, psychosocial evaluation, and physical and neurological exam must be a part of the evaluation process. A psychological and/or speech and language evaluation may also be necessary to determine whether there are problems with intelligence, specific learning disabilities, or language processing (Butross, 2007).

ADHD commonly coexists with other disorders and conditions. The differential diagnosis of ADHD and the pattern of psychiatric comorbidity, which means having two or more diagnosable conditions at the same time (Kratochvil, Vaughn, & Wetzel, 2008) vary with each age group and complicate diagnosis. Comorbidity can be seen in children with ADHD as early as pre-school age. Problems that co-exist with this population include language problems, developmental coordination disorder, and underachievement in reading and math. 60% of children with ADHD have been found to have impairments in receptive and expressive languages (Kratochvil, Vaughan, & Wetzel, 2008; Houghton, O'Donoghue, & Toner, 2006).

Comorbidities with ADHD throughout the lifespan include: ODD, CD, TD, BD, learning and communication disorders, organizational problems, social phobia, Separation Anxiety Disorder (SAD) Generalized Anxiety Disorder (GAD)-most common, Obsessive Compulsive Disorder (OCD), Depression, enuresis, sleep problems, Pervasive Development Disorder (PDD), and many forms of physical illness such as asthma and accidental injury. (Butross, 2007; Kewley & Latham, 2008; Kratochvil, Vaughan, & Wetzel, 2008; Watkins, 2009).

According to Hay, Levy, Martin, & Pieka (2006), parenting style is also a factor of comorbidity. Parents of children with ADHD may not reach criteria themselves for ADHD; however, they may have problems with organization and with impulse control. These problems may limit parents' ability to implement consistent parenting practices. This type of parenting style is termed geno-type environment correlation; the child who has the genes for ADHD lives in an environment that fails to limit ADHD-related behaviors (Hay, Levy, Martin, & Pieka, 2007).

According to Watkins (2009), the differential diagnosis in adults includes Antisocial Personality Disorder and Mood Disorders. Individuals who experience mood disorders also have difficulty with concentration. The adult diagnosis of ADHD should not be made if the individual's symptoms are explained better in other diagnoses such as Schizophrenia, an anxiety disorder, or a personality disorder (Morrison, 2006). Watkins (2009) found research suggesting that the incidence of comorbidity is higher in adults than in children. However, many of the studies looking at the issue of comorbidity were difficult to compare (Watkins, 2009).

Adults with ADHD share similar clinical features with adults who have Borderline Personality Disorder (BPD). These features include impulsivity, emotional dysregulation and cognitive impairment. ADHD in childhood has been highly associated with the diagnosis of BPD as children approach adulthood. Adult ADHD often co-occurs with BPD and other cluster B disorders found in the DSM-IV-TR (Philipson, 2006). There are many different pathways to comorbidity. Combined behavioral and molecular genetic studies are contributing to an understanding of why and how to address these issues (Hay, Levy, Martin, & Pieka, 2006).

Diagnosis and Treatment Interventions

According to Brown (2005), ADHD affects people at all levels of intelligence. Everyone sometimes experiences symptoms of this disorder; however, only individuals who experience chronic impairment warrant a diagnosis of ADHD. There is no single measure that can rule out an ADHD and a diagnosis of ADHD should not be made based on genetic testing alone. (Brown, 2005; Butross, 2007)

Butross (2007) suggested the best way to evaluate a child for ADHD is through a team approach. The team should be composed of the child or teen, parents or primary caretaker, teachers and physician. A psychologist, social worker, and speech and or occupational therapist may also be needed, depending on the symptoms present (Butross, 2007). When diagnosing ADHD in children, social living conditions play an important part. ADHD should only be diagnosed in children who live in a healthy and secure environment (Morrison, 2006).

ADHD is a clinical diagnosis based on patient interviews and collateral information. Since there are no standardized, validated technologies or testing procedures for diagnosing ADHD, multiple tools are available to supplement the clinical assessment. Screening instruments and rating scales are efficient means of identifying symptoms (Kratochvil, Vaughan, & Wetzel, 2008). Some of the most common instruments used are: