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# Is there a specific polysomnographic sleep pattern in children with attention deficit/hyperactivity disorder?

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**SUMMARY** The aim of the study was to characterize the sleep pattern in children with attention deficit/hyperactivity disorder (ADHD). By means of polysomnography (PSG), sleep patterns were studied in 17 unmedicated preadolescent boys rigorously diagnosed with ADHD and 17 control boys precisely matched for age and intelligence. Although ADHD children did not display a general sleep alteration, major PSG data showed a significant increase in the duration of the absolute rapid eye movement (REM) sleep and the number of sleep cycles in ADHD group when compared with controls. In addition, REM sleep latency tended to be shorter in ADHD children. These results suggest that in ADHD children, a forced REM sleep initiation may produce a higher incidence of sleep cycles and may also contribute to an increased duration of the absolute REM sleep. The overall pattern of the findings implies that a forced ultradian cycling appears characteristic for the sleep in ADHD children, which may be related to alterations of brain monoamines and cortical inhibitory control accompanying the ADHD psychopathology.

KEYWORDS attention deficit/hyperactivity disorder children, brain monoamines, cortical inhibition, rapid eye movement sleep, ultradian cycling

## INTRODUCTION

Attention deficit/hyperactivity disorder (ADHD) is one of the most common and socially important child psychiatric disorders. Major symptom domains of ADHD such as inattention, impulsivity, and hyperactivity (Swanson *et al.* 1998) have been associated with region-specific changes in dopaminergic and noradrenergic neurochemical modulation in the brain (Arnsten *et al.* 1996; Ernst *et al.* 1998; Johansen *et al.* 2002; Pliszka *et al.* 1996), as well as with a decreased inhibitory control of the cerebral cortex (Berger and Posner, 2000; Castellanos, 1997; Moll *et al.* 2000; Rothenberger, 1990). In a complex association with cortical processes of inhibition and activation, monoaminergic brain systems have also been assigned to play a central role in the regulation of sleep (Gottesmann 1999; Hobson *et al.* 1975; Muzur *et al.* 2002; Pace-Schott and Hobson, 2002). Hence, their functional alterations may also affect the sleep pattern in ADHD.

Behaviorally, many ADHD children experience chronic sleep problems and difficulties with adequate daytime alertness. These include reluctance or inability to settle into sleep, difficulties in initiating and maintaining sleep, frequent awakenings, higher incidence of restless sleep, nightmares and night terrors, and insufficient daytime alertness (Ball *et al.* 1997; Kaplan *et al.* 1987; Kostanecka-Endress *et al.* 2000; Marcotte *et al.* 1998; Owens *et al.* 2000; Ring *et al.* 1998; Simonds and Parraga, 1984). These sleep difficulties have been suggested to be more than just a symptom of ADHD or a consequence of treatment with stimulants. Rather, they have been assumed to reflect an underlying condition contributing to behavioral

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symptoms of ADHD and their persistence into adulthood (Brown and McMullen 2001). However, it is not precisely known whether sleep problems are intrinsic to ADHD because of a specific monoaminergic dysregulation and an imbalance in cortical inhibition/activation, or whether they are primarily associated with a range of psychosocial problems that patients with ADHD do experience (Corkum, 2001), or with sleep disturbances such as sleep-disordered breathing (SDB) and periodic leg movement disorder (PLMD) (Chervin and Archbold, 2001; Chervin *et al.* 1997, 2002; Crabtree *et al.* 2003; Picchietti *et al.* 1999). To approach this question, the objective evaluation of sleep in ADHD is of particular relevance.

However, sleep research in ADHD is complicated by the fact that, in contrast to subjective reports, objective polysomnographic (PSG) studies have not detected marked alterations in the sleep pattern of ADHD children. Only subtle PSG changes have been found that have also proved inconsistent across sleep laboratories. For example, an increased latency of rapid eye movement (REM) sleep onset (Busby *et al.* 1981), a decreased REM sleep latency (Khan, 1982), and a decreased overall REM activity (Greenhill *et al.* 1983) have been reported. Other PSG studies have found an increase in delta-wave sleep accompanied by a delayed sleep onset time and frequent awakenings (Ramos-Platon *et al.* 1990), and an increase in sleep spindles (Kiesow and Surwillo, 1987). More recently, a lack of significant changes in sleep patterns (Konofal *et al.* 2001; Lecendreux *et al.* 2000), less tolerance to the recording conditions (Palm *et al.* 1992), differences in sleep architecture through the night (Dagan *et al.* 1997), instability of the sleep–wake system (Gruber *et al.* 2000) and poor sleep quality (Andreou *et al.* 2003) have been found in ADHD children. Other recent data point to the possibility that a reduction of REM sleep in ADHD may be associated with PLMD (Crabtree *et al.* 2003; O'Brien *et al.* 2003). Increased movement-related time during sleep in ADHD appears to be the only more stable finding across PSG studies (Busby *et al.* 1981; Corkum *et al.* 1998; Konofal *et al.* 2001).

Given the incoherent and unstable findings of objective sleep parameters in mostly heterogenetic and clinically not well-defined groups of children with ADHD, the aim of the present study was to further characterize the sleep pattern of children with ADHD symptoms by means of sound clinical and PSG methods. The following criteria that may have contributed to previous inconsistent results were: (1) patients were selected according to two international diagnostic classifications after rigorous diagnosis for ADHD symptoms; (2) to control for possible stimulant therapy-related effects on sleep, patients were unmedicated before and during the PSG recording; (3) healthy controls were strictly matched for age, gender, and intelligence; (4) to control for effects of tolerance to recording conditions, PSG data from the first night were not considered and only data from the second night were compared between healthy controls and children with ADHD.

## METHODS

### Subjects

Seventeen ADHD (aged between 8.1 and 14.3 years) and 17 normal (8.0–14.4 years) preadolescent boys participated in the study (Table 1). The subjects were matched for age, and verbal and performance IQ–WISC-R (HAWIK-IQ; Tewes, 1983) tests.

Three of the 17 ADHD boys have never received any medication. The others who were medicated with methylphenidate hydrochloride (Ritalin®, Novartis Pharma GmbH, Nuremberg, Germany), discontinued their medication during the study as follows: in two patients, 4 months before the study; in two patients, 10 days before the study; and in the rest of 10 patients, 2 or 3 days before the study.

All children were examined clinically by two independent board-certified child psychiatrists. Patients with ADHD were referrals to the Department of Child and Adolescent Psychiatry at the University of Goettingen and fulfilled the diagnostic criteria for hyperkinetic disorder (HD) according to the International Classification of Diseases (ICD-10; World Health Organization 1992) and ADHD combined type according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994). Twelve children had additional symptoms of dyslexia with varying degree, three had symptoms of conduct disorder, one had symptoms of panic disorder, and one child had nocturnal enuresis. Healthy controls were recruited among friends and relatives of the clinical staff. The exclusion criteria for controls were current sleep problems, known psychiatric, neurological or internal problems, and EEG abnormalities. Likewise, ADHD children with internal medical problems were not recruited. ADHD and control children with IQ scores less than 80 were not considered for this study. This exclusion criterion was adopted to prevent variations in sleep parameters because

**Table 1** Group characteristics of ADHD ($n = 17$) and control ($n = 17$)

| | *ADHD* | *Control* | *P-value** |
|---|---|---|---|
| Age (years) | 11.2 ± 2.0 | 11.2 ± 2.3 | NS |
| WISC-R ($n = 16/16$) | | | |
| Performance – IQ | 100.9 ± 8.0 | 100.6 ± 11.6 | NS |
| Verbal – IQ | 100.1 ± 11.9 | 105.7 ± 8.1 | NS |
| CBCL (*T*-value) | | | |
| Internalizing | 58.5 ± 12.1 | 46.2 ± 7.3 | 0.001 |
| Externalizing | 62.8 ± 13.0 | 42.5 ± 8.1 | 0.001 |
| CBCL (*T*-value) | | | |
| Attention problems | 63.5 ± 7.6 | 50.5 ± 1.0 | 0.001 |
| Delinquent behavior | 60.6 ± 9.3 | 51.4 ± 2.4 | 0.001 |
| Aggressive behavior | 66.0 ± 12.9 | 50.6 ± 1.8 | 0.001 |
| Conners Parents Scale (10 items score) | | | |
| | 16.7 ± 6.3 | 3.4 ± 2.8 | 0.001 |

*Two-tailed Student's *t*-test.
Values are given as mean ± SD.
ADHD, boys with attention deficit/hyperactivity disorder; control, healthy boys; NS, not significant.
HAWIK-R is the German version of WISC-R.

of low IQ (Andreou *et al*. 2003). In addition, ADHD and control groups were carefully tested by means of psychopathological questionnaires (Table 1) including Conners Parents Rating Scale and Child Behaviour Checklist (CBCL; Achenbach and Edelbrock, 1983). This was performed to provide for a quantitative evaluation of hyperactivity and other psychopathology symptoms and statistically verify the difference between the two groups.

The study was performed according to the ethical standards of the Declaration of Helsinki and approved by the local ethical committee. A detailed description of the investigation was provided to the parents and their children. The parents of each subject gave written consent and the children gave age-appropriate consent after complete information about the investigation.

### Polysomnography

The sleep pattern in ADHD and normal children was studied by means of PSG methods in the sleep laboratory at the Center of Psychosocial Medicine (University of Goettingen, Germany). Both groups spent two consecutive nights in the sleep laboratory. The PSG included electroencephalogram recorded from C3 referenced to the right ear, electro-oculogram recorded from both sides of one of the orbits, submental electromyogram (EMG), and surface EMG from the anterior tibial muscles. To control for cardiovascular and sleep-related respiratory disturbances, electrocardiogram, oro-nasal airflow, breathing excursions by means of thoracic and abdominal sensors, and arterial oxygen saturation ($SpO_2$) by means of pulse oximetry (Apnoe Screen I, Erich Jaeger GmbH, Höchberg, Germany) were monitored during the first night. SDB was assessed as follows: apnea index (AI) was defined as the number of apneas per hour of sleep period time (SPT); hypopneas were identified as a decrease in oronasal airflow of ≥50% accompanied by a decrease in $SpO_2$ of ≥4% and/or arousals; apnea/hypopnea index (AHI) was defined as the number of apneas and hypopneas per hour of SPT (American Thoracic Society, 1996). All PSG recordings were performed on a 21-channel polyphysiograph (Nihon Kohden, Tokyo, Japan), with electrode impedance less than 5 KΩ, registered on paper with a speed of 10 mm $s^{-1}$, and stored on a computerized video-monitoring system. PSG data were analyzed visually in 30-s epochs according to standard criteria (Rechtschaffen and Kales, 1968). Sleep analyses were supplemented by counting epochs with short movements ($< 15$ s). In addition to short movements, periodic limb movements during sleep (PLMS) were scored when there were at least four movements of 0.5–5 s duration and between 5 and 90 s apart. A PLMS index of ≥5 per hour of SPT was considered as pathologically increased (Diagnostic Classification Steering Committee, 1990). In order to examine the sleep pattern quasi-naturally, subjects were allowed to have unrestrained sleep, i.e. to go to bed and to get up at their convenience. To avoid the first-night effect, only the results from the second night were included in the analyses.

### Statistics

For descriptive statistics of the variables, group means and standard deviations were calculated. For group comparisons, two-tailed Student's *t*-tests were applied. The level of significance for statistical procedures was set at $P \leq 0.05$.

## RESULTS

### Psychopathological scorings

Results from psychopathological scorings are presented in Table 1. As seen, both groups did not differ with respect to performance and verbal IQ. Compared with healthy controls, ADHD children displayed significantly higher scores in Conners Parents Rating Scale. Psychopathological scores in all CBCL (internalizing and externalizing problems, attention problems, delinquent and aggressive behavior) were also higher for the patients' group.

### Polysomnography

Major results from PSG are presented in Table 2. Children with ADHD spent significantly more time in bed and had a substantially longer SPT when compared with the healthy controls. No significant differences in the duration of total sleep time and in sleep efficiency were found between the two groups.

The latencies of the sleep stages were unaffected in ADHD as compared with healthy children. It is to be noted that in contrast to latencies of non-REM sleep stages, group means of the latency of REM sleep were essentially shorter in children with ADHD, although this difference did not reach a level of significance because of large variance.

No between-group differences were found for both the absolute and the relative duration of wake, stage I, stage II, and slow wave sleep. Importantly, ADHD children displayed a significantly longer absolute duration of REM sleep. When calculated in percentage per SPT, the relative REM sleep duration did not differ between ADHD and healthy children (Table 2). Likewise, the mean duration of REM sleep periods was not significantly longer in children with ADHD.

Notably, the number of sleep cycles was significantly higher in children with ADHD relative to healthy controls (Table 2).

No significant differences in time spent in wake after sleep onset and the number of awakenings per hour of SPT were observed between ADHD and healthy children. However, it is to be noted that despite the lack of significance, the number of awakenings per hour of SPT was much higher in ADHD children. The number of sleep stage shifts was stable across groups (Table 2).

### Movements

As shown in Table 3, the total movement time was not significantly different in ADHD compared with normal

**Table 2** Polysomnographic measures of ADHD ($n = 17$) and control ($n = 17$)

| *Second night* | *ADHD* | *Control* | *P-value** |
|---|---|---|---|
| General measures | | | |
| TIB (min) | 607.3 ± 65.2 | 568.1 ± 44.9 | 0.049 |
| SPT (min) | 584.0 ± 66.6 | 541.9 ± 46.9 | 0.039 |
| TST (min) | 566.7 ± 67.5 | 530.0 ± 42.0 | NS |
| SE (TST/TIB %) | 93.3 ± 3.1 | 93.3 ± 2.2 | NS |
| Latency of sleep stages (min) | | | |
| Stage I | 12.3 ± 10.6 | 14.0 ± 12.4 | NS |
| Stage II | 15.4 ± 11.0 | 17.9 ± 12.6 | NS |
| Stage III | 26.4 ± 13.1 | 27.0 ± 14.6 | NS |
| Stage IV | 36.7 ± 25.8 | 32.7 ± 21.2 | NS |
| REM | 115.3 ± 40.6 | 131.7 ± 40.6 | NS |
| Absolute duration of sleep stages (min) | | | |
| Wake | 36.2 ± 18.6 | 33.8 ± 13.3 | NS |
| Stage I | 18.6 ± 10.0 | 27.0 ± 17.3 | NS |
| Stage II | 262.7 ± 39.6 | 250.7 ± 39.7 | NS |
| Slow wave sleep (stages III + IV) | 139.6 ± 36.9 | 130.2 ± 32.5 | NS |
| REM | 139.3 ± 24.8 | 116.1 ± 17.0 | 0.003 |
| Relative duration of sleep stages (% SPT) | | | |
| Wake | 2.9 ± 4.2 | 2.1 ± 1.2 | NS |
| Stage I | 2.7 ± 1.8 | 4.4 ± 3.0 | NS |
| Stage II | 45.0 ± 4.8 | 46.2 ± 5.1 | NS |
| Slow wave sleep (stages III + IV) | 23.9 ± 5.8 | 24.2 ± 6.1 | NS |
| REM | 23.8 ± 2.8 | 22.1 ± 3.6 | NS |
| Mean duration of REM periods (min) | 24.2 ± 3.7 | 23.3 ± 4.9 | NS |
| Number of sleep cycles | 5.82 ± 1.07 | 5.12 ± 0.93 | 0.049 |
| Awakenings | | | |
| Wake after sleep onset (min) | 17.3 ± 11.7 | 12.0 ± 7.8 | NS |
| Number of awakenings per hour SPT | 0.97 ± 0.58 | 0.66 ± 0.23 | NS |
| Number of sleep stages shifts per hour SPT | 10.2 ± 3.4 | 9.9 ± 1.6 | NS |

*Two-tailed Student's *t*-test.
Values are given as mean ± SD.
ADHD, boys with attention deficit/hyperactivity disorder; control, healthy boys; NS, not significant; TIB, time in bed; SPT, sleep period time; TST, total sleep time; SE, sleep efficiency.

**Table 3** Movement parameters of sleep of ADHD ($n = 17$) and control ($n = 17$)

| *Second night* | *ADHD* | *Control* | *P-value** |
|---|---|---|---|
| Total movement time (% SPT) | 1.56 ± 0.63 | 1.68 ± 0.44 | NS |
| Total MRE per hour SPT | 16.8 ± 5.4 | 13.0 ± 4.0 | 0.025 |
| MRE in light sleep (stages I + II) (ep $h^{-1}$) | 21.3 ± 7.9 | 14.7 ± 6.0 | 0.011 |
| MRE in slow wave sleep (stages III + IV) (ep $h^{-1}$) | 8.0 ± 5.3 | 6.4 ± 4.1 | NS |
| MRE in REM sleep (REM) (ep $h^{-1}$) | 19.3 ± 7.6 | 20.8 ± 9.4 | NS |

*Two-tailed Student's *t*-test.
Values are given as mean ± SD.
ADHD, boys with attention deficit/hyperactivity disorder; control, healthy boys; NS, not significant; SPT, sleep period time; MRE, short movement-related epochs; ep $h^{-1}$ = epochs per hour.

children. However, the frequency of short movement-related epochs (number per hour of SPT) was significantly increased in the ADHD group. Further analyses revealed a higher frequency of short movement-related epochs in ADHD compared with normal children for the light sleep (stages I + II) only.

Two of 17 ADHD boys showed a PLMS index higher than 5 per hour of SPT, whereas in all other boys from both groups, the PLMS index was less than 3.

### Sleep-disordered breathing

Only one of 17 boys with ADHD displayed low scores of SDB: AI, 1.4 per hour of SPT; hypopneas, 1.7 per hour of SPT; AHI, 3 per hour of SPT. No signs of SDB were observed in the rest of ADHD boys and in the controls.

## DISCUSSION

The present PSG study addressed the question about the complex association between ADHD psychopathology and sleep (Brown and McMullen 2001; Corkum, 2001) by comparing the sleep patterns of unmedicated children rigorously diagnosed with ADHD and healthy controls, precisely matched for age, gender, and intelligence.

Major results revealed that several PSG parameters differed significantly between ADHD and healthy children: (1) children with ADHD spent significantly more time in bed and had a longer SPT, (2) they also manifested an increase in the absolute REM sleep duration, (3) had a higher number of sleep cycles relative to controls, and (4) showed a higher number of movement-related epochs in light sleep only.

As an unrestrained sleep schedule was used in this study, the longer SPT found for ADHD children might not be associated with a specific sleep alteration. Yet, the major observations of

an increased absolute duration of REM sleep and increased number of sleep cycles in ADHD children appear to be of special interest, because the duration of total sleep time did not differ significantly between the groups. It is therefore unlikely that these effects are simply produced by the augmentation of SPT. Instead, the increased absolute duration of REM sleep and the increased number of sleep cycles can be interpreted in the context of overall tendencies found here to accompany the sleep pattern in ADHD children. In line with previous reports (Khan, 1982), the group mean of REM sleep latency tended to be shorter in ADHD than in healthy controls (115.3 ± 40.6 versus 131.7 ± 40.6; Table 2), which, together with the increased absolute REM sleep duration might imply an elevated REM sleep pressure in ADHD children. However, the lack of significant between-group differences for the REM sleep latency, relative REM sleep duration, and mean duration of REM sleep periods, does not support this explanation. Rather, it may be assumed that the increased number of sleep cycles reflects an altered regulation of the sleep cycle oscillation in ADHD children (Gruber *et al*. 2000), which may primarily involve a major tendency for a faster transition to REM sleep manifested with a slightly elevated REM sleep activity. Hence, a forced REM sleep initiation may have produced the higher number of sleep cycles and also contributed to the increased absolute REM sleep duration. The overall pattern of these results suggests that a forced ultradian sleep cycling may be specific for ADHD children, reflecting another aspect of self-regulatory deficits in these children.

Further, the ADHD group displayed more frequent movement-related epochs per hour of SPT during light sleep (Table 3). These results are consistent with some previous PSG findings in ADHD patients (Busby *et al*. 1981; Konofal *et al*. 2001) and may reflect alterations of the general arousal as already proposed (Konofal *et al*. 2001). Whether or not this might trigger new sleep cycles remains to be answered.

One further question is whether the increased absolute duration of REM sleep in association with the higher number of sleep cycles is intrinsic for ADHD or whether it may be associated with confounding external factors. In previous studies, inconsistent diagnostic criteria and imprecise matching of ADHD patients with controls with respect to age, gender, and medication, may have introduced a high variability. Further confounding may come from inadequate control procedures, heterogeneity of tested sleep parameters, and averaging sleep data across nights (Corkum *et al*. 1998). In the present study, these sources of variation were controlled, although the rate of co-morbidity was relatively high in the sample of patients examined here, which is in fact typical for ADHD psychopathology and reflects a common mode of ADHD distribution (Banaschewski *et al*. 2003; Biederman *et al*. 1991; Rothenberger, 1990, 1998; Semrud-Clikeman *et al*. 1992). It may be argued, however, that the high rate of patients with dyslexic symptoms may be responsible for the results. This seems unlikely, because children with dyslexia have been reported to display a decrease in REM sleep and an increase in REM sleep latency (Mercier *et al*. 1993), which is in contrast to the present observations. Also, three of the ADHD patients with symptoms of conduct disorder, one with symptoms of panic disorder and another with nocturnal enuresis may not have affected significantly the results. Moreover, these patients were not outliers with respect to their sleep measures.

With regard to possible effects of previous drug treatment on PSG findings, it is to be noted that none of the patients were medicated several days before and during sleep recordings. Furthermore, stimulant medication has been demonstrated not to change substantially the PSG-sleep pattern in ADHD patients (Efron *et al*. 1997; Stein *et al*. 1996), although increased latency of falling into sleep has been observed. It has been also found that methylphenidate may even normalize primarily impaired sleep pattern in ADHD children (Tirosh *et al*. 1993).

Further, a non-specific and/or psychosocial stress-related effect may have produced the PSG differences observed here (Gregory and O'Connor, 2002). In fact, Strengths and Difficulties Questionnaire scores indicative for behavioral problems in children have been previously associated with shorter but not with longer total sleep time (Smedje *et al*. 2001). In adults, emotional tension has induced delta sleep increase and changes in delta sleep distribution throughout the night (Vein *et al*. 2002). These findings on the association between stress and sleep are not consistent with the present results, thus implying that PSG differences observed here are not affected by psychological stress in ADHD. Thus, the increased absolute duration of REM sleep, the higher number of sleep cycles, and related instability of ultradian sleep cycling may be suggested to be intrinsic for ADHD psychopathology.

Another important question concerning the sleep pattern in ADHD is the suggested association between sleep disturbances such as SDB and PLMD, and hyperactivity and inattention (Chervin and Archbold, 2001; Chervin *et al*. 1997, 2002; Crabtree *et al*. 2003; Picchietti *et al*. 1999). In our sample, the presence of SDB and PLMD was negligible. Yet, an increase in absolute REM sleep duration and number of sleep cycles was found. These observations indicate that whereas ADHD psychopathology mechanisms may involve SDB (Chervin and Archbold, 2001; Chervin *et al*. 1997) and PLMD (Chervin *et al*. 2002; Crabtree *et al*. 2003; Picchietti *et al*. 1999), these mechanisms certainly relate to sleep regulation in a different manner. The possible neurobiological considerations are discussed below.

### Neurobiological considerations

Explaining further the sleep-cycle control in ADHD, studies have documented that the neurochemical regulatory mechanisms of the sleep–wake cycle are closely associated with excitatory and inhibitory cortical processes across sleep wake (Gottesmann 1999; Muzur *et al*. 2002). Briefly, during sleep, both the inhibitory aminergic and the excitatory cholinergic inputs to the cortex sustaining a balance between cortical inhibition and excitation in wake, gradually diminish. With

sleep deepening, particularly during REM sleep, the activity of monoaminergic neurons ceases, while the cholinergic neurons activate again. This produces cortical disinhibition (Gottesmann 1999) or deactivation (Muzur *et al*. 2002) during REM sleep. It has been suggested that one of the pathophysiological mechanisms implicated in ADHD symptoms is the impaired cortical inhibitory control, which can lead to a disinhibition of motor and frontal cortices (Berger and Posner, 2000; Castellanos, 1997; Moll *et al*. 2000, 2001; Rothenberger, 1990). However, both prefrontal and frontal cortices have been recognized as crucial for the regulation of sleep (Gottesmann 1999; Muzur *et al*. 2002). Regarding these pathophysiological mechanisms together with the cortical disinhibition or deactivation characteristic for REM sleep (Gottesmann 1999; Muzur *et al*. 2002), the increased absolute REM sleep and the related forced ultradian sleep cycling in ADHD may originate from a greater cortical disinhibition or less activation of motor and frontal cortices that are inherent for the disorder (Berger and Posner, 2000; Castellanos, 1997; Moll *et al*. 2000, 2001; Rothenberger, 1990). Further, the decreased dopaminergic activity in ADHD (Ernst *et al*. 1998; Johansen *et al*. 2002; Pliszka *et al*. 1996) may underlie such a lower cortical inhibition, which may be improved by the dopaminergic drug methylphenidate hydrochloride (Moll *et al*. 2000).

In conclusion, the results from the present study imply that there may be a specific sleep pattern in children with ADHD, which may be closely associated with their basic brain neurobiological alterations related to deficits in state regulation.

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