# Attention deficit hyperactivity disorder in adults: diagnostic imperatives

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#### **ABSTRACT**

At present, no biological or psychological tests with sufficient sensitivity or specificity can replace the meticulous process of distilling the clinical characteristics of attention deficit hyperactivity disorder (ADHD) in adulthood. The level of diagnostic confidence can be enhanced via a semi-structured interview, confirming the early onset, longitudinal persistence and multidimensional impairments. Obtaining collateral information to consolidate the retrospective symptomatology is mandatory, since individuals with chronic attentional problems are prone to recollection bias. A structured outline of the diagnostic trajectory is described, with special attention to age specific core symptoms, secondary problems and co-morbidity that can accompany or compound this not consistently recognised syndrome. Finally, potential pittfalls that can culminate in overand under diagnosis are critically examined.

#### **Keywords:**

Attention deficit, Hyperactivity, Adults.

### INTRODUCTION

Attention deficit hyperactivity disorder (ADHD) constitutes the most frequent behavioural disorder in childhood, with a prevalence of 3-5%. This highly heritable disorder persists in at least 30% of adults, so the estimated prevalence in this age group is approximately 1%. Genetic factors- as evidenced by family, twin and adoption studies <sup>1</sup> are the most important etiological components of this neurobiological condition, which is often found in conjunction with significant co-morbidity and pervasive impairments in all domains of life.<sup>2</sup>

The level of persistence has been inconsistent across studies. The prevalence rates of a syndromatic (less-than-full-syndrome);symptomatic (less-than-subthreshold-diagnosis) and functional (full recovery) remission were estimated in a recent study ,clearly indicating that the proportion of subjects experiencing remission varies considerably with the definition of remission used. The prevalence of syndromatic remission was greater than 60%, whereas the rates of symptomatic and functional remission were 30% and 10% respectively.<sup>3</sup>

It should be clear from the outset that ADHD is not a maturation problem that one eventually outgrows. Neither is it based on the emotional sequelae of intrapsychic conflicts or poor parenting. For these patients who are frequently denigrated for being lackadaisical, unmotivated or incompetent, the diagnosis of ADHD is not an excuse, but a welcome explanation.

The purpose of this article is to set out some guidelines regarding the diagnosis of ADHD in adults.

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#### **SYMPTOMATOLOGY**

Examples of the typical core triad are listed in Table I.They do not represent a diagnostic tool, but are intended to describe some of the cognitive, emotional and behavioural patterns frequently observed amongst these patients.

# Table I ADHD symptomatology in adults

#### Attentional problems

- · Distractability, aversion of boredom
- · Many projects going simultaneously
- · Difficulties completing tasks
- · Failure to see the bigger picture
- · Fails to organise, fails to plan
- · Lack punctuality
- · Indecisiveness
- · Chronic procrastination
- Fails to prioritise
- · Difficulties reading or listening
- Forgetfulness
- Losing posessions
- Chaotic lifestyle

# Hyperactivity

- · Inner or outer restlessness
- " on the go"
- unable to relax
- pressure of speech
- fidgitiveness

#### Impulsivity

- Tendency to say what comes to mind
- Impatience
- Low frustration tolerance
- Spending sprees, gambling, binge eating, promiscuity
- Substance abuse
- Interrupting conversations
- · Transgression of rules
- Not concidering consequences of behavior

# Course of the core symptoms

The symptomatology varies widely in severity and is context sensitive, influenced by the level of stress, lifestyle and lifecycle. The traditional core symptoms of ADHD change across the course of development. In general, hyperactivity tends to diminish with age, impulsivity changes quality, and attentional problems remain the same but are more disabling as organisational demands increase. Because the attentional problems are the most resilient to change, the behavioural problems are gradually substituted by cognitive difficulties as one ages .

By adulthood, the aimless restlessness of childhood may have been channelled into more purposeful and adaptive behaviours. The patient may hold two jobs, work long hours or indulge in frantic sporting activities, so that the hyperactivity becomes less noticeable.

Impulse dyscontrol in adulthood has more serious consequences than its childhood equivalent. Abrupt terminations of a relationship, speeding or quitting a job at the spur of a moment can have perilous social, legal and financial repercussions, respectively.

ADHD is not so much a disorder of attention deficit, but more of attentional inconsistency. The downstream effects of not being able to sustain or shift attention have a profound impact on the patient's executive function which can be defined as the ability to maintain an appropriate problem solving set for attainment of a future goal. Because their insufficient time management, lack of planning and poor organisational skills are further compounded by an impairment of abstract thinking, these patients tend to stagger under the organisational demands of every day life. Patients suffering from ADHD are frequently unable to maintain an overview, with the unfortunate result that their administrative and budgetary discipline are significantly compromised. Their "time-blindness" culminates in them often being late, rushed and unprepared.

### Secondary problems

The continuance of ADHD into adulthood is often associated with a kaleidoscopic scala of secondary problems and co-morbid disorders, all of which can manifest as the presenting complaint. Underperformance at work or poor academic attainment can result from their poor executive skills, inability to motivate themselves to carry out monotonous tasks and error-proneness. Premature dismissal or resignations can be ascribed to interpersonal conflicts with supervisors emanating from impulsivity and low frustration tolerance. On the more positive side, some patients may gravitate to holding two jobs, or work in an environment where diversity prevails or where sustained attention is not a prerequisite.

Their pernicious social skills and inability to keep up appointments prevent enduring relationships, unless a stable, structuring partner assumes the role of an "executive prosthesis".

Their interactive styles are hallmarked by a variegated spectrum of personality traits. Indecisiveness and procrastination can mimic a neurotic disposition. Their inability to listen, and their tendency to run regrettable commentaries, give them a reputation

Table II		
Co-morbid disorder prevalence	(%)	
Unipolar depression	20-30	
Anxiety disorder	20-30	
Bipolar disorder	8	
Substance abuse/dependence	25-45	
Personality disorder	25	

of non empathic egocentricity. The never ending cascade of failures, missed opportunities, and a chronic awareness of "being different" has erosive effects on the self esteem. To compensate for profound feelings of inadequacy and incompetence, individuals with ADHD may develop a defensive grandiosity, masquerading as a narcistic personality. 6

Sensation seeking behavior can be understood as a coping style, because of the arousal it generates. The corrupted impulse control can translate into speeding citations, license suspensions and motor vehicle accidents. The compulsiveness these individuals sometimes display can also be interpreted as a coping strategy to combat inner chaos, and should be differentiated from obsessive compulsive disorder.

Severe mood swings, which can be rapid, often a few times within a day, may resemble rapid cycling bipolarity.

#### **Co-morbidity**

Approximately 70% of patients with ADHD have an additional disorder (Table II) making co-morbidity the rule rather than the exception. To differentiate these co-morbid conditions from ADHD can be a puzzling enterprise. ADHD starts much earlier and runs a persistent course as opposed to the co-morbid disorders which have a later onset and tend to have an episodic nature.

#### **DIAGNOSTIC TRAJECTORY**

There are no biological or psychological tests with sufficient sensitivity and specificity which can contribute to the diagnostic process. The diagnostic process comprises several components, based on a careful and systematic review of the clinical characteristics as solicited from different informants

An accurate diagnosis starts with a high index of suspicion. Individuals with any of the listed co-morbid conditions failing to respond to long term conventional therapies should be screened for a masked ADHD. The same applies to those who claim to experience a soporific effect after using cocaine, which alludes to the self medication hypothesis. Patients whose lives are hallmarked by chaos, or personality disorders whose background is devoid of developmental fractures or traumata, may also harbour an unrecognised ADHD.

The diagnosis is fundamentally based on the meticulous mapping of core symptoms, secondary problems, co-morbid conditions and ancillary data, initially obtained via a semistructured interview. It is essential that symptoms commenced before seven years of age and that the symptoms and cumulative impairments persist from childhood into adulthood. For every symptom pertaining to ADHD, its duration, intensity, associated impairment and longitudinal course should be determined. Since co-morbidity is rife, concurrent psychiatric disorders need to be assessed, and, if possible, the relationship of the ADHD-symptoms with these disorders delineated. Because adults suffering from ADHD are compromised in their retrospective recollections of symptoms, a host of spotty deficits may go undetected. To mitigate such recollection bias, collateral information is mandatory.

A perinatal history could highlight some of the environmental factors that may have contributed to the pathogenesis. Indeed, premature birth, maternal nicotine or alcohol abuse, placenta insufficiency and maternal hypertension have been associated with the generation of suboptimal attentional circuits.

In view of the strong genetic underpinnings, a family history may reveal a pedigree infested with chaotic individuals, who may have been treated erroneously for co-morbid conditions.

Old school reports often reflect these adults were labelled as lazy, restless or daydreamers. Neuropsychological investigation serves no diagnostic purpose at present as patients may be sufficiently aroused by the novelty of the setting. The subsequent ability to hyper focus allows them to perform satisfactorily. The most consistent deficits to date, are those referring to response inhibition tests and working memory. The chances that a patient presents with a substandard score are higher when he or she is fatigued or under aroused.

No pathognomonic features are to be ilicited during a mental status interview, but at times patients may present with verbal and motor unrest, nullifying a constructive dialogue by constantly interrupting the conversation.

Laboratory parameters are only required to verify the etiology of an acquired attention deficit. The morphological abnormalities and functional aberrations of the fronto-striatal circuits as revealed by neuroimaging studies are at this stage of no practical diagnostic value.<sup>1</sup>

Finally, the DSM-4 describes three subtypes. The inattentive, the hyperactive/impulsive and the combined type. To formalise the diagnosis, the symptoms can be checked against the operational inclusion criteria, but the nosological flaws that are inherent to the DSM, limit its usefulness as a diagnostic tool.

In short, the level of diagnostic confidence can be enhanced via thorough retrospective journey, confirming the early onset, longitudinal persistence, and multidimensional impairments.

#### **PITFALLS**

Successful treatment of ADHD in adulthood relies heavily on the accuracy of its diagnosis. It 's therefore important to appreciate the potential pitfalls that flummox the diagnostic climate.

There is a danger to medicalise buoyant individuals with a "strong joie de vivre". Normal people may experience attentional fluctuations, impulsive dyscontrol or hyperactive behavior during states of stress and exhaustion. These oscillations are transient as opposed to the protracted nature of ADHD. An inflation of the diagnosis can also be fuelled by patients who uncritically "recognise" the ADHD constellation via the press or the internet, and use the ADHD hype as an excuse for a life littered with self induced failures.

Underdiagnosis remains a vexing issue, especially if a syndromal definition of remission is in vogue. Patients with the ADD subtype-who lack the external behavioural component -go unnoticed for an extended period. Within the structured contours of the parental home, their cognitive handicap will be buffered , only to surface when the patient leaves home, and has to confront the multiple demands of life. Because of chronic forgetfulness , patients will underreport their juvenile symptomatology, which reiterates the fact that collateral data capturing is obligatory.

During times of arousal, the ADHD patient is able to hyperfocus, a situation that deceptively creates a sense of cognitive intactness. In other words, "they can do everything, but not every day".

Underreporting could also arise when the DSM taxonomy is uncritically adhered to.

If the cut off point —to meet six out of nine criteria-is applicable to both children and adults, many adults will go unreported, since some of their symptoms dilute over time. To avoid the risks of false negatives, it has been suggested to lower the cut off point to five out of nine criteria, thereby introducing an age adjusted qualifier.

ADHD exists along a continuum from mild to severe, a dimension that is lost in the categorical menu-style of the DSM-IV. By

using the DSM nosological system patients with a mild form of ADHD may therefore not be detected. The degree of impairment with the same symptom, may have more serious repercussions for adults than children. For example, pertaining to impulsivity, a child jumping a cue will be reprimanded, whereas an adult who impulsively jumps a stoplight may inflict a carnage.

Since the DSM-IV is insensitive to the wealth of life history data on which a solid diagnostic process is build, alternative classification systems, such as Hallowel and Ratey's, should be contemplated.<sup>7</sup>

### **DIFFERENTIAL DIAGNOSIS**

ADHD has no patent on the cardinal symptomatic core triad, since attentional deficit, hyperactivity and impulsivity are ubiquitous, featuring to some extent in most psychopathological syndromes.

Attentional problems often accompany affective, psychotic and substance use disordes, whereas hyperactivity and impulsivity are commonly encountered during maniform states, agitated depression, stimulant abuse and borderline personality disorder.

A proper history and physical examination, will determine whether an acquired attention deficit such as frontal lobe syndrome or foetal alcohol syndrome should be excluded.

#### **CONCLUSION**

Suffering from ADHD means patients will be more challenged than others in their quest to meet the demands of life. The diagnosis should be considered for any individual who manifests with a profile of global underachievement in the absence of a disjointed background.

Because the core clinical triad of inattention, hyperactivity and impulsivity is not disorder specific, a healthy degree of scepticism is required to prevent "ADHD- hyped" individuals to embark on a reprehensible self-diagnostic pathway that will fuel an epidemic wildfire.

Greater clinical awareness of the nature and co-morbidity will advance the therapeutic success rate of a neglected but treatable disorder.

# Acknowledgement

"I would like to thank Dr JJS Kooij whose academic zest inspired me to write this article.

#### References

- Zametkin AJ, Liotta W.The neurobiology of attention-deficit/ hyperactivity disorder. Journal of Clinical Psychiatry 1998;59 (suppl 7): 17-23.
- 2. JJS Kooij, JK Buitelaar, W Van Tilburg. Tijdschrift voor psychiatrie 1999; 41 (6):349-358.
- Biederman J, Mick E, Faraone SV.Age dependent decline of symptoms of attention deficit hyperactivity disorder: Impact of remission definition and symptom type. American Journal of Psychiatry 2000; 157(5):816-8
- JK Buitelaar, JJS Kooij. Ned tijdschrift Geneeskd 2000, 144(36): 1716-1723
- 5. Barkley RA. The executive function in ADHD.Paper presented at the CINP conference; July 9-13, 2000, Brussels, Belgium.
- 6. Gabbard G O. Psychodynamic psychiatry in clinical practice. Third edition. American Psychiatric Press Inc: Washington, DC, 1994.
- M Weiss,LT Hechtman, G Weiss: ADHD in adulthood: a guide to current therapy, diagnosis and treatment. John Hopkins University Press: Baltimore/London, 1999.

# **Commentary**

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There is a growing realization that Attention Deficit Disorders persist into adulthood for many individuals, and the insistence of many professionals that this is not the case, is counter productive. ADD in adults is a "hidden" disorder as the symptoms are often obscured by co-morbidity. They often present with issues of relationship dysfunction, substance abuse or problems with impulse control. In fact, poor impulse control (or impulse dyscontrol as it is called in the paper by Verbeeck) is one of the hallmarks of Attention Deficit Disorder in adults.

Furthermore low self-esteem, depression and social isolation may camouflage the underlying Attention Deficit Disorders. Their symptoms do not only affect them as individuals, but in adulthood it also affects their family, their friends and everybody they come into contact with. Many adults grew up in a time when clinicians and other professionals knew little about ADD, even less so about its diagnosis and treatment in adults. The DSM IV criteria are not very useful for diagnosis in this group but one of the hallmarks in adults is poor organizational skills and all the ramifications of this deficit in their daily lives. These secondary and co-morbid problems are well reviewed in the article.

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Although ADD is thought to have a prevalence of 3-5% in childhood, the impression in the United States is that it might be as high as 15%. This may imply that up to 5% of adults may in fact have this condition. The point made in the article that co-lateral information for diagnosis is mandatory cannot be stressed enough and an interview with the patient or client alone is not sufficient. The points made regarding neuro-psychological testing are also valid. It should be borne in mind that one of the characteristics of these adults is ostensible deterioration on psychometric tests, if they are repeatedly tested. This should not be seen as a deterioration of skills, but rather as boredom due to repetitive assessments.

The aberrations of the frontal striatal circuits, that are referred to in the article, may in future become an important diagnostic tool, but this still requires further investigation. Specific questionnaires for adults are being developed, but few of these have been standardized.

ADD should be identified in adults, because effective intervention can improve self-esteem, work performance and skills, educational achievement and social interaction. In the adult population stimulant medication alone is seldom beneficial. These patients also need education and counseling, as well as the management of their co-morbid disorders. Attention Deficit Disorders in adults need to be recognized and dealt with and should always be kept in mind when a cluster of recognizable symptoms in adults emerge, once presenting psychiatric signs had been dealt with.

**OPINION** 

# Progress in pharmacological treatment of bipolar disorder

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Recent years have seen an increase in awareness and interest in both the diagnosis and treatment of bipolar disorder. This has been driven in part by a re-evaluation of the concept of unstable mood and its significance in relation to diagnosis, and partly by an increase in interest in available pharmacological interventions for this group of disorders. Prof. Hagop Akiskal has been at the forefront of looking at broadening the concept and increasing the awareness of bipolar disorder and in particular the conceptualization of bipolar II disorder.

As a consequence of these developments we are, in clinical practice, more thorough in our evaluation of patients presenting with mood symptoms and are more frequently prescribing mood stabilizers in patients previously considered having either unipolar mood disorders or personality disorders (particularly cluster B).

Unipolar patients are found to be bipolar I on careful history taking and personality pathology (particularly cluster B) is now rather assessed as a bipolar II disorder as a result of this concept being broadened.

Currently available therapeutic options are varied. An optimal mood stabilizer would be required to meet the following primary considerations. It would need to effectively treat mania and depression, to prevent episodes of

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Dr F Daubenton e-mail: dauby@iafrica.com mania and depression, and should not induce mania or depression. Important secondary considerations would be effectiveness in rapid cycling and mixed state patients and reduction of residual symptoms. Currently no such drug exists. The currently accepted practice in treating the more severe cases of bipolar disorder is that several mood stabilizers sometimes with the addition of a novel antipsychotic are required to achieve the best possible response.

One of the most troubling aspects in treating patients with bipolar disorder is weight gain. The prospect of topiramate being an effective mood stabilizer with weight maintenance or loss is appealing. Lamotrigine is a valuable addition to the available drugs because of its antidepressant efficacy. A combination of lithium and lamotrigine is rapidly becoming the most popular mood stabilizer combination in clinical practice. This is because of the often debilitating consequences of mild persistent depressive states in patients with bipolar disorder stabilized on the traditional three mood stabilizers (lithium, sodium valproate, and carbamazepine). Gabapentin has thus far been found of value as an add-on or second mood stabilizer.

The advent of the novel antipsychotics, with their significantly reduced side effect profile, has been most welcome in the treatment of bipolar disorder. These patients have an increased risk for the development of most of the side effects of the traditional antipsychotics. Research is currently ongoing into the potential use of some of the novel antipsychotics as mood stabilizers and not just for the treatment of mania. I look forward to the results of this work with anticipation.