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Sleep Disorders Diagnosis and Management in Children with Attention Deficit/Hyperactivity Disorder (ADHD)

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1. Introduction

ADHD is an increasingly prevalent developmental disorder, especially in the western world where prevalence rates are estimated around 12%.

According to both the DSM-IV and the ICD-10 it refers to three major problematic domains: attention, hyperactivity and impulsivity. Subtypes of ADHD have been coded accordingly as a predominantly hyperactive-impulsive type (H), predominantly inattentive (I) and a combined type (C).

Age is an important factor in the clinical/behavioral manifestations of ADHD. Symptoms, in fact, vary according to brain maturation. The hyperactive aspects, for instance, tend to subside with age, even if longitudinal research demonstrated that over 30% of children with ADHD grow up to be adults with significant ADHD related problems.

Gender also plays an important role in ADHD, with a 1:10 male prevalence in clinical samples (Cortese et al., 2006). It also appears to have a strong influence over behavioral symptoms and co-morbid disorders, which appear generally less disruptive in girls.

ADHD holds a high potential for psychiatric and cognitive co-morbidity (mood, anxiety, conduct disorder and learning disability), with a nearly 50% rate of oppositional defiant conduct in males.

2. ADHD and sleep

Sleep disturbance is by far one of the most reported problems (>80%) especially by parents and care-takers, who commonly recount restless, inadequate and often delayed and/or fragmented sleep in their children.

A clear distinction, however, needs to be drawn between subjective and objective sleep reports and, with respect to the latter, actigraphic versus video-polysomnographic (vPSG) studies present a palpable difference in terms of method and quality of data accessed.

Several metanalytic reviews have been published within the last ten years, dealing with many confounding factors including gender, referral source, age range, co-morbid disorders, first versus follow-up visits, medications, number of studied nights with or without adaptation. (Owens, 2005; Cortese et al., 2006; Sadeh et al., 2006).

They all address, through various approaches, the multilevel/dimensional relationship among sleep alterations and neurobehavioral/neurocognitive functioning.

In particular, the interaction of sleep with attention/arousal mechanisms in children has been highlighted by most recent studies.

2.1 Subjective reports

Items more often referred to by subjective studies on sleep and alertness in ADHD include (Cortese et al., 2006): bedtime resistance, sleep onset insomnia, night awakening, sleep duration, restless sleep, parasomnias, problems with morning awakening, sleep disordered breathing (SDB) excessive daytime sleepiness (EDS).

No major differences between adolescents with ADHD and controls were detected (Mick et al., 2000) after excluding confounding factors such as medications and psychiatric comorbidity. As for ADHD children, significantly over reported by comparison with controls were EDS (Marcotte et al., 1998, Owens et al., 2000), whether or not sleep disordered breathing (SDB) related, movements during sleep (Corkum et al., 1999, Owens et al., 2000). Also a longer sleep duration with increased night awakenings and parasomnias were observed upon comparison with controls (Owens et al., 2000). Despite the fact that bedtime resistance and sleep onset insomnia did not come across as significantly different by comparison with control subjects, after controlling for psychiatric co-morbidity and medications, it cannot be ruled out that a subgroup of ADHD children may display significant difficulties with sleep onset. Endogenous circadian alterations have been postulated by several authors (Vander Heijden et al., 2005) along with forced ultradian cycling (Kirov et al., 2004), which would make these children more prone to a delayed sleep phase (DSP). In this respect, melatonin use before bedtime with different regimen schedules and dosing has been acknowledged by several clinicians (Hodgkins et al., 2011; Owens et al., 2010; Larzelere et al., 2010).

2.2 Objective studies

Only a few actigraphic studies (Wiggs et al., 2005; Dagan et al., 1997; Corkum et al., 2001) and few video PSG studies were obtained in ADHD children, probably due to objective constraints as imposed by health policies, children restless and oppositional behavior and parents' reticence to over-night hospitalization. Concerning actigraphic studies, measures of objective sleep patterns (sleep duration, activity mean, wake time and number of awakenings) resulted not predictive of ADHD symptom severity after regression analysis (Wiggs et al., 2005) and did not correspond to parents' reports except for waking time in the morning. In particular, bedtime for the IADHD children was usually grossly underestimated by their parents, probably because of less externalizing behaviors during daytime.

Correspondence between subjective and objective assessment has been usually inconsistent with a few exceptions (Acebo et al., 1999). In fact, despite an overall very high parental report of sleep disturbance, actigraphic data did not confirm parents' concerns. Of course, some of these results could rely on the inherent incapacity of actigraphy to confirm specific sleep disorders such as SDB or sleep fragmentation at a microstructural level. Nevertheless, these results may still prove of some utility in order to provide parental reassurance and correct some of their distorted beliefs.

3. Sleep architecture

Sleep architecture with phase distribution, wake time, arousal and sleep fragmentation could be seen at best only via PSG studies. A major issue in this context is the exclusion of

confounding factors such as the effect of medications, co-morbid neurological and psychiatric conditions and, above all, primary sleep disorders. In fact, ADHD children could be generally subdivided in children without sleep disorders, probably less than 50% according to most estimates, and children with sleep disorders.

Many authors claim that in the absence of an abnormal apnea hypopnea index (AHI) or periodic leg movements (PLMs) index, sleep variables in ADHD children are not far from age normative values (Sangal et al., 2005). In a recent thorough metanalysis of polysomnographic (PSG) studies, Sadeh et al. (2006) examined other factors of variance, including age and gender. Age, in fact, reflects maturational changes of neurobehavioral and neurotransmitter systems, which may deeply influence sleep patterns.

Females with ADHD usually present less disruptive behaviors, which can also differentially influence sleep attitude and propensity.

Whether or not an adaptation night is performed, it may enable the exclusion of the effects of first night sleep deprivation and adaptation to the lab conditions.

All considered, total sleep time appears to be longer in comparison to controls in ADHD children who underwent an adaptation night. The same subjects also exhibited longer stage 2 than controls (Sadeh et al., 2006); there appears to be a gender-related effect also over time spent in stage 2, but the most consistent effect of TST and stage 1 time was age-related, with a shorter TST and longer stage 1 in younger children (<9 years) compared to older children (>9 years), as if to indicate a more severe sleep impact in the younger group, which is usually also more severely affected in terms of ADHD symptoms, especially as far as hyperactivity is concerned.

A critical review (Bullock & Schall, 2004) examining dyssomnia in ADHD children, reports an overall concordance between authors (O'Brien et al., 2003a; Miano et al., 2006; Silvestri et al., 2009) with respect to a decreased REM percentage and an increased REM latency in ADHD kids. Most reports from France, however, contradict these studies (Lecendreux et al., 2000, Konofal et al., 2001). Furthermore, these data are not confirmed in most studies on HADHD children with nighttime periodic limb movements (Crabtree et al., 2003), as if the REM effect were more consistent with the IADHD type.

Kirov et al. (2004), instead, noticed an increased duration of REM sleep and of the number of sleep cycles in ADHD children compared to controls. Also, REM latency resulted shorter in his subjects as already previously reported by Kahn (1982) and Greenhill (1983), as if a forced REM initiation may have produced a longer REM sleep duration along with an increased number of sleep cycles.

A decreased dopaminergic activity in ADHD may be responsible for cortical dysinhibition of the motor frontal cortices, which would in turn result in the forced ultradian cycle of ADHD with REM-increased propensity. Later on, the same group reported an increased REM drive with shorter REM latency in children with coexisting tick disorders and ADHD, ascribing this type of impact to hypermotor symptoms (Kirov et al., 2007), partially contradicting previous findings (Crabtree et al., 2003) on reduced REM sleep in ADHD children with periodic limb movements.

3.1 Microstructural aspects of sleep

What almost all authors agree about is an increase of sleep oscillations within the night that contribute to the overall daytime "hypoarousal" phenotype via a possible decrease of sleep efficiency (Gruber et al., 2007). However, while a few authors notice an increase of

spontaneous or event-related arousals in their subjects' PSG (Silvestri et al., 2009; O'Brien et al., 2003a), arousals have mostly not been formally identified or reported in ADHD PSG studies. Rather, an increased number of phase shifts has been reported (Miano et al., 2006) with the same clinical significance.

The only dedicated paper in terms of a formal approach to explore the microstructural aspects of sleep in ADHD has been written by Miano et al. (2006) who analyzed the cyclic alternating pattern (CAP) in ADHD children without abnormal AHI or PLMs index. The authors reported an overall reduction of CAP rate, an index of sleep instability, in comparison to normal controls, with ongoing reduction of CAP sequences and A1 index, reflecting hypersynchronous delta waves with a protective effect on sleep continuity. This paper would then reconcile the increased fragmentation and low efficiency seen by other authors in ADHD sleep, with the relative compensatory increase of A2 and A3 subtypes, expressing sleep discontinuity.

A striking CAP similarity between ADHD and narcolepsy (Ferri et al., 2005) has been observed along with increased daytime somnolence on multiple sleep latency tests (MSLT). The latter observation is of seminal importance for the interpretation of ADHD as a primary disorder of vigilance (Weinberg & Brumback, 1990). A deficit of the arousal level fluctuations would underlie the concept and clinical considerations which tend to interpret ADHD as "a hypoarousal state" despite its contradictory daytime paradoxical hyperactivity. Further detailing of this theory and related studies are to follow in the sleep disorders section under "Narcolepsy".

4. Sleep disorders

4.1 Insomnia

Chronic sleep onset insomnia (SOI) is a frequent finding in ADHD children (Mick et al., 2000; Smedje et al., 2001; Corkum et al., 2001; Owens et al., 2000a; O'Brien et al., 2003a) with a prevalence rate of nearly 28% in unmedicated children (Corkum et al., 1999), almost double than the corresponding rate in the normal child population (Owens et al., 2000b; Meijer et al., 2000). Its daytime sequelae heavily impact the cognitive domain of children and, specific to this age group, also behavioral attitude and social conduct. Hyperactivity in fact, rather than overt EDS, is the general marker of insufficient sleep in most children, therefore aggravating the typical features of ADHD (Wiggs & Stores, 1999).

SOI in ADHD was demonstrated to co-occur with a delayed dim-light melatonin onset and sleep-wake circadian rhythm, whereas sleep continuity proved unaffected (Van der Heijden et al., 2005). These findings suggest a possible disturbance of the circadian pacemaker which, in turn, would be due to the alteration of clock genes (Archer et al., 2001), but no clear evidence has been found to confirm this assumption. Sleep hygiene habits of unmedicated ADHD children with SOI were later compared to those of ADHD subjects without insomnia, so as to ascertain whether poor sleep hygiene could at least partially explain insomnia in the affected group (Van der Heijden et al., 2006). The negative results of this study suggest that sleep hygiene practice is not related to sleep characteristics in ADHD children and does not differ significantly whether or not children complain of insomnia.

As early as 1991, Dahl et al. submitted a 10-year-old girl with ADHD and a long-standing SOI to chronotherapy, obtaining a significant improvement of the ADHD-related symptoms along with circadian sleep-phase advancement.

Subsequently, Ryback et al. (2006) confirmed these results in 29 ADHD adults with an open trial of bright-light therapy in the morning.

Interestingly, the reverse relationship does not appear to occur since ADHD symptoms are not commonly found in DSP. Therefore one might assume that SOI per se, as typical of DSP, is not enough to produce daytime feature of ADHD unless accompanied by nighttime hyperactivity/sleep fragmentation, as in most ADHD children (Walters et al., 2008).

As for sleep maintenance insomnia (SMI), several primary sleep disorders such as obstructive sleep apnea syndrome (OSAS), periodic leg movement disorder (PLMD), and restless legs syndrome (RLS) concur in increasing wakefulness after sleep onset (WASO) with night-time awakenings and lowering sleep efficiency with related detrimental effects on performance (Gruber et al., 2007).

These aspects will be developed later on in the course of this chapter, within their respective sections.

Early morning insomnia is not typical of children with ADHD, unless severely depressed and is described only in older groups of ADHD patients.

4.2 Excessive Daytime Sleepiness (EDS) and narcolepsy in ADHD

As previously reported in this chapter, the “hypoarousal state” theory regarding ADHD claims that there may be decreased sleep oscillations (CAP) in ADHD children without major co-occurring sleep disorders, and this might explain their daytime drowsiness as confirmed by MSLT data and, indirectly, by the Epworth Sleepiness Scale (ESS) results in ADHD adults (Oosterloo et al., 2006).

On a modified version of MSLT, ADHD children were found to be objectively more sleepy than controls, albeit a higher rate of OSAS in probands with respect to controls (50% vs. 22%) could represent a strong bias in this study (Golan et al., 2004).

Also, Lecendreux et al. (2000) found a shorter mean sleep latency in ADHD children compared to controls, with a significant correlation to hyperactivity impulsivity and inattentive-passivity indexes, as measured by Conners Parent and Teacher rating scales (CPRS & CTRS respectively).

Differences in nocturnal sleep could not account for any of these results. None of these authors, however, sought for the instrumental hallmark of narcolepsy (2 Sleep onset REMs) in their studies, nor reported on the clinical secondary features of narcolepsy (cataplexy, hypnagogic hallucinations and paralysis) in ADHD children.

The fact that reaction time values in these studies (Lecendreux et al., 2000) hold a negative correlation with the hyperactivity-impulsivity index of the CPRS indicates that a major distinction must be drawn between IADHD and HADHD. Generally, only a subgroup of ADHD children are found to be sleepier than most average ADHD subjects, thus suggesting an impaired control of their arousal system, which may induce them to switch rapidly from wakefulness to sleep when insufficiently externally stimulated during daytime (Ramos Platon et al., 1990).

A consequent implication of this theory would be an improved therapeutic option through the employment of noradrenergic α -1 agonists (Biederman & Spencer, 1999) such as modafinil, rather than the commonly used dopaminergic stimulants.

In a more recent study (Prihodova et al., 2010), SL on MSLT in unmedicated ADHD children exhibited significant time-related changes compared to the control group, but no inter-group differences were found regarding MSLT mean sleep latency when comparing the whole ADHD group with controls.

Likewise, no such differences were found when comparing different subgroups of ADHD with or without SDB/PLMs.

Again, a dysregulated arousal mechanism could be postulated in the absence of an overall objectively proven daytime sleepiness in ADHD.

Conversely, as for signs of ADHD in narcoleptic patients, memory, attention and executive functions were found to be affected in most, but not all, studies (Naumann & Daum, 2003). In particular, divided attention and complex cognitive tasks prove selectively sensitive to arousal fluctuations in narcolepsy (Hood & Bruck, 1996). However, automatic but not hyperactive behaviors have been reported or searched for in narcolepsy research in an attempt to allow a better clinical comparison between the two disorders.

4.3 Sleep apnea (OSAS)

Most of the recent attention to the ADHD co-morbidity with OSAS came from a paper reporting the impact of adenotonsillectomy on adverse events and behavioral problems in SDB children (Li et al., 2006). The authors described an overall improvement of all measures [AHI, tests of variable attention (TOVA), and child behavior check list (CBCL) scores] with no correlation, however, between AHI and TOVA values.

The correlation between OSAS and ADHD is difficult to explore, due to several confounding factors such as age, gender, recruitment sources and methodological approaches including the definition and the measurement of respiratory events in children (Sadeh et al., 2006).

Cortese et al. (2006) found an AHI 1/hour greater than controls in children with ADHD.

Race plays also an important role in the association between ADHD and OSAS. Hispanic children show a greater co-morbidity than Caucasian probands as far as learning problems, snoring and witnessed apneas (Goodwin et al., 2003).

Also, Huang et al. (2004), reported worse attention deficit and higher hyperactivity on the CBCL of ADHD with SDB compared to ADHD without SDB children, with an overall 57% of elevated AHI (>1) in their total group of ADHD subjects, against an AHI (>1) in only 4% of their control group. Both the cranio-facial predisposition to OSAS and the high prevalence of ADHD in Taiwan could, however, impede a generalization of these results. Adenotonsillectomy more than pharmacological treatment with stimulants lead to a favorable outcome in the same group (Huang et al., 2007).

Interestingly, most researchers agree on the association of only mildly severe OSAS to ADHD (O'Brien et al., 2003b; Sangal et al., 2005; Silvestri et al., 2009) suggesting that SDB leads to a mild mimic of ADHD, rather than a true form of it.

Possible mechanisms accounting for the association between ADHD and OSAS are intermittent hypoxia and sleep fragmentation which could both be responsible for neurochemical alterations of the pre-frontal cortex and their related effects including executive dysfunction with emotional lability and impulse control disorders (ICD).

Severe OSAS in the pediatric community is rare and usually linked to EDS rather than to hyperactivity, with a phenotypical change of behavior from mild to severe ADHD. It is unclear so far whether SDB may contribute only to mild ADHD mimics or really impact ADHD clinical expression and therapeutic management. While it is known that surgical treatment of OSAS may improve ADHD symptoms, no classic non-stimulant drugs (atomoxetine, clonidine, modafinil) used for the management of ADHD (Walters et al., 2008) induce a parallel improvement of OSAS.

4.4 Sleep related movement disorders

4.4.1 Periodic Leg Movements (PLMD) and RLS

Symptoms of ADHD have been found in 44% of children with PSG evidence of PLMs (Crabtree et al., 2003). Conversely, between 26% and 64% of ADHD children have a PLMs index $>5/h$ of sleep (Picchietti et al., 1998; 1999). Furthermore, between 44% and 67% of the positive probands have a parental history of RLS, suggesting a possible genetic link between ADHD and RLS/PLMD.

When an additional co-morbidity with SDB was reported, the link between PLMs and ADHD appeared to be stronger, mediating the secondary SDB-ADHD association (Gaultney et al., 2005).

The reported prevalence of RLS in ADHD children ranges from 44% (Cortese et al., 2005) to no association at all (Gamaldo et al., 2007). Silvestri et al. (2009) reported that an RLS prevalence of 12% by interview and clinical criteria was increased to 25.4% after PSG evaluation, reflecting the frequent difficulties to elicit an appropriate history from children depending on their age-related verbal abilities.

PLM during wakefulness (PLMW) but not RLS itself were associated to lower ferritin values, unlike previous reports (Konofal et al., 2007, 2008; Oner et al., 2008). In particular, lower ferritin values in ADHD children, whether or not RLS+, were reported by Konofal et al. (2004), compatible with the ADHD dopaminergic dysfunction hypothesis. Iron, in fact, is a co-factor of the rate-limiting enzyme, tyrosine-hydroxylase, regulating dopamine synthesis.

Cerebrospinal fluid (CSF), Magnetic Resonance Imaging (MRI) and autoptic studies also proved reduced iron stores in the brain of RLS subjects.

Iron deficiency could therefore represent a link and an interpretation key to the dual pathology of RLS and ADHD related disorders (Cortese et al., 2008), D1 and D2 receptor density being also altered by an iron-deficient state (Walters et al., 2000; Konofal et al., 2008). RLS was found to be significantly associated with H- and CADHD, rather than IADHD, with a strong impact on CPRS, CTRS and SNAP IV Teacher and Parent Rating Scale (Swanson, 1992) hyperactive and oppositional scores (Silvestri et al., 2009).

Preliminary data on dopaminergic treatment of ADHD-associated RLS suggested a dual improvement of RLS and ADHD symptoms (Walters et al., 2000). However, these results were not further replicated (England et al., 2011) since dopaminergic treatment of a larger number of ADHD-RLS+ children led to RLS improvement without any change in the ADHD-related symptoms and scores.

Iron supplementation (Konofal et al., 2008), or most recently levetiracetam (Gagliano et al., 2011) seem to hold promising results for the management of RLS in ADHD.

Even if it is not yet clear whether RLS and ADHD share a common genetic basis or have a distinct pathogenesis with one disorder (RLS) mimicking or leading to the other (ADHD), it is certainly important to look for RLS-related aggravation of ADHD symptoms to address separate or additional treatment of sleep related symptoms.

4.4.2 Rhythmic Movement Disorder (RMD)

RMD consists of head banging or body rocking behaviors primarily occurring in young children prior to sleep onset or during subsequent sleep.

The disorder often disappears with age over 18 months, but it is estimated to last sometimes in adolescents or adults with psychiatric problems or epilepsy (Simonds & Parraga, 1984; Mayer et al., 2007). Other PSG studies in pediatric populations disclosed RMD in 6/10 ADHD children, mostly with CADHD (Stepanova et al., 2005).

A percentage (21.8) similar to that of RLS (25.4) was reported in a group of 55 unmedicated ADHD children evaluated by means of PSG (Silvestri et al., 2009) co-occurring with other sleep related movement disorders, in particular PLMD and bruxism, but not SDB, at odds with previous records (Mayer et al., 2007).

A functional impairment of the pre-motor and striatal circuitry akin to that responsible for RLS could be hypothesized as a link between RMD and ADHD.

4.4.3 Bruxism

Highly co-occurring with other sleep related motor disorders such as PLMD, RLS and RMD, bruxism has also been described in ADHD reports, both subjectively and after PSG confirmation (Silvestri et al., 2009) up to 33% of the studied ADHD sample and with a gender distribution (female prevalence) confirming data in the general population (Hojo et al., 2007).

A dopaminergic dysfunction has already been accounted for this disorder (Lavigne & Montplaisir, 1994) and treated accordingly.

4.5 Parasomnias

Only few subjective studies report an overall increased number of parasomnias in ADHD children (Owens et al., 2000; Gau, 2006; Kraenz et al., 2004), whereas most studies (Corkum et al., 1999; Mick et al., 2000) notice no difference between ADHD children and controls after accounting for co-morbidity and pharmacotherapy.

As far as objective studies are concerned, they mostly do not report about parasomnias with few exceptions (Miano et al., 2006), accounting for a maximum of 35% of generic parasomnias overall.

An increased prevalence of sleep talking (O'Brien et al., 2003b; Corkum et al., 1999) and enuresis is reported (O'Brien et al., 2003a; Kaplan et al., 1987; O'Brien et al., 2003c).

As far as REM-related parasomnias are concerned, an increased prevalence of nightmares in ADHD children was reported by three studies (Owens et al., 2000; O'Brien et al., 2003b; 2003c). Disorders of arousal (DOA), which include sleep walking (SW), night terrors (NT) and confusional arousals (CA), albeit optimal candidates on the basis of familiar predisposition and SDB common association for a possible ADHD co-morbid occurrence have rarely been reported in ADHD subjects.

An early report (Ishii et al., 2003) found an overall low incidence (2.9%) of NT and SW in ADHD children, mean age 9.7 years. Most patients had mild ADHD, all being out-patients studied on the basis of subjective reports with an overall low co-morbidity load compared to Western countries.

On a broader sample of ADHD pediatric subjects, Gau et al. (2007) found an OR of 2.4 (95% CI 1.3-4.5) among subjects with definite ADHD, and 1.8 in probably ADHD probands (95% CI 1.4-2.3) between NT and inattention, with lower OR values between NT and hyperactivity. Similar results, not as strong, were reported for SW.

As a result of low CAP with decreased A1 sequences (Miano et al., 2006) suggested a possible disorder of arousal with a tendency to hypoarousability in their ADHD children who had otherwise no SDB nor other objectively identified sleep disorders.

On clinical interview Silvestri et al. (2009) found a 50% prevalence of DOA in a group of 55 ADHD children (28.5% CA, 47.6% SW, 38% NT), whereas on PSG, CA were recorded in 45.2% of their patients, SW in 2.3% and NT in 4.7%.

One patient reporting both dream enactment and SW episodes had a PSG evidence of CAs and REM without atonia, thus matching criteria for parasomnia overall disorder according to the International Classification of Sleep Disorders (ICSD-2).

Interictal epileptic discharges (IEDs), mostly on centro-temporal or frontal leads were seen in >50% of these unmedicated ADHD subjects and among them, in >40% of the DOA+ children, with nocturnal hypermotor seizures occurring in three children, none of which had ever presented evidence of diurnal paroxysmal disorders.

Complex behaviors during the DOA episodes were, however, easy to distinguish from nocturnal seizures in these children, even when co-occurring with IEDs.

Vulnerability of ADHD children to rolandic seizures and foci is well known (Holtmann et al., 2003), along with an increased rate of DOA in patients with benign focal epilepsy of childhood.

A positive significant association of DOA with SDB in the form of snoring and with increased sleep instability was also described by the same authors (Silvestri et al., 2009), akin to previous reports emphasizing the same associations (Lopes & Guilleminault, 2006; Guilleminault et al., 2005).

A preferential impact on the cognitive domain rather than behavioral indicators is most typical of children with DOA and slow wave sleep (SWS) dysfunction, opposite to the effect of nocturnal hyperactivity which seems to preferentially influence daytime hyperactivity and oppositional behaviors.

Levetiracetam 750-1000 mg/day effectively controlled seizures and lead to total cessation of DOA with a >50% reduction of IEDs during a follow-up period of 24 months (Walters et al., 2008).

5. Therapeutic management and options to address co-morbid sleep disorders in ADHD

The effect of immediate (IR) or extended (ER) release stimulants in ADHD is well known and beyond the purpose of this review.

Stimulants still represent the first line of treatment of ADHD in pediatric populations across the world.

The majority of subjective report studies indicate increased parental complaints of sleep disturbance in medicated versus unmedicated ADHD children, irrespective of stimulant type or regimen (Cohen-Zion & Ancoli-Israel, 2004). However, objective studies, whether actigraphic or PSG, show overall conflicting results as far as sleep measures, continuity and architecture, major differences going in opposite directions with regard, in particular, to REM sleep (Chatoor et al., 1983; Greenhill et al., 1983); no influence, though, on specific sleep disorders such as SDB or PLMD.

A consistent co-morbidity with depression in many ADHD children could account for increased subjective and actigraphically confirmed sleep fragmentation in the most severe cases.

Besides stimulants (Smoot et al., 2007), nonstimulant drugs have been successfully employed for the treatment of ADHD including atomoxetine (Kemner et al., 2005), bupropion and now less commonly used, tri-cyclic antidepressants. Clonidine, Guanfacine and other adrenergic α -1 agonists along with modafinil might help ADHD children with the hypoarousal phenotype, whereas SSRIs and venlafaxine could be used to fight depression/anxiety-related sleep symptoms. Also, atypical anti-psychotic drugs such as

risperidone (Reyes et al., 2006) might be employed to counteract conduct/behavior disorders as aforementioned several melatonin trials have addressed rhythmicity disorders and SOI, whereas levetiracetam, an anti-epileptic drug with antimyoclonic properties has been employed either in ADHD-RLS+ children or to treat DOA, seizures and related IEDs in ADHD children.

6. Conclusions

Sleep disorders comorbid to ADHD may aggravate cognitive and behavioral impairment in affected children.

They also severely affect parental negative attitudes and wellbeing.

Addressing and fostering a correct sleep diagnosis may add to the therapeutic benefit obtainable in ADHD children. In fact, a differential diagnosis according to subgroups of children in relation to their phenotypic sleep expression, allows to better characterize children and parental needs and to implement oriented therapeutic options. Future research needs to address better care and divulgate easy diagnostic means (Holter-PSG and self-administered questionnaires) for a home-based monitoring of children in their natural environment.

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For progress to be maintained in a clinical field like sleep medicine, unimpeded, unrestricted access to data and the advances in clinical practice should be available. The reason why this book is exciting is that it breaks down the barriers to dissemination of information, providing scientists, physicians, researchers and interested individuals with a valuable insight into the latest diverse developments within the study of sleep disorders. This book is a collection of chapters, which can be viewed as independent units dealing with different aspects and issues connected to sleep disorders, having in common that they reflect leading edge ideas, reflections and observations. The authors take into account the medical and social aspects of sleep-related disorders, concentrating on different focus groups, from adults to pregnant women, adolescents, children and professional workers.

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