PSYCHOLOGY RESEARCH PROGRESS

New Developments in Clinical Psychology Research

Drozdstoj St. Stoyanov
Rolf-Dieter Stieglitz
Editors

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DROZDSTOJ ST. STOYANOV
AND
ROLF-DIETER STIEGLITZ
EDITORS

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Chapter 6

DIFFERENTIAL DIAGNOSIS OF ATTENTION-DEFICIT/HYPERACTIVITY DISORDER (ADHD)

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ABSTRACT

The hallmarks of Attention-Deficit/Hyperactivity Disorder (ADHD) are reflected in the symptom areas of inattention, impulsivity and hyperactivity. ADHD was long regarded as a childhood disease, but various studies have since found that adults are equally affected by the disorder. Differential diagnostic considerations, therefore, as well as the need for regard to potential comorbidity are of great importance for the diagnostic process. As possible examples are mentioned autism spectrum disorder, substance use disorders, schizophrenia, affective disorders, anxiety disorders and personality disorders. These groups of disorder form the subject of discussion in this chapter.

1. INTRODUCTION

The hallmarks of Attention-Deficit/Hyperactivity Disorder (ADHD) are reflected in the symptom areas of inattention, impulsivity and hyperactivity. ICD-10 subdivides these symptom areas into those of simple impaired activity and attention deficit disorder (F90.0: disturbance of activity and attention), hyperkinetic disorder with impaired social interaction (F90.1: hyperkinetic conduct disorder), other hyperkinetic disorders (F90.8 or F90.9) and attention deficit disorder without hyperactivity (F98.8). Compared to DSM-IV, DSM-5 no longer lists explicit subtypes for ADHD but instead focuses on three so-called „specifiers"

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based on predominant phenotypes, i.e., the combined presentation, the predominantly inattentive presentation and the predominantly hyperactive-impulsive presentation.

ADHD was long regarded as a childhood disease, but various studies have since found that adults are equally affected by the disorder. It is today believed that around 50% of those diagnosed with ADHD during childhood and adolescence continue to be affected by the disorder in adulthood. With a world-wide prevalence rate of 1-7%, ADHD represents one of the most common disorders in adulthood (de Zwaan et al. 2012; Fayyad et al. 2007; Kessler et al. 2006). Figures for prevalence rates vary considerably depending on the country and the samples researched. With a gender ratio of 1.6:1, more men than women are affected by the disorder in adulthood (Stieglitz et al. 2012).

In the context of phenomenology, the diagnostic criteria relate to symptoms which also feature as diagnostic criteria for other disorders or which can frequently appear as concurrent clinical phenomena. Thus, for example concentration difficulties represent a diagnostic criterion for manic and depressive episodes but can occur equally with many mental disorders (e.g., schizophrenia). This raises poignant questions in relation to the differential diagnostic process. In addition, ADHD shows a high frequency of comorbidity with a series of other disorders with estimated prevalence rates reaching a figure of as high as 80%. This is important because comorbid patients represent a substantial number of people in treatment and present greater disorder severity from both the clinical and social perspectives than those people diagnosed with only one type of disorder (Torrens et al. 2012: 1005).

Differential diagnostic considerations, therefore, as well as the need for regard to potential comorbidity are of great importance for the diagnostic process. DSM-5, for example, at criterion E carries a reference to the fact that ADHD symptoms are not exclusively particular to schizophrenia or other psychotic disorders but may be explained in the context of other mental disorders. As possible examples are mentioned autism spectrum disorder, substance use disorders, schizophrenia, affective disorders, anxiety disorders and personality disorders. These groups of disorder form the subject of discussion in this article.

2. DIAGNOSTICS OF ADHD

Compared to other disorders, the diagnostic process of ADHD in adulthood presents itself as rather more complex. This is not so much for psychopathological reasons but rather as a result of the lifespan perspective which warrants particular consideration, as is similarly the case with personality disorders. The following factors can in some cases render establishing a diagnosis difficult:

- The root of the symptoms must be sought in childhood. This is particularly important on account of the fact that many adult ADHD patients were never diagnosed during childhood or adolescence.
- As a consequence, the patient‟s own reliable personal memories or those of third parties involved (e.g., parents) play a significant role in the diagnostic process.
- Additional sources of data such as school reports must form part of the assessment process. School reports are, however, often not available or limited to lists of grades only which are of little value. The availability of detailed accounts of displays of

irregular forms of behaviour (e.g., disruptive behaviour in class, absent-mindedness) or performance levels (eg. concentration problems) is rare.
- Testing for potential comorbidities is essential.

Weiss et al. (1999) recommend various practical steps to be included in the diagnostic process which do not mandatorily need to be followed in the order listed here: the clinical interview, testing for ADHD symptoms through the implementation of rating scales, testing for other psychiatric disorders, testing for symptoms in childhood, further tests, and the recording of impairments.

Step 1: The clinical interview. The purpose of the clinical interview is to document the patient and their life history as well as their present life experience and behaviour. It serves to diagnose ADHD symptoms and other disorders and to evaluate the patient‟s current level of functioning.

Step 2: Testing for ADHD symptoms through the implementation of rating scales. Rating scales in the form of self-evaluation and third party evaluation procedures can facilitate the quantification of present and past symptoms.

Step 3: Testing for other psychiatric disorders. As already mentioned, prevalence rates of comorbidity with other psychiatric disorders are high. Comprehensive screening for other psychiatric disorders is therefore of primary importance. Where indications of other psychiatric disorders exist, diagnostic criteria of potential comorbid disorder(s) require thorough examination (e.g., in the form of diagnostic interviews).

Step 4: Testing for symptoms in childhood. A diagnosis for ADHD in adulthood is based on information relating to the patient‟s childhood. Adults diagnosed with ADHD often relate extremely vivid childhood memories. Noteworthy examples are e.g., emotional reactions of teachers and parents.

In cases where patients find it difficult to recall childhood memories, the examining clinician is forced to rely on external sources of information. Consulting school reports can in some cases yield fruitful results. The implementation of diagnostic scales may further assist the diagnostic process.

Step 5: Further tests. Although there are no compulsory testing procedures to assess a patient‟s levels of performance in order to establish an ADHD diagnosis, such tests can nevertheless deliver useful supplementary information. These include e.g., (neuro-)psychological examinations in relation to performance levels in either pencil-and-paper or computerised forms. Such tests, however, are no replacement for the clinical interview and the rating and recording of the psychopathology by means of specific scales.

Step 6: Recording of impairments. As ADHD is accompanied by various impairments, differentiated investigation into possible affected areas such as quality of life, family situation or work, personal relationships, education and training as well as activities in everyday life is necessary. Here, too, the use of rating scales may prove helpful.

As with other mental disorders, the starting point of the diagnostic process for ADHD lies in the careful recording of a patient‟s medical history, whereby with ADHD special regard must be paid to certain particularities and additional aspects. Table 1 summarises the most important areas in relation to medical history-taking as well as supplementary diagnostic findings.

The core areas of a medical history in ADHD cases are similar to those of medical histories in cases of other disorders (e.g., family medical history, physical illnesses).

Table 1. Diagnostic components of adult ADHD

■ complete psychiatric anamnesis
■ ADHD-specific contents of the interview
■ differential diagnoses and comorbidities
■ exclusion of organic mental disorders
■ internistic and neurological examination
■ interview with parents and/or care givers
■ complete ADHD related psychometric assessment
■ neuropsychological assessment

The following aspects require special attention:

- *(early) Childhood development:* e.g., complications at birth, irregularities observed already before school age (such as delayed development)
- *School:* e.g., problems during class and with homework, problems with discipline, behavioural problems, comments in school reports, early diagnostic assessment
- *Work:* e.g., training and education, recurring difficulties, change of work place

Psychological testing procedures can, in view of the assessment targets discussed, contribute usefully to the diagnostic process. In keeping with general conventions, a diagnosis for ADHD is secured at the start of a course of therapy. Although it is widely accepted today that a diagnosis ultimately represents a clinical diagnosis, the results of psychological examination procedures can contribute important elements. This is especially the case with procedures developed in close association with a classification system. Depending on the procedure, individual diagnostic symptom criteria are recorded and in some cases quantified, as are aspects of the impairment.

No direct diagnoses can be derived from neuropsychological procedures as there are, so far, no specific procedures available for this, and the multifarious facets of the disorder are not specifiable solely by means of neuropsychological procedures. The results of a neuropsychological examination can, however, yield supplementary information (see the sections below). Specific deficits become recognisable and quantifiable. Following the establishment of a diagnosis for ADHD and before medical regulation of the disorder with stimulants or other suitable substances, a thorough clinical somatic examination is required. This consists of a somatic medical history, a physical medical examination as well as laboratory tests. In addition, further examinations may be undertaken as deemed necessary.

Especially with adult ADHD patients, consideration must be given to a wider spectrum of potential comorbid disorders. Some of these may develop as secondary disorders as a result of repeated and sustained experiences of frustration and failure over many years (e.g., depressions, disorders provoked through psychotropic substance abuse). Other mental disorders also typically have their onset in adolescence or in early adulthood. Noteworthy are e.g., personality disorders, especially borderline personality disorder, but also schizophrenia. These may be comorbid but must equally be considered as possible differential diagnoses for other disorders. Adults in particular are also prone to suffer from physical illnesses which display ADHD-like symptoms such as hypothyreosis or hyperthyreosis, diabetes or certain heart conditions.

3. ADHD AND OTHER PSYCHIATRIC DISORDERS

3.1. ADHD and Autism Spectrum Disorders

3.1.1. Prevalence

In both ADHD and Autism Spectrum Disorder (ASD) cases the majority of children affected continue to fulfil the diagnostic criteria for these disorders also in adulthood. With ADHD, the percentage of cases where symptoms persist through adolescence and into adulthood lies between 50-70% (Rasmussen and Gillberg 2000; Biederman et al. 2007), with ASD at around 80% (Billstedt et al. 2005; McGovern and Sigman 2005), whereby the figures vary between studies and the development of individual cases depends on personal and environmental factors. Thus the probability of a reduction in the severity of autism symptoms during a lifespan in high-functioning autism or Asperger‟s syndrome patients respectively is markedly higher than in cases affected by other forms of autism (Kočovská et al. 2013; Helles et al. 2014; McGovern and Sigman 2005).

Prevalence rates of autism spectrum disorders (ASD) in children are estimated at 1% (Lehnhardt et al. 2013; Baron-Cohen et al. 2009) with no reliable estimates available so far for ASD in adulthood. ADHD with estimated rates of up to 7% (see Section above) is thus a more frequently occurring type of disorder than ASD. Among affected children both ASD as well as ADHD appear to be more commonly found in boys rather than in girls (Roy et al. 2009; Kohn and Esser 2008). Prevalence estimates for ASD conditions in childhood indicate that for every three diagnosed cases of children at primary school age two remain undetected (Baron-Cohen et al. 2009). This suggests that a large number of cases reach adulthood remaining undiagnosed. Especially autism sufferers with normal cognitive development can, on account of masking their difficulties through effective compensation strategies, remain clinically undetected over extended periods of time. It is frequently not until they face special life challenges beyond school age such as seeking employment or leaving the parental home that their limitations become evident. ASD, although a clinical condition, can, depending on symptom severity and level of cognitive ability, allow sufferers to successfully lead a largely clinically indifferent life. In DSM-IV and ICD-10, ASD and ADHD diagnoses remain mutually exclusive, as a result of which past literature largely ignored comorbidity with both these disorders. Only a small number of studies so far have investigated symptoms overlap and potential comorbidity of ADHD and ASD. The new DSM-5 permits both diagnoses simultaneously and thus also allows for possible comorbidity of both disorders.

3.1.2. Differential Diagnosis

Core symptoms of autistic disorders are 1) persistent impaired social communication and interaction in various contexts and 2) restricted, repetitive range of behaviour patterns, interests or activities. Concerning this point DSM-5 now also includes hyper- or hypo-reactive responses to sensory stimuli or unusually intense levels of interest in environmental stimuli. This stands in contrast to DSM-IV which, unlike DSM-5, attributes no significance to these issues. Additionally, DSM-5 treats autism as a spectrum disorder, which dispenses with the previously necessary differentiation between various subtypes such as Kanner‟s autism, atypical autism or Asperger‟s autism.

In diagnostic differentiation of ASD from ADHD, it is important to remember that with ASD, too, inattention remains an observed symptom (Roy et al. 2013; Lehnhardt et al. 2013). While with ADHD attention deficits occur predominantly as a result of increased distractibility in response to external stimuli, with ASD such symptoms occur as a manifestation of limited inner flexibility in adjusting to changed foci of attention. Equally, however, both cases may display an increased in-depth focus of attention in particular areas of interest which, in the case of ADHD, emerges as a hyperfocus on a specifically targeted interest area, whilst in ASD it manifests itself within the parameters of avidly pursued special interests. Impulsivity is a symptom observed in both ADHD and ASD. Whilst in ADHD impulsivity constitutes a core symptom, in ASD it may be explained as a result of previously experienced high levels of stress caused by, e.g., involuntary disruption to familiar patterns of behaviour or routine sequences of events. Difficulties at an interpersonal level of social interaction can be present in both ADHD and ASD, in the case of ADHD in the form of increased levels of impulsivity and attention deficits, in ASD through impaired cognitive empathy and social intuition. ASD may also lead to increased physical activity (such as rocking and shaking) which, as stereotypical and repetitive behaviour, may erroneously be interpreted as symptoms of hyperactivity attributable to ADHD. Sensory over-responsivity can be symptomatic of ADHD as well as ASD (in the case of ASD hyposensitivity is equally possible), whilst difficulties with motor coordination are further symptoms associated with both disorders (Roy et al. 2013; Lenhardt et al. 2013). An overview of symptoms particular to ASD and/or ADHD is provided in Table 2. Phenomenologically, ASD is distinguished from ADHD by the following symptoms (cf. Lehnhardt et al. 2013):

a) More severe problems at social and emotional interactional levels
b) More severely impaired verbal and non-verbal communication
c) Stereotypic or repetitive/ritualistic behaviours
d) Obsessive adherence to routines and rituals
e) Highly specific, fixated areas of interest, unusual in level of intensity or specificity of contents
f) Detail-focused perceptual processing
g) Compared to ADHD, in ADS more rarely observed higher levels of disorganisation as well as flightiness of thought and action

3.1.3. Comorbidity

In diagnosing ADHD or autism it is important to allow for the possibility that both these disorders may be comorbid, a fact which DSM-5 recognises. In 30-40% of adult ASD patients, diagnostic criteria for ADHD are fulfilled also (Hallerbäck et al. 2014; Rydén and Bejerot 2008; Rybakowski et al. 2014; Stahlberg et al. 2004). Amongst adult patients with a first-time ADHD diagnosis, too, a sub-group of patients is identified with comorbid ASD. In 15% of patients diagnosed with ADHD, Roy et al. (2013) found a comorbid occurrence of Asperger‟s syndrome. From a neurobiological viewpoint there clearly appear to be certain shared traits, although research findings in this area remain as yet vague and inconclusive (Gargaro et al. 2011).

Table 2. Symptoms in ADHD and Autism

Symptoms	ADHD		ASD	
	present?	reason	present?	reason
Deficits in social interaction	✓	Might be present in ADHD due to impatience, attentional problems, impulsivity. Empathy might be constrained by attentional problems.	✓	Core symptom of ASD Impaired cognitive empathy/Theory of Mind, deficits in non-verbal communication and socio-emotional interaction.
Repetitive or stereotypic behaviour	X		✓	Core symptoms of Autism Might be misinterpreted as ADHD symptoms.
Routines or rituals	X		✓	Core symptom of ASD
Special interests	✓	Possibly hyper-focusing on high-interest tasks	✓	Core symptom of ASD Highly restricted interests, that are abnormal in intensity or focus.
Sensoric hypo- or hypersensitivity	✓	Heightened sensitivity, e.g., towards sounds, due to increased distractibility.	✓	Lack of/increased sensitivity to sensory stimuli such as sounds, touch, pain, light.
Attentional problems	✓	Core symptom of ADHD Distractibility, especially due to external cues	✓	Low flexibility when shifting attention, heightened distractibility due to sensory stimuli and stress intolerance.
Hyperactivity	✓	Core symptom of ADHD	✓	Increased bodily movements in the context of repetitive or stereotypic behavior might be misinterpreted as ADHD-hyperactivity.

Table 2. (Continued)

Symptoms	ADHD		ASD	
	present?	reason	present?	reason
Impulsivity	✓	Core symptom of ADHD	✓	Might occur in case of involuntary disruption of habitual courses of events. Deviation of the status quo might cause stress and anger, which appears as impulsive behaviour.
Detail-oriented perception	X		✓	Impairment of central coherence
Motoric abnormalities	✓	Motoric hyperactivity and clumsiness might be present.	✓	Clumsiness might be present.

✓: present; X: not present.

A further debatable aspect is whether ADHD-like symptoms in autism cases do in effect point to comorbidity or whether they are, instead, indicative of, e.g., a specific subtype of ASD with ADHD-like symptoms (Sinzig et al. 2009).

Various studies demonstrate that ASD and ADHD are highly heritable neurodevelopmental disorders. Around 80% of phenotypic variance is attributable to genetic factors (Lichtenstein et al. 2010; Taurines et al. 2012). Family studies with ADHD patients show a familial aggregation of ASD symptoms within ADHD families. Studies on twin pairs suggest that 50-70% of the covariance of ADHD and autism symptoms is attributable to common genetic factors (Rommelse et al. 2010; Taurines et al. 2012). However, despite this highly heritable component, ADHD and autism patients do not necessarily remain permanently afflicted, since the brain displays high levels of neural plasticity also in adulthood which, under the influence of appropriate treatment or particular environmental factors, can enable neurodevelopmental disturbances to be outgrown (Ehninger et al. 2008).

So far, only scant research into the comorbidity between ASD and ADHD exists, with inconclusive results. Thus the question remains whether a comorbidity between ADHD and ASD is representative of an additive symptoms overlap between the two disorders, or, indeed, of a new distinct clinical condition. The former assumption finds support in the results of a meta-study, albeit limited to childhood cases (Berenguer-Forner et al. 2015), where a combined disorder shows a more severe clinical profile than that found in patients diagnosed with either ASD or ADHD only, which indicates a symptoms overlap between ASD and ADHD. Research by Nydén et al. (2010) contradicts these findings, however, where of the three patient groups investigated (ASD, ADHD, ASD + ADHD), it was the ADHD group

rather than the group with comorbid ASD + ADHD which emerged as the most severely affected.

3.1.4. Neuropsychology

ADHD and ASD are both characterised by deficits of executive function abilities. Executive functions comprise various mental operations such as switching focus of attention (set-switching), cognitive flexibility, initiation and choice of action, strategic action planning, self-monitoring and working knowledge (Leung et al. 2015). Such deficits result from dysfunctions in the frontostriatal circuit which impair goal-oriented behaviour (Gonzalez-Gadea et al. 2013; Gargaro et al. 2011). Abnormalities in frontostriatal brain regions and biochemical imbalances are thus suspected at the root of observed behavioural deficits in ADHD and ASD, which would explain the similarities between these disorders (Gargaro et al. 2011).

Differences in severity of symptoms become evident in so far as with autism, difficulties with cognitive flexibility and planning are most prominent, whilst the problem of impaired executive functioning tends to be absent (Gargaro et al. 2011; Ambery 2006). By contrast, an ADHD profile contains impaired executive functioning and working memory and not cognitive flexibility and planning as primary symptoms (Gargaro et al. 2011; Sinzig 2008). In the areas of social cognition and, in particular, Theory of Mind (ToM) the findings are contradictory. Some research points to impaired ToM in ASD patients, which appears less likely to be the case with ADHD (Gonzalez-Gadea et al. 2013; Lehnhardt et al. 2011). Other findings contradict this hypothesis (Nydén et al. 2010), albeit under caveat by those authors that a display of lack of empathy/affection in relation to ToM in the ASD group may have emerged as a result of flawed ToM test validity (Nydén et al. 2010). Further studies on ASD patients found deficits in visual memory functioning (Ambery et al. 2006), linguistic pragmatics and holistic perception (Lehnhardt et al. 2011).

3.2. ADHD and Substance Use Disorder

3.2.1. Prevalence

Psychotropic substance use disorders as defined in ICD-10, or substance-related and additive disorders as defined in DSM-5, form a heterogeneous group with widely differing estimates for prevalence rates. The variability in these rates partly stems from the differentiated sub-typification of the disorders according to substance type and respective complications associated with its use (e.g., in ICD-10 acute intoxication, substance abuse, substance dependence). Such worldwide variability disallows establishing standardised, reliable estimates for global prevalence. Problems related to alcohol use, however, are relatively consistently regarded as being of particular importance in the context of ADHD and comorbid substance use disorder. Alcohol use disorder is a common disorder on a worldwide scale. In the United States, for example, its prevalence rate amongst adults aged 18 and above is estimated at 8.5% (12-month prevalence; APA 2013). For other substances, prevalence rates are considerably lower, e.g., for cannabinoids 1.5% and opioids 0.37% (APA 2013). In comparison with figures for prevalence rates for ADHD, only estimates for the prevalence of alcohol use are higher, while all other substance-related and additive disorders rank decidedly

lower. From a differential diagnostic viewpoint, however, consideration of disorders related to psychotropic substances is essential, particularly in view of ADHD and its potential comorbidity with such disorders, especially in the case of alcohol use.

3.2.2. Differential Diagnosis

There are several challenges involved in establishing a diagnosis for ADHD and comorbid substance use disorder (SUD). Firstly, there is an overlap between a number of symptoms common to both disorders, as illustrated in Table 3, which warrants careful consideration. In addition, a further diagnostic problem emerges from the fact that patients are often unable to recall substance-free periods, which renders their ability to differentiate between individual symptoms difficult, not least because ADHD-like symptoms present also during episodes of substance withdrawal and abstinence. A course of detoxification treatment can further contribute to enhanced manifestation of ADHD symptoms. Ideally, a precondition for an ADHD diagnosis should therefore, wherever possible, be a completed course of detoxification therapy followed by a supervised sustained period of abstinence of a recommended minimum of 1 month. Also Tuckman (2007) confirms that in cases of prolonged and complex substance abuse it can become diagnostically difficult to determine which type of disorder a set of particular symptoms is attributable to. It would therefore be sensible to prioritise treatment for substance disorder and subsequently, after a 5-month period of abstinence, conduct a renewed assessment of the patient for ADHD symptoms. However, the proposition to defer a firm ADHD diagnosis until such time as successful completion of a prolonged episode of supervised sustained abstinence is attained remains, in a clinical context, often unrealistic. A more pragmatic approach would be an attempt to identify those symptoms which were attributable to ADHD before the onset of substance use, for which purpose the gathering and evaluation of information from third parties may become necessary.

3.2.3. Comorbidity

With the generally high rate of comorbidity in ADHD, comorbidity with psychotropic substance disorders is of particular interest. According to Wilens et al. (2003), untreated ADHD cases run a 1.5-fold increased risk of proceeding to develop substance abuse-related disorders. The significance of the combination of ADHD with substance disorders has been conclusively confirmed in a series of empirical studies. As well as other substances, this is especially the case in relation to alcohol.

Prevalence rates for comorbidity of ADHD with SUD are high, although the literature fails to present an entirely coherent picture. The National Comorbidity Survey Replication (NCS-R, cited in Mariani and Levin 2007) established a rate of prevalence for ADHD of 44%. Of the cases diagnosed with ADHD, 15.2% additionally met criteria for a SUD diagnosis; conversely, 10.8% of patients diagnosed with SUD were additionally found to be suffering from ADHD. In clinical treatment settings, these figures appear to be markedly higher. Torrens et al. (2012) identified 33% of ADHD patients as suffering from SUD, while 17-50% of SUD patients were diagnosed with comorbid ADHD.

Table 3. Clinical characteristics and outcome of patients with ADHD and SUD in relation to patients with ADHD or SUD

Earlier onset of substance use and abuse
More likely to initiate smoking and at a younger age, as well as become dependent on nicotine, and have more difficulty quitting smoking
More severity and chronicity of the addictive disorder
A more severe course of illness of ADHD
Less likely to achieve abstinence
Higher rates of poly-substance abuse
Increased psychiatric comorbidity
Lower treatment retention rates

(See Martinez-Raga et al. 2013)

In a clinical context, an analysis and explanation of the mechanics of the association between ADHD and comorbid SUD is highly desirable. Figure 1 contains an example which serves to illustrate a possible scenario for the development of the association between these two disorders over time.

ADHD is essentially a psychiatric disorder triggered by neurobiological factors, the symptoms of which become of relevance on their first manifestation in childhood (phase 1).

ADHD symptoms, which frequently fail to be diagnosed at their earliest stage of onset can, through aggregation of deficits over time (phase 2), result in a depressive episode (phase 3) during which a patient may resort to inadequate self-help and coping strategies in the form of substance use (phase 4). Such self-medication assumes particular significance for the adult patient, which is characteristic of that stage in the temporal developmental trajectory of ADHD.

Inattention is one of the symptoms which persist into adulthood, while childhood impulsivity and hyperactivity tend to decline with increasing age. In many adult patients, impulsivity and hyperactivity are replaced by increasing inner tension which can act as the catalyst for self-medication in the form of substance use. This is merely a simple, yet sufficiently plausible scenario and, particularly in a clinical setting, representative of an often observed combination of overall symptoms. It is important to remember that the sequences of symptoms development described here as phases 3 and 4 can, of course, occur in reverse order as well.

A further point is that this course of development is likely to manifest only in cases where ADHD failed to be recognised at an early stage of onset. Experience indicates, however, that in the context of adult psychiatry in special outpatient units, up to 90% of recorded cases were never previously assessed for or diagnosed with ADHD and had, correspondingly, never received appropriate treatment.

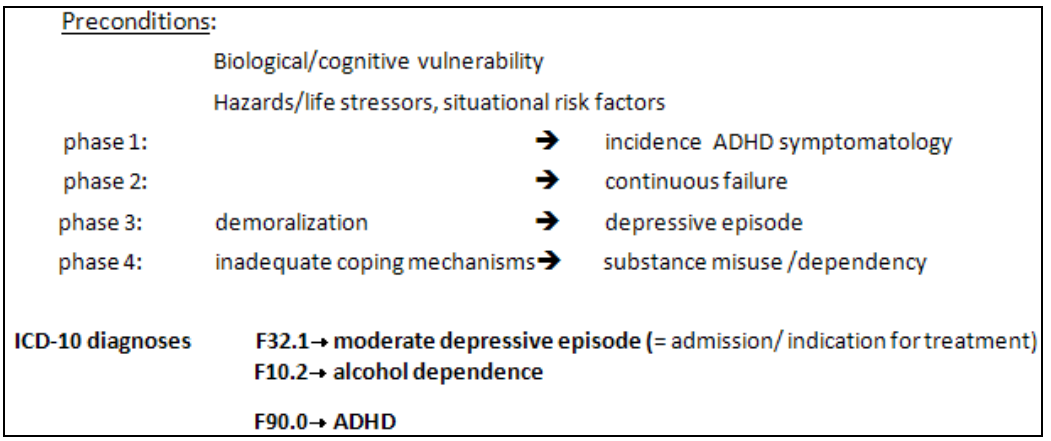

Figure 1. Example of the development of comorbidity in adult ADHD.

Studies on childhood patients (e.g., Biedermann et al. 2000) indicate that failure to recognise ADHD symptoms in children can already during childhood lead to severe complications, one of which is an especially early onset of alcohol use. According to Tuckman (2007), stimulant therapy for ADHD in children exercises a protective effect in relation to later substance abuse. The reasons for this are twofold in that, firstly, patients who benefited from stimulant therapy to treat their childhood ADHD are in a better position to master their adult lives and, secondly, as a result they are less likely to resort to self-help strategies in the form of self-medication and psychoactive substance use.

The various problems arising from a combination of ADHD and comorbid substance disorder, and their respective outcome variables, are summarised in Table 4. This overview demonstrates the significance of the various consequences resulting from this combination of disorders and the need for their inclusion in any considerations regarding possible courses of treatment.

It further emphasises the clearly enhanced complexity of the association between ADHD and substance disorder and the concomitant high demands put upon the clinician in the management and treatment of these.

In the context of establishing a clinical diagnosis, the following two possible scenarios are of relevance:

- a patient with suspected ADHD seeks clinical assessment with an additional requirement for screening for SUD;
- a patient suffering from SUD seeks treatment or, alternatively, is already in receipt of treatment, and has an additional requirement for screening for ADHD.

Given the high rate of comorbidity of ADHD and SUD, and the fact that ADHD symptoms manifest prior to substance use, a timely and accurate ADHD diagnosis is essential in order to initiate prompt treatment and prevent adverse developments. ADHD patients have a predisposition for early development of more severe instances of substance abuse and dependence disorders. Cases of alcohol dependence are often also accompanied by risk-related patterns and practices of substance use as well as a progression from legal to illegal

drug use. In all treatment settings where clinicians are confronted with potential ADHD cases, diagnostic precision and accuracy are of the essence.

Table 4. Symptomatology: overlapping aspects between ADHD and addiction

Components	psychopathology	clinical fatures
Substance effect	Acute intoxication or withdrawal symptoms	Agitation, mood instability, anxiety, intolerance, prone to frustration, restlessness, impulsivity, concentration, and memory difficulties
Addictive process	Craving and loss of control	Impulsivity, poor decision making, planning difficulties, mood instability, dysphoria, anxiety, continued substance use in spite of adverse consequences
Preexisting neuropsychological conditions	Deficits in executive functioning	Impulsivity, risk-taking behavior, inattention, inability to inhibit responses
Psychiatric comorbidity	Mood and anxiety disorders, borderline and antisocial personality disorders	Mood instability, anxiety, impulsivity, risk-taking behavior, intolerance to frustration

(see Fatseas et al. 2012).

It is imperative that patients diagnosed with ADHD are also screened for potential psychotropic substance disorders, for which a number of relevant screening instruments for ADHD as well as substance disorders are now available (e.g., Stieglitz and Raes 2015).

The importance of accurate diagnosis has been confirmed in various studies. McAweeny et al. (2010) for example point out that treatment programmes for psychotropic disorders often overlook ADHD symptoms. In their own study, McAweeny et al. further found that a mere 3% of the clinical medical histories of patients from his study contained an ADHD diagnosis which, however, in a comprehensive follow-up study, rose to an astonishing 44%. From these findings it may be concluded that there is, indeed, a pronounced risk that against the background of psychotropic substance use ADHD symptoms remain underdiagnosed, which is largely on account of the fact that the initial clinical picture obtained is more strongly defined by the symptoms relating to psychotropic substance use than it is by ADHD symptoms. According to Bukstein (2011) the cause for this often lies in SUD patients failing to be screened for ADHD symptoms, and in diagnoses for disorders such as depression and hypomania which serve to further obfuscate the path to an ADHD diagnosis. As a result, van der Glind et al. (2013) postulate that in view of the high prevalence of adult ADHD, all

patients seeking treatment for SUD should be screened for ADHD and, in the case of a confirmed diagnosis, receive treatment for ADHD in order to prevent unfavourable treatment outcomes which, as outlined in the literature, would otherwise be likely to follow.

3.2.4. Neuropsychology

In a clinical context, the various facets of executive functioning (EF) play an important role in ADHD (cf. also other sections). EF-related impairments such as organisation deficits are often the reason behind a first assessment for ADHD and are at the root of long-term problems that patients encounter in the workplace and in social relationships. Also of central significance are attention deficit related problems. In contrast to hyperactivity and impulsivity, these represent the most persistent phenomena over time in so far as hyperactivity and impulsivity tend to decline with age, while attention deficits persist. In addition, patients repeatedly mention numerous problems related to various memory processes such as general forgetfulness, forgetfulness in respect of received instructions, or forgetfulness in connection with task fulfilment requirements. Various meta-analyses confirm, in comparison with healthy controls, the presence of such deficits in ADHD patients (e.g., Schoechlin and Engel 2005).

A similar picture emerges in patients with psychotropic substance use disorders. It has been demonstrated repeatedly that one to two thirds of persons with chronic alcohol or other psychoactive substance use disorders show deficits in various cognitive areas such as memory, attention, executive functions, information processing and other cognitive abilities (Scheurich and Brokate 2009; Verdejo-García et al. 2004). As may be expected, most publications available on psychotropic substance disorders relate to alcohol use. As a result, the majority of studies on alcohol-dependent persons found memory tests to show either no impairments, or impairments of medium severity only. With regard to executive functions, most diagnoses in alcohol-dependent patients indicated impairment or limitations to at least parts of the areas of executive functions. Studies also found, however, that in some cases, after a given period of abstinence, it was possible for patients to go into remission (Pawlikoswki and Brand 2013).

In relation to other substances, Verdejo-García et al. (2004) showed that chronic heroin use leads to various cognitive impairments in the areas of executive functions (e.g., working memory), attention, impulse control and decision making behaviour. As concerns use of cocaine, their study contains few conclusive findings. They identified especially the areas of short-term memory, attention, inhibition control, abstract reasoning and psychomotor activity as being affected by chronic cocaine use. Concerning cannabis use, the available data remains inconclusive as well. Initial studies indicate minor impairments at the levels of executive functions, memory and attention. Whilst with most substances only chronic use leads to impairments, studies have shown that with MDMA (Ecstasy) cognitive impairments in the areas of memory and working memory become already evident with mere sporadic use. Further extensive studies are needed in order to arrive at conclusive results, yet it clearly transpires already now that the neurotoxicity of the various substances appears to lead to specific neuropsychological deficits, and that chronic substance use impairs cognitive functional abilities (Pawlikowski and Brand 2013). In summary, it is evident that both SUD and ADHD show impairments in various neuropsychological relevant domains which, in parts, share certain similarities (e.g., affected executive functions and, to some degree, memory).

On the one hand, the presence of neuropsychological symptoms underlines the relevance. On the other hand, however, on account of the similarities between the symptoms of ADHD and SUD, the application of neuropsychological assessment procedures for differential diagnostic purposes is called into question.

3.3. Schizophrenia

3.3.1. Prevalence

Compared to ADHD, the prevalence of schizophrenia is considerably lower. Lifespan prevalence rates are estimated at between 0.5% and 1.6%. The onset of schizophrenia is believed to occur between the ages of 15 – 35, with two thirds of patients showing first symptoms before the age of 30. According to recent studies, however, first indications of a possible onset of the disorder may show at a still younger age. Men tend to become affected three to four years earlier than women. The gender ratio between male and female patients is roughly even.

3.3.2. Differential Diagnosis

In daily clinical practice, the differential diagnosis for either ADHD or schizophrenia does not generally pose a significant challenge, although schizophrenia does explicitly feature as an exclusion criterion in DSM-5 (APA 2013) („symptoms do not occur exclusively during the course of schizophrenia," p. 60). There exist common features between both disorders, which, however, do not stand up to a more thorough exploration according to diagnostic criteria.

Initially, on first comparison of the two symptom areas, no overlap is recognisable (cf. Table 5). Whilst the symptoms of schizophrenia are primarily those of the classic psychopathological type such as delusions, hallucinations, ego disorders and thought disorders, the symptoms of ADHD tend to be more in the emotional and behavioural areas. This greatly simplifies the process of medical history taking.

Table 5. Symptomatology of ADHD and Schizophrenia

	ADHD	Schizophrenia
Core symptoms (diagnostic criteria)	Inattention	
	Hyperactivity	
	Impulsivity	
		Ego-disorders
		Delusions
		Hallucinations
		Thought disoders
		Catatonic behavior
		Negative Symptoms
Associated features (examples)	Inner restlesness	Cognitive deficits like memory problems, inattention

Problems are more likely to arise at the level of associated features of schizophrenia, where a symptoms overlap with ADHD becomes possible in the context of symptoms like attention deficits, inner tension and disorganised behaviour. Studies on the subject of ADHD and comorbid schizophrenia are rare, as the validity of the concept of comorbidity between the two disorders remains disputed.

3.3.3. Comorbidity

Brown‟s reader (2009), for instance, contains no section on ADHD and schizophrenia, and the term *schizophrenia* does not feature in the index. There is a small number of studies, however, which do point towards the existence of a possible comorbidity between schizophrenia and ADHD. Ross et al. (2006) conducted research focused on children and adolescents aged 4-15. In their sample of 82 schizophrenia patients, 84% also tested positive for ADHD. Unenge Hallerbäck et al. (2014), in their sample of 41 adult patients with schizophrenia, diagnosed 10% with additional ADHD. Differences in diagnostic approaches and methods in areas such as conceptual problems (e.g., analysis and evaluation) prevent conclusive answers in respect of comorbidity. However, there seems to be agreement that treatment of a potential comorbidity between these two disorders requires approaches to their management and treatment to be adapted accordingly. Thus, the treatment of ADHD symptoms with methylphenidate in the presence of potential comorbid schizophrenia is generally regarded as contraindicative (cf. Kraemer et al. 2010).

3.3.4. Neuropsychology

The perception of neuropsychological deficits in schizophrenic patients has been modified over the past few years. Whilst cognitive deficits were initially regarded as an epiphenomenon or associated with long-term pharmacological treatment, they are now considered as core deficits of the disorder, as endophenotypes of schizophrenic psychosis, and as such count as mediating factors between genotype and phenotype of the disorder (Pflüger et al. 2013).

Neuropsychological deficits are a frequent and characteristic feature of schizophrenic disorders. They are generally reliably assessed, as there is now an almost endless array of studies available on the various aspects of cognitive performance in schizophrenic patients.

Despite considerable heterogeneity in methodological approaches adopted in individual studies, and heterogeneity also in type and extent of cognitive deficits present in patient populations, the findings of existing meta-analyses are surprisingly consistant (Exner and Lincoln 2012). Across the whole spectrum of functional areas included in a variety of assessment instruments deployed for diagnostic purposes, the performance levels of schizophrenic patients are found to be roughly one standard deviation below the mean of that of healthy controls. It is questionable whether, against the background of such globally impaired cognitive functioning, it remains possible to identify areas which are selectively more heavily impaired. The discussion centres around whether, for example, verbal episodic memory, executive functions, and cognitive processing and encoding speeds respectively are especially heavily affected, and to what extent attention-related sub-areas such as those of selective, shared or sustained attention may be impaired (Exner and Lincoln 2012). Schaefer et al. (2013) have confirmed findings from past meta-analyses in a recent meta-analysis of their own. They were able to demonstrate that patients with schizophrenia showed considerably lower values than controls in all cognitive tests and all areas of cognitive

functioning (grand mean effect size, g = -1.03). The situation is thus of similar complexity to that of ADHD, in which partly similar symptoms present, with the result that neuropsychological procedures are likely to be less suited for differential diagnostic purposes. Direct comparisons between the two disorders remain, however, rare. Gansner et al. (in preparation) compared the neuropsychological performance of 233 ADHD patients against the performance of 127 ARMS (at-risk mental state) patients. In the *Continuous Performance Test* and the *California Verbal Learning Test*, the ADHD group performed significantly worse than ARMS subjects. In the Tower of Hanoi task, ARMS patients were markedly slower but made fewer mistakes than ADHD subjects. These results indicate that patients with ADHD perform considerably worse in tasks of executive functions, sustained attention and verbal learning and memory than ARMS individuals.

3.4. ADHD and Affective Disorders

3.4.1. Prevalence

Like ADHD, affective disorders, too, count amongst the most common psychiatric disorders (Kessler et al. 2009) and with their lifetime prevalence rate of 12% slightly exceed that of ADHD, which is estimated at a figure of 7%. Depending on the subtype of disorder, prevalence rates for affective disorders vary between 7.6 and 17.9%. In particular, the following lifespan rates of prevalence emerge: depressive disorders 4.0-10.0%; bipolar disorders 0.8-1.5% (for an overview, see Kessler et al. 2009). In industrialised countries rates for depressive disorders are higher than in the developing world, with women being almost twice as likely to be affected than men (1.7:1) (Kessler et al. 1993; Kessler et al. 2009). In contrast to ADHD, no such marked gender differences are recorded with bipolar disorders (Merikangas et al. 2007).

3.4.2. Differential diagnosis

Against the background of a high symptom overlap between affective disorders, in particular bipolar disorders, and adult ADHD, diagnostic differentiation between the two types of disorders in each instance of ADHD assessment is essential. Establishing a differential diagnosis between the symptoms of these two disorders remains, however, challenging, as both show a large overlap between at times barely distinguishable symptoms (see Table 6). A differentiation based on symptom areas and level of symptom severity becomes therefore only partly realisable.

Only certain symptom areas are involved in a symptoms overlap between depressive disorders and ADHD (in particular attention-deficit disorders and, to some extent, inner restlessness). As a result, symptoms such as severe depressive mood swings and profound loss of interest and inner drive, symptomatic of a depressive disorder, remain relatively clearly distinguishable from persistent hyperactivity and impulsivity found in ADHD (Kooij et al. 2012). By contrast, differentiation between ADHD and bipolar disorders with manic/hypomanic episodes proves more difficult, as these disorders show an overlap in various symptom areas. A differentiation of disorders on the basis of a differentiation between their symptoms only proves thus difficult (Asherson et al. 2014; Perroud et al. 2014; Skirrow et al. 2012).

However, with bipolar disorders and ADHD, too, specific characteristics are present in their respective symptoms which enable differentiation between them: whilst a reduced need for sleep, the presence of additional psychotic symptoms, inflated self-esteem and a subjective feeling of racing thoughts are specific to bipolar disorders, in ADHD low self-esteem and an increased distractibility are often dominant (Asherson et al. 2014; Perroud et al. 2014).

Table 6. Symptoms of ADHD, Major Depression and Bipolar I/II Disorder according to DSM-5

ADHD	Major depressive disorder (criteria of a major depressive episode)	Bipolar I/II disorder (criteria of a (hypo-) manic episode)
Inattention • Fails to give close attention to details/makes careless mistakes • Difficulty sustaining attention • Does not seem to listen when spoken to directly • Not follow through on instructions and fails to finish schoolwork, chores, or duties in the workspace • Difficulty organizating tasks and activities • Avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort • Loses things necessary for tasks or activities • Easily distracted by extraneous stimuli • Forgetful in daily activities	A-criteria: 8. Diminished ability to think or concentrate, or indecisiveness	B-criteria: 4. Flight of ideas or subjective experience that thoughts are racing 5. Distractibility
Hyperactivity and impulsivity • Fidgets with or taps hands or feet or squirms in seat • Leaves seat in situations when remaining seated is expected • Runs about or climbs in situations where it is inappropiate • Unable to play or engage in leisure activities quietly • "On the go", acting as if "driven by a motor" • Talks excessively	5. Psychomotor agitation or retardation nearly every day	6. Increase in goal-directed activity or psychomotor agitation 3. More talkaktive than

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ADHD	Major depressive disorder (criteria of a major depressive episode)	Bipolar I/II disorder (criteria of a (hypo-) manic episode)
• Blurts out an answer before question has been completed • Difficulty waiting his or her turn • Interrupts or intrudes on others		usual or pressure to keep talking
Additional symptoms • affective lability • Stress intolerance • Outbursts of rage • Sleeping disorders • Low self-esteem/low self-concept	1. Depressed mood most of the day 4. Insomnia or hypersomnia 7. feeling of worthlessness or excessive or inappropriate guilt 2. Markedly diminished interest or pleasure in activities 3. Significant weight loss	2. Decreased need to sleep 1. Inflated self-esteem or grandiosity 7. Excessive involvement in activities that have a high potential for painful consequences
	when not dieting or weight gain or decrease or increase in appetite 6. Fatigue or loss of energy 9. Recurrent thoughts of death, suicidal ideation, or a suicide attempt	
Progress • Several symptoms were present prior to age 12 years • Chronic progress: symptoms remain after onset (fluctuations in the severity of symptoms possible)	• Onset in childhood and adulthood possible • Incidence appears to peak in the 20s • Five or more symptoms have been present during the same 2-week-period • Change from previous functioning within a period	• Onset in childhood and adulthood possible • Mean age at onset of the first episode is approximately 18 years • Distinct periods of abnormally and persistently elevated, expansive, or irritable mood and abnormally and persistently increased goal-directed activity or energy, lasting at least 4 days/1 week

Note. Modified criteria of DSM-5 (APA; 2013).

In addition, consideration of temporal progression should also always form part of the differential diagnostic process: while affective disorders progress through clearly definable episodes of pronounced manifestation of symptoms, ADHD is a chronic developmental disorder with persistent symptoms (Asherson et al. 2014; Skirrow et al. 2012). Various

researchers regard careful and precise application of this criterion in the differential diagnostic process as imperative in order to enable differentiation between the symptoms of these disorders (e.g., Asherson et al. 2014; Perroud et al. 2014; Skirrow et al. 2012).

Differential diagnostic assessment should thus occur during the course of a euthymic phase (i.e., an interval of a balanced state of mood between manic/depressive phases). This approach should serve to diminish the potential for confusion and lead to more effective discrimination between the two types of disorder.

3.4.3. Comorbidity

Consideration must be given not only to the need for clear clinical differentiation between affective disorders and ADHD but also to the potential for concurrence of affective disorders with ADHD. The prevalence rates for comorbidity between ADHD and an affective disorder lie between 19% and 37% (Stieglitz et al. 2012), whereby comorbidity with dysthymia is particularly frequent. Different studies put prevalence rates for comorbidity of ADHD with depressive disorders at between 9.7% and 40.7% (Biedermann et al. 2008; Kessler et al. 2006; Pineiro-Dieguez et al. 2014). Still larger variability in comorbidity rates appear to exist between ADHD and bipolar disorders. In their review, Frías et al. (2015) report comorbidity rates of between 4% and 94% (with a mean value of 48%), depending on respective studies. They attribute such variability inter alia to starkly differing random sampling methodology adopted in the studies reviewed (e.g., outpatient vs. inpatient sampling; sampling of childhood onset bipolar disorder vs. adult bipolar disorder). According to Wilens et al. (2003), around 10% of ADHD patients develop an additional bipolar disorder, and 15% of patients with a bipolar disorder also suffer from ADHD. Later studies, too, yield equally inconsistent results. Di Nicola et al. (2014), for example, recorded in persons with bipolar disorders a prevalence rate of comorbidity between ADHD and bipolar disorders of 15.7%, and between ADHD and depressive disorders of 7.5%; whereas Pineiro-Dieguez et al. (2014) found in persons with ADHD a comorbidity rate between depressive disorders and ADHD of 9.7% and between bipolar disorders and ADHD of 2.5%. Studies further demonstrate a correlation between ADHD and comorbid bipolar disorders and an early onset of the bipolar disorder, a higher number of depressive and mixed episodes, fewer asymptomatic periods, worse outcomes and increased negative reaction to treatment (Karaahmet et al. 2013; Tamam et al. 2008). Furthermore, it has been shown that also the severity of ADHD symptoms during manic/hypomanic and mixed episodes becomes enhanced. Also, an increased number of comorbid disorders, in particular anxiety, substance use and borderline personality disorders, were found in patients suffering from both ADHD and bipolar disorders.

3.4.4. Neuropsychology

From a neuropsychological viewpoint, symptoms of all three disorders contain impaired executive functions which in depressive disorders remain largely restricted to affective episodes, whilst in ADHD they are of a chronic nature. Attention deficit problems in patients with bipolar disorders were, however, also found outside acute phases (for an overview see Hegerl et al. 2010).

Patients with ADHD as well as patients with bipolar disorders show similar cognitive impairments in various areas. In both groups, visual and verbal working memory and response inhibition are affected, while control and planning deficits are more associated with

bipolar disorders, and deficits in spatial working memory and language fluency are reported to be more specific to ADHD. Different studies arrive at partly contradictory results as to which areas of impairment are more likely attributable to which disorder, resulting in a disparate picture which serves to complicate rather than facilitate the application of neurobiological testing procedures in the differentiation between the two disorders (Skirrow et al. 2012). Over the past few years, research has begun to focus on the neuropathology of ADHD and bipolar disorders. Under discussion are, amongst other issues, a potential genetic, neuropsychological and brain structural overlap (Hegerl et al. 2010; Di Nicola et al. 2014; Skirrow et al. 2012). Present findings and results appear here, too, to project a rather heterogeneous picture; while some findings demonstrate agreement on neural correlates, others indicate differing neural correlates for the two disorders (for an overview, see Hegerl et al. 2010; Skirrow et al. 2012). Currently, depending on the type of disorder, an increasing number of findings seem to speak for the application of imaging technology in the detection of symptoms in different anatomical and functional regions of the brain (Biederman et al. 2008; Brown et al. 2012; Faraone et al. 2001). These findings lead to the conclusion that the pathogenesis of each of the two types of disorder tends to develop independently of each other. Nevertheless, above-average prevalence rates of comorbidity point at least partly to a common pathogenesis of the disorders.

3.5. ADHD and Anxiety Disorders

3.5.1. Prevalence

ADHD affects roughly 4-12% of school-age children and 2-6% of adults amongst the normal population. This incidence rates have found to be stable across different cultures (e.g., White and Shah 2006). While in certain cultures or occupations, a predisposition towards ADHD might not affect the individuals a lot and it might even be regarded as a gift, it usually clashes with the values and way of life in Western cultures. This is why many people with ADHD develop comorbid disorders over the life time, such as depression, low self-esteem and, last but not least, anxiety disorders.

The prevalence of anxiety disorders in the normal population lies between 5 to 15%, whereby the life time prevalence of the group of the disorder can be as high as 25%. Furthermore, they tend to be highly comorbid with each other as well as with other psychiatric disorders. In their review, Somers et al. (2006) calculated the pooled 1-year and lifetime prevalence rates for all anxiety disorders being between 10.6% and 16.6%, with an average life-time prevalence rate for panic disorder of 3.8%, agoraphobia of 3.4%, social phobia of 7.8%, specific phobia of 10.1%, Obsessive Compulsive Disorders (OCD) of 0.9%, General Anxiety Disorder (GAD) of 2.3% (Bijl et al. 1998, with a total life-time anxiety rate of 19.3%), and Post-Traumatic Stress Disorder (PTSD) of 7.8% (Kessler et al. 1994, with a total life-time anxiety rate of 24.9%).

3.5.2. Differential Diagnostics

The differentiation between ADHD and other disorders is not easy, as many psychiatric disorders result in symptoms of inattention, hyperactivity and/or impulsivity. A general rule of thumb in differentiating between ADHD and anxiety disorders are persistent fears and

worries accompanied by somatic symptoms in people with anxiety disorders, whereas ADHD subjects tend to be anxious in relation to specific academic or social situations in which they fear failure due to inattention or impulsivity (Quintana et al. 2007). More accurate methods include EEG measures, neuropsychological tests and highly elaborated psychometric assessment instruments. Table 7 provides an overview for discriminating between ADHD and anxiety disorders.

Turning to Electroencephalography (EEG) the most accurate measure in order to discriminate between ADHD populations and normal controls is the EEG theta-beta ratio, with a specificity as high as 94-98% (Monastra et al. 2001)**.** Barry et al. (2003) carved out three different EEG patterns in people with ADHD: a hypoarousal group, showing increased theta waves, but lowered beta waves; a hyperarousal group, with increased beta waves, but decreased theta waves; and, the biggest group with increased delta and theta waves, but lowered alpha and beta waves.

Although seemingly obvious, these three EEG groups have been found to not correspond to the three subtypes of ADHD. Rather, the three subtypes differ in the strengths of the EEG abnormalities, with the combined type showing the highest degree of abnormality, followed by the hyperactive type; the inattentive type displayed the lowest degree of abnormal EEG patterns. In anxiety disorders, EEG abnormalities highly differ from those in ADHD subjects. For panic disorders, for example, a low alpha wave with normally functioning beta, theta and delta waves seems to be typical (Enoch et al. 1995). In OCD patients, beta waves tend to be increased rather than decreased as in ADHD individuals (Hammond 2005). Moreover, anxious individuals tend to show a greater relative right frontal electroencephalographic (EEG) activity compared to normal controls (Blackhart et al. 2006). In contrast, an over-connectivity between the two hemispheres has been observed in ADHD (Murias et al. 2006). Often, ADHD subjects clearly show higher cerebellar and basal ganglia dysfunctions than subjects with anxiety disorders.

Besides, there exists a raw of assessment instruments for diagnosing ADHD. Hereby it is essential not only to examine ADHD specific symptoms in ADHD diagnostic procedures, but to ask for typical symptoms of other disorders, as well, for avoiding false-positives. In regards to anxiety disorders, one could include screening instruments such as the BAI Beck Anxiety Inventory (Beck and Steer 1993), the M.I.N.I. Mini International Neuropsychiatric Interview (Sheehan et al. 1998) or the K-SADS-PL Supplements for Anxiety Disorders and Affective Disorders (Quintana et al. 2007). Sensitivity and specificity for anxiety disorders have been found to be good or very good for the M.I.N.I., besides for generalized anxiety disorders; in the latter case, one might use the GAD-7 Generalized Anxiety Disorder (7 items) instead, with high sensitivity (89%) and specificity (82%) (Spitzer et al. 2007). For further investigation of all anxiety disorders, which is not too time-consuming, the anxiety section of the SCID I (structured clinical interview for DSM-IV, axis I) might be suitable, with high sensitivity and specificity.

3.5.3. Comorbidity

Schatz and Rostain (2006) speak of a 25% comorbidity rate of ADHD with anxiety disorder; hence, every fourth adult presenting with ADHD in our psychotherapeutic office is likely to have some kind of comorbid anxiety disorder. Anxiety disorders in general have been found to affect ADHD symptoms in specific ways, whereby:

Table 7. Differential features between ADHD and Anxiety disorders

ADHD	Anxiety disorders
• High impulsivity and low cognitive inhibition, esp. response inhibition	• problems with working memory and general cognition other than cognitive inhibition
• dysfunctional social behaviour: disrupted family and peer relationships	• socially phobics tend to focus on themselves and worry about embarrassment
• lack of planning and control of cognitive processes • disorganized general cognition as a result of lack of executive functioning (independent of external cues) • poor executive regulatory control • lower sustained attention • strong preference for immediate rather than future rewards	• planning and attentional shifting normal; spatial recognition, working memory and motor initiation impaired (esp. OCD)
• disruptive and inappropriate behaviour • anxious in relation to specific academic or social situations in which they fear failure due to inattention or impulsivity	• persistent fears and worries accompanied by somatic symptoms
• deficits in monitoring attentional resources • lower error monitoring; lower interference control	• no association between error monitoring and anxious behaviour
• attentional disengagement • spontaneous, impulsive decision making; delay aversion • emotional dysregulation, sudden mood swings, high moment-to-moment fluctuations • lower inhibition of a pre-potent response • higher-than-average abilities in creativity	• attentional hyper-focussing • slower decision making than normal controls • serotonine dysregulation, more permanent mood
• increased cerebellar dysfunction • Impaired basal ganglia higher motor speed, higher capability of motor fluency and accuracy and increased choreoform and athetoid movements	• lower verbal creativity in OCD than in normal control
• an impaired nucleus accumbens-striatum-orbitofrontal cortex circuit	• spatial working memory and spatial recognition impaired, motor initiation and execution slower than in normal controls; • lower visuo-constructional functioning • impaired *septo-hippocampal system*, *amygdala*, *hypothalamus and periaqueductal grey;* especially hyper-activation of the *hypothalamus–pituitary axis;*
• dopamine dysregulation • BAS system higher activated -> greater impulsivity and risk-seeking behaviour • general memory malfunctions due to storage and retrieval problems.	• acetylcholine dysregulation • avoidance of novelty seeking
	• Panic disorder with/without agoraphobia, social phobia, OCD: impairments in episodic memory.

See: Schatz and Rostain (2006); Quintana et al. (2007); Gupta and Kar (2010); Vance et al. (2006).

- impulsivity and response inhibition deficits are likely to decrease,
- working memory deficits and other cognitive problems usually get worse.

Therefore, although there is not a higher rate of anxiety amongst the inattentive subtype compared to the mixed or more hyperactive subtypes, anxiety does increase the symptoms in the inattentive subtype, rendering a tendency towards sluggish cognitive tempo even worse. This, in turn, feeds feelings of anxiety, insufficiency and depression, so that a vicious circle develops affecting many areas in life, which explains why comorbid anxiety in ADHD usually appears differently compared to more phobic types of anxiety seen in pure anxiety samples.

3.5.4. Neuropsychology

People with ADHD display specific deficits in monitoring attentional resources, which negatively influence response inhibition, error monitoring, attentional disengagement, decision making processes and emotion regulation (Gupta and Kar 2010).The core difficulty for ADHD patients has been proven to be sustained and selective attention, affecting the attentional subsystems of alerting, orienting and executive functioning. Especially, staying in an alert state without warning signals is almost impossible for individuals with ADHD. Deficits in attentional processes also involve higher-order processes such as inhibition of a pre-potent response, interference control and emotion regulation. Moreover, individuals with ADHD tend to show high moment-to-moment fluctuations, which not only explains their sudden, uncontrollable mood swings, but also signifies a considerable difficulty when assessing ADHD per psychometric assessment instruments. It is believed, that this deficiency is the result of abnormal error processing. Although delayed responses to errors have been suggested to be influenced by ADHD individuals‟ emotional states, there has been no association between error monitoring and anxious behaviour.

Neurophysiological, a central deficiency of ADHD lies in the under-activated anterior cingulate, which is the reason why neurofeedback therapy focusses on this brain area in particular (Drechsler 2011). This explains the vast amount of symptoms in ADHD, for example in the study of Wu et al. (2006) examining the differences between ADHD individuals and normal controls in delay aversion; here, it evolved that while in both groups the ventral and dorsolateral prefrontal cortices as well as the insula were activated, only the normal controls showed an activation of the anterior cingulate and the hippocampus. As a consequence, ADHD individuals not only have problems in decision making, but also in memory and emotional processes. ERP studies and experiments using the Attentional Network Task (ANT) or Go/NoGo tasks replicated abnormalities in alerting in terms of slower response times to abrupt visual cues as well as difficulties in prefrontal response regulation and dealing with conflicting stimuli (Müller et al. 2011). Moreover, recent research about ADHD focus on cerebellar deficits and its connections to the basal ganglia, thalamic nuclei, prefrontal cortex, primary motor cortex and varied brainstem structures. Vance et al. (2006) were able to provide evidence for an increased cerebellar dysfunction in ADHD (combined type) compared to anxiety disorders. In concordance with the observations that the linkage of the cerebellum to the basal ganglia is important to consider, the latter has been found to be stronger impaired in ADHD than in anxiety disorders, leading to a higher motor speed; this includes positive effects, as well, such as a higher capability of motor fluency and

accuracy and increased choreoform and athetoid movements, which might hint to the bio-evolutionary suggestion that the predisposition to ADHD might allow for better hunting abilities, having been an evolutionary advantage in former times. This is in concordance to the observation that in ADHD subjects, the behavioural activation system (BAS) is often higher activated accompanied by a greater impulsivity measure and a tendency to risk-seeking behaviour. In anxiety disorders, cognitive dysfunctions are very different from ADHD. For example, in OCD, planning and even attention are functioning normally, whereas spatial recognition and working memory as well as motor initiation (rather than inhibition, as in ADHD) are impaired. Social phobia has some overlaps with ADHD in terms of attentional problems, whereby socially phobics tend to focus on themselves, a problem not shared by ADHD patients. They also differ from ADHD populations in visuo-constructional functions. Moreover, OCD individuals usually display lower verbal creativity, whereas ADHD is characterized by higher-than-average abilities in creativity. Panic disorder with and without agoraphobia, social phobias as well as OCD are linked to impairments in episodic memory; ADHD individuals rather have problems with working memory (e.g.: Holmes et al. 2010). One of the main neurotransmitter involved in anxiety disorders is known to be serotonin, which also strongly influences our mood. Three main systems are involved in fear and anxiety: the *septo-hippocampal system*, the *amygdala* and the *hypothalamus and periaqueductal grey* (McNaughton et al. 2004). Further neurotransmitters involved in anxiety are acetylcholine and adrenaline; especially acetylcholine is responsible for common chronic stress-diseases due to a permanent hyper-activation of the *hypothalamus–pituitary axis*. On the contrary to ADHD patients, which display a tendency to spontaneous, impulsive decisions due to delay aversion, socially phobic individuals have difficulties in quick decision making and need more time for decisions than normal controls (Kaplan et al. 2006).

There are also common neuropsychological features in ADHD and anxiety disorders. Schatz and Rostain (2006) pointed out that a state of nonspecific arousal provoked by the lack of rewarding signals in ADHD might increase the disposition towards anxious reactions. Likewise, when stronger focussing on threatening stimuli, the BIS (behavioural inhibition system) tends to be higher activated in ADHD patients due to the inability to focus on relevant input. Obviously, also people with anxiety disorders are strongly BIS-orientated. Moreover, anxiety can evoke ADHD like symptoms via the neurotransmitter acetylcholine, which plays an important role in a range of cognitive domains; across empirical methods, working memory, attention, episodic memory encoding, and spatial memory processing are repeatedly, found to depend upon acetylcholine for normal functioning (Newman et al. 2012). In concordance with these findings, children of anxious mothers have a higher risk for developing ADHD (Perrin et al. 1996).

3.6. ADHD and Personality Disorders

3.6.1. Prevalence

Information on prevalence rates for personality disorders (PD) is sparse. Epidemiological studies on the prevalence of PD in the general population arrive at prevalence rates of between 4.4% and 15.7% (Coid et al. 2006; Lenzenweger et al. 2007; Samuels et al. 2002; Torgersen et al. 2001). In the US, estimated prevalence rates for the various types of PD as subdivided into the three DSM-IV/-5 Clusters, were 5.7% for Cluster A, 1.5% for Cluster B

and 6.0% for Cluster C (Lenzenweger et al. 2007). In the same study, 5.2% of the research population were found to meet diagnostic criteria for avoidant PD, 4.9% for schizoid, 3.3% for schizotypal, 2.4% for obsessive-compulsive, 2.3% for paranoid, 1.6% for borderline, 1% for anti-social, and 0% each for histrionic and narcissistic PD. With psychiatric patients, prevalence rates for PD of between 40% and 60% are markedly higher (Bohus et al. 2012; Newton-Howes et al. 2010). In a more recent study conducted in the United Kingdom, the following estimates of prevalence rates for PD in psychiatric patients were established: 16.5% for avoidant, 16.0% for dependent, 8.5% for borderline, 6.7% for obsessive-compulsive, 5.0% for paranoid, 3.2% each for anti-social and histrionic, and 2.8% for schizoid PD (Newton-Howes et al. 2010).

3.6.2. Differential Diagnosis

Viewed from a phenomenological perspective, borderline personality disorder (BPD) and anti-social personality disorder (ASPD) show a certain symptoms overlap with ADHD, particularly in respect of impulsivity and emotional dysregulation. For this reason, these two types of PD warrant special attention, as only a very limited amount of specific studies on remaining PDs in the context of ADHD is available to date. An overview of the symptoms overlap between BPD, ASPD and ADHD is contained in Table 8 below.

Similarly to BPD, adult ADHD, too, is characterised by impulsivity, extreme outbursts of rage or persistent inner tension, mood lability and emotional over-reactivity in stressful situations (Corbisiero et al. 2013; Matthies and Philipsen 2014; Prada et al. 2014; Wender 1995).

Additionally, low self-esteem and impaired social relationships are observed in both disorders (Matthies and Philipsen 2014; Newark et al. 2012). A marked difference between the symptoms of the two disorders is found predominantly in their respective onsets: whilst ADHD is a developmental disorder already diagnosable in childhood (before the age of 12), symptoms of BPD are first observed in adolescence, with a full diagnosis following in adulthood only (DSM-5). A further marked difference between the two disorders lies in a chronic suicidal tendency and a tendency to self-harm, both core symptoms of BPD (DSM-5). These symptoms play a minor role in relation to ADHD, where they are regarded as possible consequences of the disorder (e.g., within the context of a depressive disorder) rather than defining characteristics (Philipsen et al. 2006; Prada et al. 2014). In ADHD, the cardinal symptom is inattention, the presence of which is mandatory to establishing an ADHD diagnosis (DSM-5; Wender 1995).

A similar relationship is found between ADHD and ASPD in relation to symptoms of lack of impulse control, emotional dysregulation and impaired interpersonal relationship problems.

In addition, however, ADHD patients also show high-risk behaviour with a pronounced need to seek out novelty value (Philipsen et al. 2006), e.g., display of aggressive behaviour in road traffic (Barkley and Murphy 2010; Sobanski et al. 2013) and, in the case of prisoners with ADHD, delinquency (Gudjonsson et al. 2012; Moore et al. 2013; Storebø and Simonsen 2013) in the accompanying absence of any sense of guilt or remorse (DSM-IV/-5).

Table 8. Symptomatic overlap and shared features between ADHD, BPD and ASPD (DSM-5; Utah-Criteria by Wender 1995)

Symptom/feature	ADHD[1]	BPD[2]	ASPD[3]
Inattention	✓		
Hyperactivity	✓		
Inner restlessness	✓	✓	✓
Impulsivity	✓	✓	✓
Emotional dysregulation	✓	✓	✓
• Affective lability	✓	✓	✓
• Hot temper	✓	✓	✓
• Stress intolerance	✓	✓	✓
Disorganisation	✓		✓
Interpersonal deficits	✓	✓	✓
Low self-esteem	✓	✓	
Risk behaviour	✓	✓	✓
Non suicidal self-injuries		✓	
Chronic suicidality		✓	
Chronic emptiness		✓	
Dissociation		✓	
Absence of repentance			✓
Substance abuse	✓	✓	✓

Note. [1]Attention Deficit Hyperactivity Disorder; [2]Borderline Personality Disorder; [3]Antisocial Personality Disorder.

✓: present.

3.6.3. Comorbidity

Prevalence rates for PD in ADHD patients are as high as 64.3% (Miller et al. 2008). In their recent study, Vidal et al. (2014) found that 24.78% of ADHD patients also met the diagnostic criteria for a PD. Cumyn et al. (2009), in a larger study with 335 ADHD patients, identified an overall figure of 50.7% of patients with a PD, with breakdown percentages of 27.68% fulfilling the criteria for compulsive PD, 24.11% for BPD, 12.54% for avoidant PD, 12.50% for narcissistic PD, and 5.07% for ASPD. The remaining types of PD, however, were also found concurrent with ADHD. In Williams et al. (2010) for example, 11% of their small sample of 47 ADHD patients tested positive for depressive PD, 9% each for paranoid and passive-aggressive PD, 6% for dependent PD and 2% for histrionic PD. Matthies et al. (2011) also found schizoid PD in 6.6%, and schizotypal PD in 6.7% of their research population 0). In general, patients tested positive for more than one PD (cf. Matthies et al. 2010; Williams et al. 2010). This leads to the conclusion that it is predominantly PD types from DSM-IV/-5 Clusters B (dramatic-emotional) and C (anxious-avoidant) which tend to co-occur with ADHD (Matthies et al. 2011).

Two types of PD which are closely linked to adult ADHD are BPD and ASPD. These two disorders show considerable overlap with ADHD both in respect of symptoms as well as at a neurobiological level. The more consistent prevalence rates for comorbidity are found between BPD and ADHD and are estimated at between 18.3% - 38.1% (Cumyn et al. 2009;

Ferrer et al. 2010; Instanes et al. 2013; Matthies et al. 2011; Miller et al. 2008). The estimated prevalence rate for comorbid ASPD among ADHD patients of 40.3%, however, was even higher (Instanes et al. 2013). In criminal offenders with ADHD the estimated percentage rate is reported to be higher still (cf. Gudjonsson et al., 2012; Moore et al. 2013).

Philipsen et al. (2008), in their study on a clinical population, reported that 41.5% of women diagnosed with BPD had displayed ADHD symptoms in childhood, with 16.1% proceeding to meet the diagnostic criteria for ADHD. The study also suggested that the higher the number of current adult BPD symptoms patients were able to relate, the greater the likelihood of a former presence of childhood ADHD in these patients, and that, as a consequence, childhood ADHD might pose a possible risk for the development of BPD in later life. Compared to patients diagnosed with BPD only, ADHD patients with comorbid BPD were reported to be at an increased risk of developing suicidal tendencies and additional mental disorders such as anxiety and substance disorders as well as ASPD. ADHD patients with comorbid BPD also told of higher incidence rates of emotional abuse than did patients diagnosed with BPD only (cf. inter alia Storebø and Simonsen 2014). In comparison with BPD patients and a healthy control group, ADHD patients displayed more BPD symptoms than the healthy controls. Self-harming behaviour and emotional dysregulation were, however, less frequently observed in ADHD patients with comorbid BPD than in patients diagnosed with BPD only (Philipsen et al. 2008). Findings of further studies also indicated that ADHD patients with comorbid BPD showed higher levels of impulsivity and aggressive behaviour than patients diagnosed with either ADHD or BPD only, or healthy controls (Prada, et al. 2014). A connection between ADHD and BPD was also found at a genetic level (Distel et al. 2011) with an estimated phenotypic correlation between ADHD and BPD symptoms of $r = .59$, of which 49% were attributable to genetic factors.

The development of ASPD is an aspect under consideration in connection with ADHD and social conduct disorder (SCD). Childhood ADHD with or without comorbid SCD appears to enhance the overall clinical symptomatology and with greater probably leads to the development of a later ASPD (Retz et al. 2013; Storebø and Simonsen 2013). Children with ADHD and comorbid SCD run an additional enhanced risk of slipping into crime and delinquency at a later stage in their lives (Storebø and Simonsen 2013). Emotional neglect, parental divorce, early separation from primary carers or attachment figures as well as past suicide attempts appear to be more frequent in persons with ASPD and comorbid ADHD; especially the level of impulsivity appears to feature as a prominent factor in the dynamics of the association between these two disorders (Storebø and Simonsen 2013).

3.6.4. Neuropsychology

The described symptoms overlap between ADHD, BPD and ASPD also manifests itself in a neuropsychological context. The neuropsychological deficits common to all three disorders are located in the prefrontal cortex (the core region of attention mechanisms) and in the orbito-frontal cortex (the core region of impulsivity and emotional dysregulation) (Dolan 2012; Philipsen 2006). ADHD as well as BPD patients display impaired impulse control and appear to suffer also from response inhibition, particularly in stressful situations (Krause-Utz et al. 2013; Matthies and Philipsen 2014; van Dijk et al. 2014). Impulse control in BPD patients, however, becomes particularly impaired in the face of emotional and interpersonal relationship difficulties or in the presence of a comorbid ADHD (Sebastian, Jacob, Lieb and Tüscher 2013). While with BPD patients lack of impulse control indicates primarily

prefrontal impaired functioning in orbito-frontal, dorsomedial and dorsolateral brain areas, impaired activity in ADHD patients tends to be more concentrated in ventrolateral and medial prefrontal regions (Sebastian et al. 2014). Only few studies so far have compared the neuropsychological mechanisms of ADHD and BPD. Krause-Utz et al. (2013), in their study of various groups (BPD, BPD with and without ADHD, ADHD, and healthy controls), found that response inhibition (assessed through the *Immediate and Delayed Memory Task*; Dougherty and Marsh 2003) was particularly impaired in patients with BPD and co-occurring ADHD. These findings, however, stand in contradiction to a study by van Dijk et al. (2014), who observed response inhibition (assessed through the *Expectancy AX-CPT Task*) in both ADHD as well as BPD patients (regardless of whether comorbid BPD or ADHD respectively was present or not).

Emotional dysregulation has, until now, been generally defined as core to BPD symptoms (Matthies and Philipsen 2010) and has only in the past few years become the subject of more intensive research in the context of ADHD (Corbisiero et al. 2013). In ADHD, inattention appears to play an important role in the regulation of emotions, yet the neuropsychological mechanisms of the correlation between inattention and emotional dysregulation remains still unclear (Shaw et al. 2014). In the case of BPD, however, it is assumed that in the regulation of emotions, attention deficits play a less decisive role (Lampe et al. 2007).

The neuropsychological deficits in persons with ASPD are, similarly to ADHD, present in executive functions, although empirical evidence to this effect remains scarce: in particular, difficulties with planning ability, behavioural inhibition and set shifting were observed (Dolan 2012). A recently published study on ASPD patients and ADHD with ASPD patients investigated their ability to recognise emotions (joy and happiness, sadness, fear, disgust, anger, surprise, and neutral facial expression), which was measured through the *Emotion Recognition Test* containing facial expressions from Ekman and Friesen‟s series (Ekman 1999). The two groups displayed significant differences only in respect of the ability to recognise the emotion of disgust (Bagcioglu et al. 2014).

SUMMARY AND PERSPECTIVES

The clinical diagnostics of ADHD represent a complex process. In contrast to most other mental disorders, establishing a differential diagnosis in ADHD cases necessitates consideration of a lifespan in its entirety (not dissimilar to personality disorders). There is therefore a plethora of information to be gathered, to which end an increasingly large variety of assessment tools have become available. As well as information from patients themselves and from third parties, various additional sources of data require consideration and evaluation in the process. The diagnostic criteria, which meanwhile have been adapted in DSM-5 to adulthood, are less difficult to explore than in other disorders, making hardly any psychopathological knowledge necessary. Yet, partially overlapping symptom areas and the potential comorbidity of ADHD with other disorders pose particular challenges in establishing a differential diagnosis. On the one hand, the results of a variety of studies containing comprehensive accounts of clinical experiences offer a foundation upon which to base a reliable diagnosis. Conversely, however, a host of questions still remains unanswered.

In particular with regard to the relationship between ADHD and schizophrenia, further empirical research is required to shed light on the many open issues involved.

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