REVIEW ARTICLE

Attention Deficit Hyperactivity Disorder in Adulthood

Diagnosis, Etiology and Therapy

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SUMMARY

Introduction: Until the late nineties, attention deficit and hyperactivity disorder (ADHD) was often regarded in Germany as a disorder that fades away in late adolescence. However, it has recently become clear from numerous studies that core symptoms of ADHD persist into adulthood in a substantial subgroup of patients.

Methods: Selective review of relevant literature in Medline, up to September 2007.

Results: The prevalence of ADHD in adulthood is estimated at about 2%. Core symptoms include attention deficit in the presence of understimulation, chronic restlessness, impulsivity, disorganized behaviour, and disorders of affect regulation. The extent of psychosocial impairment depends on symptom severity, psychiatric comorbidity (such as addiction or depression), and psychosocial support. As in childhood, ADHD in adulthood is a clinical diagnosis. Genetic factors probably play a key role in primary ADHD. Treatment should include psychotherapy and medical treatment.

Discussion: ADHD in adulthood is commoner than for example bipolar disorder or schizophrenia. It may be regarded as a risk factor for the development of other psychiatric conditions. Highly effective treatment is possible not only in childhood but also in adulthood. The problem of off-label use of psychotropic medication in adults limits treatment in adult ADHD.

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Key words: ADHD, adulthood, diagnosis, treatment, methylphenidate

Until the late 1990s, attention deficit hyperactivity disorder (ADHD) was often regarded in the German speaking countries as a disorder affecting only children and adolescents and which fades away with the onset of adulthood. Research has shown, however, that ADHD frequently persists into adulthood and is a relevant differential diagnosis for many psychiatric disorders (1–3). A factor of particular importance for family medicine is the high genetic penetrance of ADHD. In the majority of cases several generations of a family are affected.

Methods

An overview of the diagnosis, etiology and therapeutic options of adult ADHD is presented, based on an analysis of the results of a selective literature search in Medline up to August 2007 including review articles and monographs.

Diagnosis and classification systems

The prevalence of ADHD is estimated at 4% to 5% in childhood and about 2% in adulthood when the diagnosis is based on ICD-10 (2, 4). When applying the DSM-IV diagnostic system of the American Psychiatric Association the prevalence rates are twice as high (2).

The diagnosis of ADHD is a clinical diagnosis established on the basis of patient-reported medical history – whenever possible supported by a carer based history – and the results of a psychopathologic evaluation. As with other psychiatric disorders there are no somatic findings to demonstrate the correctness of the diagnosis.

The accepted diagnostic systems require the symptoms to have been present before the age of seven years, a fact often difficult to validate in older adults. Moreover, there is a subgroup of patients in whom the onset of this condition can only be demonstrated in early youth.

The German guidelines according to Ebert et al., which were elaborated with the support of the German Society for Psychiatry, Psychotherapy and Neurology (DGPPN) (4) do not give preference to any of the internationally established diagnostic systems, but do require physicians to state which system was used.

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BOX 1

Diagnostic criteria for ADHD (DSM-IV)

Diagnostic criteria of hyperactivity and impulsivity modified according to DSM-IV

(6 or more of the 9 criteria must be fulfilled for the diagnosis)

- Fidgets with hands or feet or squirms in seat
- Gets up from seat when remaining in seat is expected
- Runs about or climbs when and where it is not appropriate (in adulthood often a feeling of inner restlessness)
- Has trouble pursuing activities quietly; unnecessarily loud
- Is often "on the go" or often acts as if "driven by a motor"
- Talks excessively
- Blurts out answers before questions have been finished
- Impatience, often has trouble waiting one's turn
- Often interrupts or intrudes on others

Diagnostic criteria of attention deficit modified according to DSM-IV

(6 or more of the 9 criteria must be fulfilled for the diagnosis)

- Does not give close attention to details, often makes careless mistakes
- Has trouble sustaining attention over longer periods
- Does not seem to listen
- Fails to finish tasks
- Has trouble organizing and planning tasks and activities
- Avoids, dislikes things that take a lot of mental effort for a long period of time
- Often loses things needed for tasks and activities
- Is easily distracted by external stimuli.
- Is forgetful in daily activities.

For the **combined subtype** the required criteria of both subtypes must be fulfilled.

ICD international classification system

The "childhood hyperkinetic syndrome" (F90.0) was first introduced in 1978 within the WHO ICD-9 Classification System. In the ICD-10, the option of also diagnosing ADHD in adults was added in 1992. However, there are no specific diagnostic criteria for the diagnosis in adulthood.

DSM diagnostic system

According to the DSM-IV diagnostic system of the American Psychiatric Association, persistence of the symptoms into adulthood was already defined as "residual type" in 1980. As with the ICD-10, one problem is that the criteria are defined on a child-specific

basis and have to be modified for adults (*box 1*) (5). According to DSM-IV, the inattentive, hyperactive-impulsive and combined subtype are distinguished. The combined subtype is most commonly encountered in adulthood. A further requirement is that the symptoms should cause marked impairments in at least two areas of life.

Wender Utah criteria

In contrast, the Utah criteria for ADHD were specially developed for adulthood (modified from [4]):

1. Attention disorder in the absence of stimulation
2. Hyperactivity (for example "feeling of inner restlessness"/"nervousness")
3. Emotional lability
4. Disorganized behavior
5. Impaired affect control
6. Impulsivity
7. Emotional hyperreactivity

The diagnosis is made according to the Utah criteria when: 1. and 2. plus two criteria from 3. to 7. are fulfilled. The Utah criteria take into account the pronounced mood fluctuations commonly present in ADHD to a much greater extent than ICD-10 or DSM-IV.

Course

ADHD usually has its onset in early childhood, more rarely in adolescence. No cases with adult onset have been described. The course is chronic (3), and a phasic course should suggest the differential diagnosis of an affective disorder. When ADHD persists into adulthood, the disorder may be of mild intensity and may appear as merely a variant of "normal" personality traits ("erratic, temperamental"), but may also retain the severity of a disease associated with considerable lifestyle impairment. ADHD is associated with significantly increased rates of early unplanned pregnancies, venereal diseases, traffic accidents, divorces, lower educational achievement, frequent job changes, and unemployment (6).

Supplementary and exclusion diagnosis

Questionnaires

Questionnaires can be quite helpful to assess the extent and course of clinical symptoms and they ensure a structured assessment of symptoms as an additional expedient (7). As with other psychiatric disorders, however, they are of only indicative value and confirm neither the diagnosis nor its exclusion (8).

Neuropsychological tests

According to the guidelines (4), an examination based on psychological tests, for example of attention performance, working memory and impulse control, may contribute to confirming the diagnosis. An individual diagnosis, however, cannot be made on the basis of a test value. When interpreting the results it should be remembered that adults with ADHD can even achieve very good results if interested and stimulated, although they may be suffering from relevant restrictions in daily life.

Somatic exclusion diagnosis

A medical and neurological examination must be performed and a medicine and illicit drug abuse history should be taken for exclusion and differential diagnostic purposes. The guidelines applied in the German speaking countries recommend thyroid function tests and electroencephalography (EEG) (4).

Comorbidities

The high rate of comorbid disorders (80%) and psychosocial consequences is particularly significant in adult psychiatry and psychotherapy (1). Depression (40% to 60%), anxiety disorders (20% to 60%) and addictive diseases (50% to 60%) are among the commonest comorbidities (8). The prevalence rates of ADHD among drug dependent persons and the prison population are significantly higher compared to the general population and run at about 25%. ADHD is thus a considerable risk factor for further psychiatric morbidities (*box 2*).

At the same time, some comorbidities are also differential diagnoses, such as depressive disorder with its impairments of concentration. However, it can be distinguished from ADHD based on its usually phasic course.

Differential diagnosis from borderline personality disorder (BPD) can be particularly difficult due to the high overlap of clinical symptoms – such as impulsivity and emotional lability – and comorbidity. BPD is frequently dominated clinically by states of tension followed by self harm, chronic suicidal ideation and possible symptoms of posttraumatic stress disorder.

The results of the Nordbaden childhood study (9) and our own clinical observations in adults have shown that somatic diseases such as allergies and arterial hypertension are frequent comorbidities.

Neurobiology and differential diagnosis

The exact cause of ADHD remains unknown. Most experts agree, however, that ADHD is not a single clinical disorder but rather represents a group of etiologically heterogeneous entities which share a group of core symptoms.

Numerous genetic studies have shown that children of parents with ADHD also suffer more frequently from ADHD themselves. Parents and siblings of affected patients have a two to eight-fold risk of developing ADHD symptoms (10). A metaanalysis of six twin studies revealed that 80% of the variance of the clinical symptoms can be explained in terms of genetic factors. Adopted siblings of ADHD children have a lower risk than biological siblings, and biological siblings perform more poorly than adopted siblings in neuropsychological tests of sustained attention (10). All these findings point to an important role of genetic factors in the etiology of ADHD symptoms.

Many cerebral imaging studies have demonstrated both structural as well as neurochemical and functional abnormalities in ADHD patients. For example, reductions in total brain volume, prefrontal brain

BOX 2

ADHD as risk factor for other psychiatric disorders

- Depression
- Addiction, substance-bound and non-substance-bound
- Personality disorder
- Anxiety
- Compulsiveness
- Partial performance disorders
- Adaptation disorders (e.g. to unemployment and/or terminated relationships)
- Eating disorders
- Sleep disorder
- Bipolar disorders

(especially right-sided), basal ganglia (especially the caudate nucleus) and the cerebellum (especially the vermix) have been reported (10–13). Neurochemical abnormalities in various areas of the brain have also been reported (14). The good efficacy of adrenergic and dopaminergic substances point to an important role of these systems in the pathogenesis of ADHD. Abnormalities have also been demonstrated for the cerebral dopamine transporter and pre-synaptic dopamine decarboxylase activity in PET and SPECT studies (15). However, these findings have not so far been robustly replicated, and the individual measured values do not reliably differentiate between healthy and sick persons but only become significant in the group mean.

A further risk factor is chronic intrauterine nicotine exposure (10), which is associated with a 2 to 2.7-fold elevated risk for the later development of ADHD (e1). Other factors such as certain diets, lead exposure, sugar and food additives or metabolic diseases such as cryptopyrroluria are also contentiously debated as possible causes of ADHD (10). A recently published study provided support for the hypothesis that certain food additives are associated with the development of hyperactive symptoms later in life (e2). The authors are however unaware of any data that could conclusively resolve these controversies as regards the other factors mentioned.

Chronic familial conflicts, reduced familial cohesion and confrontation with parental (especially maternal) psychopathology are more often observed in families with ADHD sufferers compared to control families (13). For example, depending on the extent of the psychosocial handicap (Rutter's indicator [RI] 1–4), the odds ratio for children from psychosocially handicapped families for developing an attention deficit hyperactivity disorder increases to values of 7.4 (for RI 1) to 41.7 (for RI 4) (e3). Odds ratios > 1 indicate an increased risk.

When thinking about the causes of ADHD it is important to distinguish the elements causality (etiolo-

TABLE

Differential diagnosis and etiologic/pathogenic considerations in ADHD and ADD

Clinical symptoms	Classification	Etiopathogenic context	Findings/indicators	
			Basic diagnosis	Focus and additional diagnosis
ADHD or ADD	Primary ADHD/ADD	No evidence of other pathogenesis or etiology	Medical, neurological and psychiatric examination, EEG, possibly MRI, lab tests incl. thyroid parameters	Positive familial history (FH)
	Secondary ADHD/ADD	Congenital and/or comorbid factors:		
		Fragile X syndrome		FH, typical facies, other stigmata of FX syndrome
		Pyknolepsies		EEG, FH, medical history
		Other familial epileptic syndromes		EEG, FH, medical history
		Gilles-de-la-Tourette syndrome		Typical clinical symptoms, biographical history
		Asperger's syndrome		Typical clinical symptoms, biographical history
		Acquired factors:		
		Birth complications		Birth history
		Inflammatory brain diseases (encephalitis/meningitis)		Childhood medical history
		Intrauterine nicotine exposure		Extraneous medical history
		Illicit drug and toxic substance exposure		Medical history
		Adverse drug reactions		Medical history
		Head trauma		Medical history, EEG, MRI
		Systemic diseases (vasculitis, collagenoses etc.)		Medical history, additional laboratory tests

gy), mechanisms of action (pathogenesis) and clinical picture (syndrome) from each other. The capacity of attention control, impulse control and affect regulation are pathogenically closely associated with the fronto-striato-thalamo-frontal feedback loop systems. However, these are distributed cerebral neuronal networks. Their function may be disturbed at various sites for various reasons, for example due to lesions of greatly varying origin such as perinatal asphyxia, encephalitis, metabolic disorder, intoxication and febrile seizure.

Lesions at various sites in the brain can thereby lead to a similar clinical deficit if an identical feedback loop system is affected somewhere along its course. This means that it is no longer possible to reliably deduce the site of a functional lesion and even less the cause of a disorder, on the basis of the clinical presentation. The function of these feedback loops, however, may also be systematically compromised, i.e. independently of individual lesions, due to functional disorders of the adrenergic or dopaminergic system. Because of the genetic component and the good efficacy of dopaminergic and adrenergic substances on the core symptom of attention control, it may readily be assumed that the adrenergic and dopamine systems play a central pathogenic role at least in a large subgroup of ADHD patients.

Against this background, there is much that argues in favor of distinguishing between a primary and a secondary attention deficit hyperactivity disorder from the etiologic perspective. A positive familial history and lacking evidence of mild cerebral dysfunctions then point to primary ADHD. Birth complications, inflammatory brain diseases, intoxications, head traumas or possibly a familial history of convulsive disorder would rather suggest secondary ADHD (table 1).

Treatment

The guidelines (4) recommend treatment if, in the presence of a definite clinical diagnosis of ADHD, at least one area of life is severely impaired or two areas of life are slightly impaired (box 3).

Treatment should – as in childhood and adolescence – consist of a combination of pharmacotherapy and psychotherapy. A rationale should be provided for monotherapy. If comorbid disorders such as depression or addiction dominate the clinical picture, they should be treated first, for instance with antidepressant medication, detoxification and withdrawal treatment. After treating the comorbid disorder, the extent of ADHD related impairment should be reassessed.

Pharmacotherapy

In contrast to child and adolescent psychiatry, no medication has yet (as at October 2007) been approved in Germany for the management of adult ADHD (off-label use) (box 4).

Stimulant therapy

According to the German guidelines, the first-line medication is methylphenidate. Its prescription is subject to the provisions of the German Narcotics Prescription Act. The available meta-analyses rate the efficacy of methylphenidate as very good (16). The dosage and choice of product – for example as a sustained release formulation – depend on the patient's needs and therapeutic response (17). The Federal Institute for Drugs and Medical Devices recommends a dose range similar to that for pediatric use of 0.5 to 1.0 mg/kg body weight (BW) daily, although in some studies better efficacy was achieved with higher dosages up to 1.4 mg/kg BW daily. In daily clinical practice, however, it has been found that lower dosages are often sufficient for long-term therapy, especially since many adults aspire to achieve a reduction but not complete suppression of the symptoms.

Medical contraindications (box 5) for methylphenidate include untreated arterial hypertension and cardiac arrhythmias. Before starting medication, electrocardiography (ECG) and measurement of blood pressure and pulse are recommended. These parameters should be monitored in all patients receiving methylphenidate treatment since patients without arterial hypertension may also experience a mild increase in blood pressure and pulse rate. Monitoring of body weight is also recommended because loss of appetite is a common undesirable effect. Methylphenidate should not be prescribed during pregnancy and lactation.

Abuse potential of methylphenidate

Oral use as directed in the therapeutic dose range is not generally associated with an increased dependence potential (18), although some cases of abuse involving intranasal or intravenous use have been documented. The sustained release formulations have an even lower abuse potential because of the slower rate of drug influx.

Comorbid addictions are usually treated initially with alternative therapy options such as noradrenergic substances like atomoxetine.

However, patients with dependence diseases can also be treated with stimulants under controlled conditions, i.e. with regular negative substance abuse screening.

Atomoxetine and other substances

If methylphenidate is ineffective or if the patient has contraindications or comorbidities such as depression or anxiety disorders, frequently used alternative medications include atomoxetine or antidepressants such as venlafaxine, reboxetine and desipramine. As regards significant efficacy in adult ADHD, however, so far only the selective noradrenaline reuptake inhibitor atomoxetine has been evaluated – also in larger studies

BOX 3

Multimodal therapy – guideline based

- No need for treatment based on the diagnosis
- Treatment only if – conclusively because of ADHD – there are marked impairments in one life area, slight impairments in several life areas or symptoms of pathological significance
- Methylphenidate is the first-line medication
- Psychotherapy: apply disorder-specific elements
- Combination of medication and psychotherapy, rationale to be provided for monotherapies
- Therapy should always be guided by comorbid disorders, if present
- Further studies, especially long-term studies, are needed.

BOX 4

Off-label use in adult ADHD

Off label use as defined by the German Federal Social Court (Bundessozialgericht) is applicable in adult ADHD when

- quality of life is permanently impaired due to severe ADHD
- no other, approved medications are available
- the database shows that there is a reasonable expectation of therapeutic success

(Point 1 is to be clarified in the individual case, Points 2 and 3 are applicable for adult ADHD)

– in comparison to placebo (19, 20). Atomoxetine is approved for the treatment of adult ADHD if it was already prescribed before the patient was 18 years old. Atomoxetine is also associated with mild increases in blood pressure and resting pulse as well as palpitations. The target dose is 1.2 mg/kg BW. Treatment should be in progress for 3 to 4 weeks before performing a maximal efficacy assessment. Comparative studies on the efficacy of methylphenidate and atomoxetine are still lacking. Other active agents such as modafinil, bupropion and nicotine patch are under investigation but usually in small case numbers and over only a few weeks.

Duration of treatment

Adult ADHD usually exhibits a chronic course and therefore requires long-term medication. In most cases, symptoms reappear after terminating medication.

BOX 5

Contraindications for the prescription of methylphenidate

Absolute contraindications for methylphenidate:

- Pregnancy and lactation
- Untreated arterial hypertension
- Cardiac tachyarrhythmias
- Coronary heart disease
- Arterial occlusive disease
- Cerebral ischemias
- Schizophrenia
- Medicine and/or illicit drug dependence

Relative contraindications for methylphenidate:

- Tic disorders and Tourette's syndrome (deterioration possible)
- Anxiety disorders (exacerbation possible)
- Epilepsies (prescription only under sufficient anticonvulsive protection)
- Bipolar disorders (prescription only with reliable phase prophylaxis)
- Anorexia nervosa

Psychotherapy

Since the psychosocial consequences such as job loss and/or interrupted relationships are frequently predominant features in adult ADHD and these aspects cannot be influenced directly by medicinal therapy, psychotherapeutic interventions are also recommended. The group and individual psychotherapy concepts evaluated so far are based on cognitive-behavioral and/or dialectic-behavioral therapy and show positive results (21–24). Both patients without medication and patients who still have residual symptoms after ADHD specific medication derive benefits. Psychotherapy can reduce the severity of ADHD and provide an improvement in commonly associated symptoms such as depression and anxiety and in self esteem. Initial evidence suggests that combination therapy comprising pharmacologic and psychotherapeutic components may be superior to medication alone (25). However, no study has yet evaluated the efficacy of psychotherapy compared to ADHD specific medication on a randomized, blind basis. The Federal Ministry for Education and Research is therefore sponsoring a large, randomized, multicenter study to further evaluate structured psychotherapy compared to one-to-one psychiatric interview (clinical management) in combination with methylphenidate or placebo.

Conclusions

Adult ADHD is a disorder that can be validly diagnosed and which, as in pediatric and adolescent psychiatry, can be treated medicinally and with psychotherapy with positive results. Further scientific research into the long-term course of this condition and the efficacy and tolerability of different therapeutic strategies is necessary.

The treatment of adult patients in clinical practice is impeded at present by the lack of an approved medication.

Conflict of Interest Statement

Dr. Philipsen has received fees for lectures and advisory activities and travel expense reimbursements from the companies Medice, Janssen-Cilag, Novartis, and Lilly. She has also received research sponsorship from Medice and Janssen-Cilag. PD Dr. Heßlinger has received lecture fees from the companies Boehringer, AstraZeneca, Lilly, Pfizer, and Medice. Prof. Dr. Tebartz van Elst has received lecture fees and travel expense reimbursements from the companies Janssen-Cilag, Lilly, Wyeth, AstraZeneca, and Otsuka.

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