ADHD in Adults

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Attention-deficit/hyperactivity disorder (ADHD) in adults is a common-and, frequently undiagnosed-psychiatric disorder. This article will focus on the symptoms, associated features, diagnosis, differential diagnosis, prevalence, etiology and treatment of this illness. Much of the information is based on the work my colleagues and I have done over the past 20 years. Further documentation of the subjects in this article, as well as the instruments discussed, can be found in my monograph, Attention-Deficit/Hyperactivity Disorder in Adults (1995).

Syndrome Symptoms

With no DSM-IV criteria developed for ADHD in adults, the first task is to define the symptoms of the syndrome in adults. The DSM-IV's symptomatic criteria refer to children and, not surprisingly, many of them are age-limited. In this category are such symptoms as "often runs about or climbs excessively" or "often has difficulty playing or engaging in leisure activities quietly." These specific behavioral criteria may increase interrater reliability in diagnosing childhood ADHD but they are not directly applicable to adults. Accordingly, we have had to develop tentative operational criteria. These were necessary when we began our investigations of ADHD in 1976 (before DSM-III), and we have continued to develop them because they specify characteristics more relevant to adults. Since ADHD begins in childhood, the first task is to determine the psychiatric status of the patient as a child and to make a retroactive diagnosis. Few of our patients had been evaluated or treated as children; nevertheless, one can inquire of an adult about the presence or absence of childhood DSM-IV symptoms for ADHD, but in most adults memory is cloudy, and we lack a measure of its reliability. As a screening measure we seek to obtain more global and seemingly better-remembered behavioral characterizations. To avoid the memory problem, we have employed three approaches. The best method is to speak to the patient's parents. Barring this, we have found it useful to have the parents rate their now-adult offspring as he or she had been in childhood on the Parents' Rating Scale (Wender and others 1981). This is a version of the workhorse of childhood ADHD assessment, the Conners Rating Scale. It has been standardized on normal and ADHD adults, and the score can be translated into the percentile of an adult's "hyperactivity" in childhood. The third technique is to administer a rating scale, the Wender Utah Rating Scale, in which the adult reports on his or her memories of 25 descriptors characteristic of ADHD in childhood (Ward and others 1993). This has been standardized on normal adults, adults with a major depressive disorder and adults with ADHD. The next step is to identify ADHD symptoms in the adult. The criteria that my collaborators and I have developed and employed are the Utah Criteria. The Utah Criteria makes hyperactivity in childhood continuing into adulthood a mandatory diagnostic symptom. This criterion obviously eliminates that subgroup of ADHD children and ADHD adults who were and are characterized by inattentiveness without hyperactivity and impulsivity. The more stringent requirements were employed in our research to limit our investigations to the most clear-cut subgroup of patients with ADHD. What was useful, however, for research purposes need not be helpful clinically, because it is clearly the case that many children and adults with inattention alone respond to the same treatments as do children and adults with hyperactivity and/or impulsivity. Our diagnostic criteria also eliminated patients with major mood disorders, schizophrenia, antisocial personality disorder, and schizotypal or borderline personality disorders. This was not to deny the often observed comorbidity; rather, it represented our desire to study a relatively homogeneous sample. Many individuals in these excluded categories also have prominent ADHD symptoms; and an important
and as yet unexplored area is the influence of drug treatment on their ADHD symptoms. As one example, it would be of interest to determine whether the affective lability and liability of temper in patients with Cluster B personality disorders respond to the medications effective in the treatment of ADHD. Another issue (which ties in with the natural history of the disorder and will be discussed presently) is that we have observed considerable historical comorbidity for conduct disorder in our adult patients. This is a predictable finding since ADHD children are often comorbid for conduct disorder. (While there are many ADHD children without conduct disorder, most conduct-disordered children have ADHD as well.) This is important prognostically because approximately one-half of children with conduct disorder go on to develop antisocial personality disorder. Little is known about the value of drug treatment of ADHD in the presence of conduct or antisocial personality disorder. We have also encountered a continuing history of learning disorders in our adult patients. These disorders should likewise have been anticipated because ADHD children have increased comorbidity for learning disorders in reading, spelling and mathematics. These skills are rarely assessed in psychiatric evaluations of adults, and it is important to evaluate them in adults with ADHD and treat them appropriately, since learning disorders may persist through adult life.

**ADHD Prevalence**

The next question is that of the prevalence of ADHD in adults. No epidemiological studies have been conducted, but one can reach an order of magnitude calculation by estimating the prevalence of ADHD in children and the fraction of these cases that persists into adulthood. Many epidemiological studies of ADHD in childhood have been conducted. Although the diagnosis of ADHD is categorical, "yes" or "no," the categorical decision is based on the number of symptoms present. Depending on the method and the cutoffs employed, the prevalence of ADHD in childhood ranges from 3 percent to 10 percent. In all instances the disorder is found to be at least two to three times as common in boys as in girls. There is a commonly overlooked issue in determining the "true" prevalence of ADHD: the absence of a gold standard. That is, we lack the microbiological, pathological and physiological measures that are associated with medical illnesses and that permit us to accurately ascertain the sensitivity and specificity of diagnostic methods. We cannot meaningfully determine how sensitive and specific our criteria for ADHD are because we do not have any means of determining whether an individual patient "really" has the disorder. Lacking any such measure, we must make a decision to employ looser or more stringent criteria, and this is relevant to whom we treat. This question will be discussed in the context of the "pay-off matrix" in my discussion of treatment. The natural history of ADHD is optimally assessed by anteropositive studies, in which children with ADHD have been followed through adolescence and into adult life. There are two such studies. Weiss and Hechtman reported on a follow-up at age 25 of "hyperactive" children they had treated when the children were 6 to 12 years old, 60 percent of whom they were able to evaluate as adults. Two-thirds of their subjects complained of at least one symptom of restlessness, distractibility or impulsivity versus 7 percent in the controls. Approximately one-half of the patients continued to have moderate or severe problems, while approximately one-quarter had developed antisocial personality disorder. Mannuzza and colleagues (1984, 1991, 1993) followed a cohort of "hyperactive" children from childhood to ages 18 and 26 and were able to obtain follow-up data from nearly all of them. At age 18, 40 percent of the patients had ADHD (compared to 3 percent of the controls), 27 percent had conduct disorder or antisocial personality disorder (versus 8 percent of the controls) and 16 percent had non-alcohol substance abuse disorder (versus 3 percent in the controls). At age 26, only 11 percent continued to have full or partial ADHD symptoms, while 18 percent had antisocial personality disorder; and the same number (16 percent) continued to have non-alcohol substance abuse disorder. The most striking feature of these studies is the relative persistence of the disorder through adolescence and its apparent decrease in early adult life. How is one to interpret the reported drop in the prevalence of ADHD between the ages of 18 and 26? One obvious answer is that the children simply outgrew the disorder. Another interpretation is that in working with the 25-year-olds, the investigators were dependent on reports by the subjects alone, while in childhood the informants had included both the subject and his or her parents. Our findings have been that adults with ADHD-having had the syndrome for their entire lives-often fail to report many of their symptoms or fail to report their severity. From a practical standpoint, we have found it necessary to include patients' spouses or "others," both in attempting an initial assessment and in determining the response to treatment. Based only on reports from the patients themselves, it is likely that these studies may have
underestimated the true prevalence of ADHD. Putting the prevalence and natural history data together, it appears that one- to two-thirds of the 3 percent to 10 percent of the childhood prevalence, or somewhere between 1 percent and 6 percent of the general population, continue to manifest appreciable ADHD symptoms into adult life.

### Etiological Factors

About 25 years ago, I advanced the hypothesis that the etiology of (as it was then called) minimal brain dysfunction (MBD) might be genetic in origin and produced by decreased catecholaminergic functioning (Wender 1971, 1972). My conjectures about a genetic origin were based on an apparently increased frequency of MBD symptoms among the siblings of children with that disorder and an increased frequency of other forms of psychopathology (alcohol abuse, antisocial personality disorder) among the parents of these patients, as well as the absence of such psychopathology in the adoptive parents of MBD children. Since that time there has been a large number of genetic studies of ADHD. These have been investigations of the familial association of ADHD; of psychopathology in the parents and siblings of ADHD children; of the concordance for ADHD in monozygotic and dizygotic twins; and of foster or adopted children with ADHD, which allows one to separate the contributions of genetic and rearing factors. The initial family studies reported an increased frequency of alcohol abuse, antisocial personality disorder and, possibly, Briquet's syndrome in the biological parents of "hyperactive" children as compared to controls; and an increased frequency of "hyperactivity" in the siblings of hyperactive children (Cantwell 1972; Morrison and Stewart; Morrison; Biederman and others 1992; Faraone and others). The clustering of ADHD, antisocial personality disorder and alcohol abuse is of interest because family studies conducted by investigators at Washington University also found that these three disorders coexist in the same families, which suggests a genetic basis for this clustering (Guze). Subsequent family studies of ADHD have indicated that the psychopathology in the parents of ADHD children correlates with ADHD children who are comorbid for conduct disorder. Nonetheless, there is clearly an increase of such parental psychopathology in the parents of children with "pure" ADHD. Using other methods, a relationship between ADHD and alcohol abuse was reported by Tarter and others and Wood and colleagues (1983), both of whom found that ADHD was associated with early-onset alcoholism. Furthermore, Goodwin and colleagues found that the adopted-away sons of alcoholics who were alcoholic were more likely than the nonalcoholics to have had symptoms of "hyperactivity" in childhood. Since the familial clustering of psychiatric disorders may be due either to genetic or to psychological transmission, other methods are necessary to differentiate between these two modes of transmission. One technique is to determine the concordance for ADHD among monozygotic and dizygotic twins. Ostensibly both types of twins share the same familial psychological environment and an increased frequency of concordance in monozygotic twin pairs, as compared to concordance in dizygotic twin pairs, is a manifestation of genetic factors. In a large sample of twins Goodman and Stevenson found an increased concordance of ADHD among monozygotic as compared to dizygotic twins (1989a,b). This method allows an estimation of "hereditability," expressed by genetic linkage, which was found to be 64 percent. Monozygotic twins, however, may in reality experience a different psychological environment from that experienced by dizygotic twins, and the twin methodology cannot completely control for this effect. The appropriate strategy to resolve this question is to study foster and adopted children. Safer investigated the status of full and half-siblings of ADHD children who had been placed in foster care. He found among the siblings an increase of ADHD-like psychopathology which was twice as great among the full as the half-siblings (as would be expected on genetic grounds). Two other adoption studies (Cantwell 1975; Deutsch) investigated the psychiatric status of the biological parents of children with ADHD, the adoptive parents of children with ADHD and the biological parents of children without psychiatric disorder. They found an increased frequency of ADHD-like psychopathology only among the biological parents of ADHD children. Taken together these studies have demonstrated the presence of genetic factors in the transmission of ADHD and suggest that children with ADHD may be at an increased risk for antisocial personality disorder and alcohol abuse. (For fuller detail, see Wender 1995.) The second etiological hypothesis was a neurophysiological one. The reasons behind the catecholaminergic hypothesis were based on several observations: the first were reports of behavioral problems among children...
who had contracted von Economo's encephalitis during the pandemic that occurred in the late teens and early '20s of this century. Many children who recovered from the acute illness developed a postencephalitic behavior disorder whose symptoms were very similar to those of mixed ADHD and conduct disorder. Another finding of interest is that adults who recovered from the acute encephalitis frequently manifested symptoms of Parkinson's disorder and that postmortem examination of adults who had died from the disorder revealed lesions in the basal ganglia and substantia nigra. Von Economo reported that the histopathological changes seen in children dying of the disease were identical with those found in adults. These observations tie in with a later finding that idiopathic Parkinson's disorder is associated with decreased dopaminergic functioning in the brain. This is due to degeneration of dopaminergic neurons in the brain areas mentioned and is associated with decreased levels of the principal metabolite of dopamine, homovanillic acid (HVA), in the cerebrospinal fluid (CSF). A second reason for a dopaminergic hypothesis is that some of the drugs that are dramatically effective in reducing or eliminating the symptoms of ADHD, the amphetamines and methylphenidate, are indirect dopamine agonists. Since the 1960s, I had wanted to do the experiments detailed below on children with ADHD but felt one could not legitimately obtain fully informed consent to conduct invasive biological tests on children. Our studies of adults allowed us to conduct these experiments. The first was to investigate the level of HVA, the principal metabolite of dopamine, in the CSF of adults with ADHD and with controls. Such a study (Reimherr and others) found a decreased level of HVA in the adults with ADHD who responded to treatment with methylphenidate, and increased levels of HVA in nonresponding ADHD patients. This replicated the findings of two smaller studies in "hyperactive" children (Shetty and Chase) and of children with minimal brain dysfunction (Shaywitz and others). The second technique was to administer pharmacological doses of the precursor amines of dopamine, namely phenylalanine, l-tyrosine, and l-dopa (Reimherr and others; Wood and others 1982, 1985). Our principal finding was that approximately half the patients receiving tyrosine experienced a moderate-to-marked improvement of their ADHD symptoms; phenylalanine and l-dopa did not have such an effect. These findings are post facto interpretable. Increasing phenylalanine or tyrosine should not increase dopamine. The primary metabolic chain is phenylala-nine(r)tyrosine(r)-l-dopa(r)dopamine; and the conversion of tyrosine to l-dopa, which is catalyzed by the enzyme tyrosine hydroxylase, is the rate-limiting step in the formation of dopamine. On the basis of the enzyme kinetics, the observed clinical effectiveness of tyrosine could be explained by increasing tyramine, which would increase dopamine by an alternative metabolic pathway. The response to l-dopa was increased fatigue and a decreased ability to concentrate. This can be attributed to its acting as a false neurotransmitter (i.e., it could have been taken up by nondopaminergic neurons and might have displaced their transmitters and inactivated them). The third approach was to administer drugs with a comparatively specific action to patients with ADHD. The hypothesized relevant neurotransmitter dopamine is metabolized in the brain by monoamine oxidase B (MAO-A metabolizes predominantly serotonin and norepinephrine). In low doses two MAO inhibitors, pargyline (no longer marketed) and l-deprenyl (selegiline [Eldepryl]), are specific MAO-B inhibitors. We found that in low doses both drugs produced moderate-to-marked improvement in about 60 percent of ADHD adults (Wender and colleagues 1983; Wood and colleagues 1983). Since at low levels these drugs presumably increase the availability of dopamine and do not increase levels of serotonin and norepinephrine, the results are compatible with the dopaminergic hypothesis. Further trials of selegiline (now available as an orphan drug) are warranted.

**Differential Diagnosis**

The characterization of the predominant symptoms of ADHD is described in my book (Wender 1995). Because ADHD individuals may exhibit depression, affective lability and irritability, ADHD may sometimes be confused with cyclothymic disorder and with borderline personality disorder (BPD). With regard to the former, the shifts of mood seen in cyclothymic disorder are of weeks' or months' duration and not from hour-to-hour or day-to-day as seen in ADHD. Anhedonia and physiological concomitants are absent in ADHD as are the depressive personality traits which Akiskal describes as characterizing "subaffective dysthymia." The ADHD and BPD patients seemingly share symptoms of impulsivity, affective instability, angry outbursts and feelings of boredom. These symptoms, however, differ both quantitatively and qualitatively between the two diagnostic groups. The ADHD
patient's impulsivity is short-lived and is thoughtless rather than "driven." The ADHD patient's anger is episodic and also short-lived compared to the brooding anger of the BPD patient. The major differences between ADHD and BPD patients is that the former do not have the intense conflicted relationships, suicidal preoccupations, self-mutilation, identity disturbances or feelings of abandonment seen in BPD. However, these differences are not clear-cut in all instances and the medications that are useful in the treatment of ADHD might be of value in such individuals' symptoms when the latter are like those seen in ADHD. From a practical standpoint, the diagnosis of ADHD requires the presence of an "other." We have employed spouses, significant others, adult children and parents of adults. Without their observations, critical symptoms are underestimated or not disclosed. The same, as said earlier, is true in regard to estimating the response of symptom treatment.

**Treatment**

We have conducted placebo-controlled and open-label drug trials in more than 300 patients. These include four double-blind placebo-controlled trials: three of methylphenidate (Ritalin) [Wood and others 1976 (n=15); Wender and others 1985 (n=37); Wender and others 1996 (n=124)]; and one of pemoline (Cyclert) [Wender and others 1981 (n=48)]; a total of 224 ADHD patients treated with stimulants. In addition, we have treated 79 patients in open label trials: pargyline, Wender and others, 1983 (n=16); l-deprenyl (selegiline), Wood and others 1983 (n=11); bupropion (Wellbutrin), Wender and Reimherr (n=19); levodopa, Wood and others 1982 (n=8); dl-phenylalanine, Wood and others 1985 (n=13); and L-tyrosine, Reimherr and others 1987 (n=12). We have found in crossover and parallel design studies that about 60 percent of patients receiving stimulant medication manifest moderate-to-marked improvement, as compared with 10 percent of those receiving placebo. These degrees of responsivity are reflected in Global Assessment of Functioning (GAF) scores in patients with moderate-to-marked improvement. The average pretreatment GAF scores in the studies are about 55 (moderate symptoms) and posttreatment scores of about 75 (slight symptoms present only in reaction to stress). As mentioned, we have also conducted open studies of pargyline and selegiline in 27 patients and have found, again, that about 60 percent exhibited moderate-to-marked improvement to treatment with an MAO inhibitor. Lastly, we have conducted a therapeutic trial of bupropion in 19 patients who had previously responded to stimulants or MAO inhibitors and found that approximately half responded to bupropion, and they decided to remain on that drug. Turning to other drugs, in general, as with children, the tricyclic antidepressants have not been useful. Patients manifest an immediate response and after six to eight weeks become tolerant to the drug despite an increased dose. They seem less tolerant of the side effects of these drugs than are depressed patients and complain of the anticholinergic effects, weight gain and impaired sexual functioning. SSRIs appear to be of no value in ADHD patients who are not concordant for major depressive disorder or dysthymia, but may be of considerable benefit for patients with these disorders and ADHD. There have been two attempts to replicate our treatment studies. Mattes and others conducted a placebo-controlled trial of methylphenidate in 26 patients with evidence of residual ADD with hyperactivity and 35 patients with similar adult symptoms but no childhood history of ADD-H. No overall benefit from methylphenidate was evident, regardless of childhood history of ADD-H. There are several reasons why this may have occurred, foremost of which was the nature of the sample: 60 percent did not meet our criteria of childhood "hyperactivity" and many of the patients were comorbid for substance abuse and BPD, patients we have excluded in our treatment. Spencer and colleagues replicated our findings in a randomized, seven-week, placebo-controlled, crossover study of methylphenidate in 23 adults patients with ADHD. Our studies, taken together, have demonstrated the efficacy of methylphenidate, pemoline and MAO-B inhibitors in the treatment of adults with ADHD by producing a robust response in at least 60 percent of patients. The most effective treatment of the ADHD adult involves education about the disorder, drug treatment and psychotherapy focused on ADHD concomitants. Although medication is the main factor in my treatment of adults with ADHD, education and psychological management play important roles. After evaluation and a discussion of ADHD symptoms, I present the patient with the therapeutic pay-off matrix, i.e., the benefits and liabilities of offering or not offering a therapeutic trial of medication when he or she does or does not have the disorder. A consideration of the four possibilities reveals that the risks of treating when the patient does not have the disorder are minimal while the disadvantage of not offering a trial of treatment when the patient does have ADHD is most
unfortunate. The above holds with the proviso that the use of stimulant drugs does not lead to abuse. We do not know whether ADHD individuals will experience “highs” if they take large or intravenous doses of methylphenidate or amphetamines. A few clinical cases suggest that they can produce euphoric “highs.” For this reason, they should be used very cautiously or not at all in persons with a history of drug abuse. In general, therapeutic trials are warranted whenever the diagnosis seems probable because the benefits can be assessed rapidly. Here is a list of the changes in symptoms seen when treatment is effective.

1. **Hyperactivity** - Fidgeting and restlessness decrease; patients are able to relax; then are able to stay at their desks or at the dinner table or at a movie or in church.

2. **Inattention** - Concentration is greatly improved. It is not only that patients can concentrate better, they can concentrate when they want to. Distractibility diminishes or disappears. Attention to spousal conversation improves and frequently is quickly manifested in better marital relations.

3. **Mood lability** - Both highs and lows decrease as do feelings of boredom; mood is described as “level” or “stable.”

4. **Temper** - The threshold for outbursts is raised. Patients are less irascible and their angry outbursts are less frequent, less extreme, and frequently disappear altogether.

5. **Disorganization** - Organizational activities become manifest. This is evident at school, in running a household, in vocational function. Patients may spontaneously establish orderly strategies.

6. **Stress sensitivity** - Patients describe themselves as having their thin skin thickened, able to take life problems in stride, feeling less "hassled" about daily existence.

7. **Impulsivity** - Patients report that they do not interrupt others while listening to them (another feature that improves conversations and relationships), that they think before they speak, that they have become tolerant drivers and that they stop impulse buying.

We have used a structured rating scale to assess these symptoms and their changes in our treatment studies (Wender 1995, Appendix E).

**Practical Comments**

The stimulant drugs are the treatments of choice, but they have one serious shortcoming: their brief duration of action, which necessitates multiple daily doses. Their advantages are that unlike many psychoactive drugs they have an immediate (30 to 45 minutes) onset of action and that methylphenidate and the amphetamines rarely produce idiosyncratic reactions. We have seen only rare tolerance develop to methylphenidate or d-amphetamine. The usual dose of methyl-phenidate is 10 to 15 mg administered every two, two and one-half, or three hours with a total daily dose of 40 to 90 mg. The long-acting formulation, Ritalin-SR, is available in only one dosage size, and many patients report that the formulation does not provide symptom suppression on once-a-day dosing as claimed by the manufacturer. The dose range for d-amphetamine is 5 to 15 mg administered every three to four hours, with a total daily dose of 20 to 45 mg. Dexedrine (dextroamphetamine) is available in an ostensibly long-acting formulation, Dexedrine spansules, but many patients report that this formulation does not provide symptom suppression for the claimed six to eight hours. Methamphetamine is available as a long-acting formulation, Desoxyn gradumets, which do indeed last eight to 10 hours, but they are extremely expensive; the total daily dose is the same as for d-amphetamine, 20 to 45 mg per day. Methylphenidate and the amphetamines seem to be equally effective; however, an individual patient may do better on one than the other, and if the response is not complete, the other stimulant drug should be tried. In all patients, pulse and blood pressure may increase and so they should be monitored; it is best to take measurement at the same time (usually about one hour) after drug administration. Pemoline is administered in a dosage range of 75 to 150 mg per day, usually in one dose, although some patients require bid dosing. Repeated liver function tests must be obtained (for an undetermined period of time) as hepatic toxicity occurs in a small fraction of patients receiving this drug. Pemoline's chief appeal is that it is relatively long-acting and
is a Schedule IV drug. However, it appears that patients do not respond to pemoline as frequently or as well as they do to methylphenidate or the amphetamines. The open studies of the efficacy of bupropion and selegiline were favorable, and these agents should be systematically evaluated because compared to the stimulants they are relatively long-acting and do not carry the abuse stigma. Management of the patient involves more than adequate drug therapy. Having made the diagnosis, I help patients to recognize the ADHD aspects of their current symptoms and behavior, and, as our relationship develops and my knowledge becomes more extensive, of the role ADHD personality characteristics have played in their life history, including academic and vocational experience, friendships, sexual relationships and functioning as a spouse and as a parent. ADHD symptomatology may be intimately woven into all these aspects of life; and it takes patients much time during continuing treatment to identify and understand ADHD contributions to their life story. In my education of patients I help them to see that because they have had ADHD their entire lives, they may have developed techniques for dealing with their symptoms that are no longer adaptive after the ADHD symptoms have remitted. These symptoms may resolve spontaneously or they may require psychotherapeutic intervention. Supportive problem-directed reality therapy (administered by persons sometimes referred to as "coaches") can help with these problems. Obviously, having ADHD does not prevent one from having other psychological problems and these may be more apparent and therapeutically accessible when the symptoms of ADHD have remitted. Couple therapy, with direct behavioral prescriptions and proscriptions, may be useful. ADHD is a life-long disorder and the duration of drug treatment may have to be life-long. Amphetamines have been used since 1937, and no long-term toxicities have been reported. However, both methylphenidate and d-amphetamine increase heart rate and blood pressure, which must be carefully monitored in all patients. Their use may require adjuvant therapy to control heart rate and blood pressure. Whether such drugs interfere with the therapeutic action of the stimulants remains to be demonstrated.

**Conclusion**

ADHD in adults is a common, genetically transmitted disorder, probably mediated by decreased brain dopaminergic functioning. It is usually undiagnosed but fairly easily diagnosed. At least 60 percent of patients experience a substantial, and in many instances a dramatic, response to drug treatment; and such drug treatment can make ADHD patients amenable to a number of psychotherapeutic

**References:**


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