Sleep Medicine 16 (2015) 1348–1351

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Brief Communication

Intraindividual long-term stability of sleep electroencephalography in school-aged children

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ARTICLE INFO

Article history:
Received 18 March 2015
Received in revised form 9 July 2015
Accepted 10 July 2015
Available online 28 August 2015

Keywords:
Sleep EEG
Sleep architecture
Middle childhood
Long-term stability

ABSTRACT

Objective: To examine the long-term stability of sleep duration, sleep continuity, and sleep architecture assessed via unattended home sleep electroencephalography (EEG) during middle childhood.
Methods: A total of 69 healthy children (18 girls and 51 boys) aged 8.2 years (standard deviation = 1.3 years) at T1 underwent unattended home sleep EEG on two nights separated by 18.5 months (standard deviation = 3.9 months). Of the children, 34 (49.3%) children were born prematurely (<32 gestational weeks; mean birth weight = 1367 g) and 35 (50.7%) children were born at term (mean birth weight = 3275 g).
Results: We found moderate to substantial stability (all $p < 0.001$) for total sleep time (TST; intraclass correlation coefficient [ICC] = 0.65), slow wave sleep (SWS; min, %: ICC = 0.49), and stage 2 sleep (min; ICC = 0.47), and found fair stability (all $p < 0.013$) for sleep efficiency (ICC = 0.28), nocturnal awakenings (ICC = 0.33), stage 2 sleep (%; ICC = 0.32), and rapid eye movement (REM) sleep (min: ICC = 0.33; %: ICC = 0.27). Prematurity status was not associated with stability of sleep EEG indices over time.
Conclusions: Long-term follow-up of one night of unattended home sleep EEG during middle childhood reveals that TST, stage 2 sleep, and SWS are relatively stable, trait-like characteristics. This applies less strongly for sleep efficiency, nocturnal awakenings, and REM sleep. Stage 1 sleep and REM latency showed no stability.

1. Introduction

Polysomnography (PSG) is the gold standard for sleep assessment, providing objective measures of sleep duration, sleep continuity, and sleep architecture. Although a growing body of research has applied PSG with childhood samples, surprisingly little is known regarding the psychometric properties of these PSG-derived sleep measures, such as the intraindividual stability, often also referred to as test–retest reliability. It is of particular importance to appraise the intraindividual stability of PSG-derived sleep measures in children to determine whether these measures reflect trait-like characteristics or simply a snapshot of children's sleep that is not representative of their habitual sleep patterns. To our knowledge, to date only two studies have addressed this question with regard to childhood samples. One laboratory study including 14 children 9–12 years of age found a high short-term stability of sleep architecture across two nights separated by one to two weeks, but not for sleep duration and continuity [1]. The small sample size, however, makes generalization from these findings difficult, and the confidence intervals around the stability coefficients are large. Long-term stability of PSG sleep in children has, to our knowledge, been examined in only one study with preschoolers, who were grouped into poor, normal, and good sleepers based on one night of sleep assessment with PSG at the children's homes. Twelve months later, the formerly poor sleepers were still more likely to show poorer sleep patterns compared to good or normal sleepers [2]. Thus, in preschoolers, sleep assessed during one night with unattended PSG showed significant stability over 12 months. However, because of the group-level approach in this study, the intraindividual long-term stability coefficients of the PSG-derived sleep indices remain unknown.

Further evidence that sleep indices are likely to show high long-term stability stems from behavioral–genetic research. In twin studies with adolescents and young adults, sleep continuity [3] and sleep architecture were strongly influenced by genetic factors, indicating trait-like (and therefore probably highly stable) characteristics

Abbreviations: TST, total sleep time; SWS, slow wave sleep; REM, rapid eye movement.

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http://dx.doi.org/10.1016/j.sleep.2015.07.025
1389-9457/

of the sleep parameters [4,5]. The strongest heritability concerning sleep stages was found for slow wave sleep (SWS), with heritability estimates ranging between 0.50 and 0.90 [4]. However, particularly during childhood, high heritability does not necessarily indicate that individual characteristics are unchangeable [6]. Thus, although behavioral–genetic studies suggest that sleep continuity and architecture are heritable characteristics and should therefore remain highly stable, none of these studies examined the long-term stability that is a prerequisite for PSG-derived sleep indices being trait-like characteristics.

To fill the gap in research regarding the stability of sleep electroencephalogram (EEG) indices, the present study examined the stability coefficients of sleep EEG parameters during middle childhood across 1.5 years. We expected high stability coefficients for sleep duration, sleep continuity, and sleep architecture, which would also indicate that one night of sleep EEG can be regarded as a reliable measure of children's habitual sleep. In particular, we assessed sleep with unattended home sleep EEG, allowing the children to sleep in their usual surroundings and according to their usual schedule, which, compared to laboratory-assessed sleep, results in better sleep quality [7,8] and provides a more convenient and cost-effective method to assess sleep in children [9].

Furthermore, we examined the stability of sleep EEG indices in children born very preterm (<32 gestational weeks), who are at higher risk for sleep-disordered breathing [10], which may be associated with less restorative sleep architecture patterns [11]. To date it is unknown whether the stability of sleep EEG indices differ between children born very preterm and at term. It is conceivable that more disrupted sleep, as shown by very preterm children, may result in decreased long-term stability of sleep EEG indices.

2. Methods

2.1. Study population and procedure

The study included 69 healthy school-aged children (18 girls and 51 boys; age: mean = 8.2 years, standard deviation [SD] = 1.3 years, range = 6.0–10.9 years), who underwent one night of unattended home sleep EEG assessment between June 2011 and September 2012 and again between May 2013 and February 2014 (9.6–27.5 months later; mean = 18.5 months, SD = 3.9 months). At follow-up, children were on average 9.8 years old (SD = 1.3 years). Of the children, 34 (49.3%) were born very premature (<32 weeks of gestation; mean birth weight = 1367 g), and 35 (50.7%) were born at term (mean birth weight = 3275 g). Very preterm children were recruited from a cohort of prematurely born children treated at the University Children's Hospital Basel (Switzerland), which has been described elsewhere in detail [11]. All children attended primary school in Switzerland. Parents provided written informed consent for the children to participate, children gave assent. The study was approved by the Ethics Committee of Basel.

2.2. Sleep assessment

Sleep was assessed using the ambulatory Compumedics Somté PSG device (Melbourne, Australia) for one night at T1 and one night at T2 on a regular school day (ie, Monday to Thursday). Polysomnographic signals C3/A2 and C4/A1 EEG, right and left electrooculogram, and bipolar submental electromyogram were obtained. Sleep EEGs were scored by experienced raters according to the standard procedures [12]. Sleep indices included the following: Total sleep time (TST; time in bed minus time spent awake, in hours), sleep efficiency (TST/time in bed × 100), nocturnal awakenings (number of arousals from sleep), and sleep architecture (stage 1 sleep, stage 2 sleep, SWS stages 3 and 4, REM sleep, and REM latency).

2.3. Statistical analysis

Paired-sample *t* tests were conducted to examine changes in the sleep EEG indices from T1 to T2. To test the longitudinal stability of sleep EEG measures, a series of regression analyses were conducted, with sleep parameters at T1 predicting the corresponding sleep parameters at T2, controlling for children's age and prematurity status. Supplementary analysis included the time period between T1 and T2 as a covariate. As an additional measure of stability, we calculated the intraclass correlation coefficients (ICCs), defined as the error-free between-subject variance, divided by the sum of the error-free between- and within-subject variances [13], and interpreted according to the following benchmarks: slight (0.0–0.2), fair (0.2–0.4), moderate (0.4–0.6), substantial (0.6–0.8), and almost perfect (0.8–1.0) [14]. To examine whether prematurity status would moderate the stability of sleep EEG indices, we further conducted hierarchical regression analyses. Statistical computations were performed with IBM SPSS Statistics 20 (IBM Corporation, Armonk NY, USA) for Apple Mac.

3. Results

Table 1 shows the descriptive statistics of the sleep EEG indices at T1 and T2, including the analysis of mean value change (paired-sample *t* test). TST, sleep efficiency, SWS (min, %) and REM sleep (min) decreased over time, while stage 1 sleep (min, %) and stage 2 sleep (%) increased between T1 and T2. Furthermore, analyses showed that TST (ICC = 0.65, Fig. 1a), sleep continuity (sleep efficiency ICC = 0.28, Fig. 1b), nocturnal awakenings (ICC = 0.33, Fig. 1c), and sleep architecture (stage 2 sleep %: ICC = 0.33, Fig. 1d; stage 2 sleep min: ICC = 0.47; SWS %: ICC = 0.49, Fig. 1e; SWS min: ICC = 0.49; REM sleep %: ICC = 0.27, Fig. 1f; REM sleep min: ICC = 0.33) showed fair to substantial stability over 18.5 months with regard to the individuals' position in the distribution, after controlling for children's age and prematurity status. No significant associations between T1 and T2 sleep parameters were found for stage 1 sleep (stage 1 sleep %: $t = 1.55$, $\beta = 0.20$, $p = .13$; ICC = 0.16; stage 1 sleep min: $t = 1.70$, $\beta = 0.21$, $p = .09$; ICC = 0.17) and REM latency ($t = 0.36$, $\beta = 0.05$, $p = .72$; ICC = 0.02).

Children's age was associated with TST ($t = -2.48$, $\beta = -0.25$, $p = 0.02$), such that sleep duration decreased with age, while prematurity status was not associated with any sleep variables (all $p > 0.43$). Hierarchical regression analyses revealed no prematurity × sleep interactions (all $p > 0.08$). Furthermore, results remained very similar when additionally controlling for the time span that elapsed between T1 and T2.

Table 1
Descriptive statistics for total sleep time, sleep continuity, and sleep architecture.

	T1		T2			
	Mean	(SD)	Mean	(SD)	*t*	*p*[a]
TST (h)	9.5	(0.7)	9.0	(0.8)	6.20	<0.001
Sleep efficiency (%)	94.5	(2.5)	93.3	(3.0)	2.89	0.01
Nocturnal awakenings	17.2	(7.3)	16.2	(7.3)	1.00	0.32
Stage 1 sleep (min)	18.6	(13.9)	23.5	(14.3)	–2.24	0.03
Stage 1 sleep (%)	2.8	(2.6)	3.9	(2.6)	–2.53	0.01
Stage 2 sleep (min)	253.1	(43.1)	256.0	(35.9)	–0.58	0.56
Stage 2 sleep (%)	44.1	(6.0)	46.7	(5.0)	–3.27	0.002
SWS, stages 3 and 4 (min)	148.7	(33.9)	122.4	(27.8)	6.82	<0.001
SWS, stages 3 and 4 (%)	25.3	(5.8)	21.7	(5.3)	5.32	<0.001
REM sleep (min)	146.8	(27.2)	139.4	(25.5)	2.01	0.05
REM sleep (%)	25.4	(4.5)	25.2	(3.9)	0.28	0.78
REM latency (min)	113.5	(43.9)	114.3	(41.8)	–0.11	0.91

REM, rapid eye movement; SD, standard deviation; SWS, slow wave sleep; TST, total sleep time.
[a] *p* value from paired-sample *t* test.