Determining Sleep Quality in Children with Sleep Disordered Breathing: EEG Spectral Analysis Compared with Conventional Polysomnography

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Study Objectives: To identify the extent of sleep disruption in children with various severities of sleep disordered breathing (SDB) using both conventional visually scored assessment of sleep stages and arousal indices together with EEG power spectral analysis.

Design: Sleep stages and power spectral analysis of the sleep EEG in children with varying severities of SDB with matched control subjects with no history of snoring were compared across the whole night, across sequential hours from sleep onset, and across sleep stages.

Measurements: Overnight polysomnography was performed on 90 children (49M/41F) aged 7-12 y with SDB and 30 age-matched healthy controls (13M/17F). Sleep stages were visually scored and the EEG spectra were analyzed in 5-s epochs.

Results: Conventional visual scoring indicated that, although sleep duration was reduced in severely affected children, sleep quality during the essential stages of SWS and REM was preserved, as evidenced by the lack of any significant decrease in their duration in SDB severity groups. This finding was supported by the lack of substantial differences in EEG spectral power between the groups over the whole night, within specific hours, and in individual sleep stages.

Conclusions: Both conventional scoring and EEG spectral analysis indicated only minor disruptions to sleep quality in children with SDB when assessed across the night, in any specific hour of the night, or in any specific sleep stage. These results suggest that reduced daytime functioning previously reported in children with SDB may not be due to sleep disruption. We speculate that in children, in contrast to adults, a stronger sleep drive may preserve sleep quality even in severe SDB.

Keywords: Pediatric, children, sleep, apnea, sleep quality, spectral analysis, EEG

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SLEEP DISORDERED BREATHING (SDB) IS A VERY COMMON CONDITION IN OTHERWISE HEALTHY CHILDREN, OCCURRING IN UP TO 34% OF THE population.[1-4] SDB ranges in severity from primary snoring (PS), which is not associated with gas exchange abnormalities or sleep disturbance as detected on conventional polysomnography (PSG), to obstructive sleep apnea (OSA) which is characterized by snoring, apnea, intermittent hypoxia, hypercarbia, and repeated arousals from sleep.[5] Children with severe SDB have been reported to exhibit a range of deficits in neurocognitive performance, behavioral problems, and poor school performance.[6-11] It has been suggested that these symptoms could be related to the repeated episodes of hypoxia and/or the disruption of sleep from repeated arousals, resulting in poor sleep quality, which is experienced by children with SDB.[12] However, while it was previously believed that only children with severe SDB required clinical intervention,[13] recent reports suggest that children with PS, traditionally considered benign, also display a wide range of neurocognitive deficits in domains such as intelligence, memory, and attention.[14,15] As PS does not cause hypoxia, close attention to the effects of SDB on sleep quality is warranted.

Conventional methods for assessing the disruption of sleep quality involve visual scoring of the EEG and an assessment of sleep architecture.[16] Unlike adults, past studies have indicated that the sleep architecture of children with SDB seems to be relatively preserved.[15,17-22] Alternatively, the frequency of arousal from sleep (the arousal index) may be used to quantify sleep disturbance. Compared to adults, children have markedly fewer arousals,[23,24] with only ~50% of apneic events terminating with a cortical arousal as defined by adult criteria.[25,26] However, it remains possible that analyses based on conventional criteria are not detecting sleep disturbance in children. This is important as previous studies have not found a good correlation between these conventional measures and behavioral and neurocognitive outcomes in children.[16]

Power spectral analysis of the EEG has previously been used to examine sleep EEG in infants,[27] children[11,28] and adults,[29,30] and may provide a more sensitive measure of sleep disruption in children with SDB than conventional sleep architecture or arousal indices. Thus the aim of the current study was to compare assessments of sleep quality using both conventional and spectral analysis measurements in children with various severities of SDB and healthy non-snoring controls.

METHODS

This study was part of a larger project which investigated the effects of different severities of SDB on the cardiovascular system, neurophysiology, neurocognition, and behavior in pri-

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mary school-aged children aged 7-12 years. It commenced after approval by the Southern Health and Monash University Human Research Ethics Committees. Verbal assent from the children and written consent from the parents was obtained prior to commencement of each sleep study. No monetary incentive was provided to participants.

Subjects

Of the 152 children recruited for the larger study, 120 children (62M/58F) had ≥ 4 h of sleep required for diagnosis of SDB severity and EEG signals recorded at 500Hz and thus could be utilized for this project. Ninety (49M/41F) were patients undergoing clinical PSG for diagnosis of SDB, and 30 (13M/17F) were age-matched healthy control children with no history of snoring, recruited from the public. Snoring was assessed by parental report. All clinically referred subjects had a parent reported history of snoring, while the control subjects had a negative history of snoring. This was verified by staff observations during the PSG study. All children underwent a complete medical examination prior to the study and children with conditions known to affect sleep, breathing, or blood pressure were excluded. Children were not receiving medication at the time of the study, apart from 12 diagnosed with asthma who were taking inhaled bronchodilators and/or inhaled steroid treatment.

Overnight Polysomnography

Children had their height and weight recorded on arrival at the sleep laboratory, before sensors were attached in preparation for PSG. PSG recordings consisted of electroencephalogram (EEG: C4-A1, O2-A1), electrocardiogram (ECG), electro-oculogram (EOG), submental and leg electromyogram (EMG), oxygen saturation (Biox 3700e, Ohmeda, Boulder, CO, USA), transcutaneous pCO_2 (TCM3, Radiometer, Copenhagen, Denmark), thoracic and abdominal respiratory movements (z-RIP belts, Pro-Tech Services Inc., Mukilteo, WA, USA), and airflow, recorded using nasal pressure and an oronasal thermistor (Triple Thermistor, Compumedics Ltd, Melbourne, Australia). In addition, blood pressure (Finometer, Finapres Medical System, Arnhem, The Netherlands) was recorded as part of the larger study. A position sensor was used to record the patient's body orientation. All signals were digitally recorded on a computerized system (S-series Sleep System, Compumedics Ltd, Melbourne, Australia) and stored for offline analysis. Once the sensors had been attached and signal integrity verified, the room was darkened and the patient was monitored from an external station via infra-red camera. The room temperature was kept at a constant range of 21-24°C. All patients were awoken at 06:00, and leads were removed by sleep laboratory staff.

Clinical Analysis of PSG

Sleep architecture was assessed by trained sleep technicians and manually sleep staged in 30-s epochs following standard criteria,[31] with both cortical (ASDA arousal criteria of a change in EEG frequency lasting ≥ 3 s and requiring an EMG increase in REM)[32] and subcortical (≥ 2 of: increase in heart rate, increase in EMG, distortion of respiratory effort belts) arousals included as per our current clinical practice.[33] Respiratory events were scored if ≥ 2 respiratory cycles in duration. An obstructive apnea was defined as the complete cessation of airflow at both the mouth and nose in conjunction with continued respiratory effort. A central apnea was characterized by a cessation in airflow without ongoing respiratory effort. A mixed apnea was defined as a respiratory event displaying characteristics of both an obstructive and a central apnea. A hypopnea was defined as ≥ 50% decrease in airflow followed by either a ≥ 3% decrease in oxygen saturation or an arousal.[34]

Standard measures of sleep quality were calculated for each participant and included: respiratory disturbance index (RDI), central apnea index (CnAI), obstructive apnea index (OAI), obstructive apnea hypopnea index (OAHI, including obstructive apneas, obstructive hypopneas and mixed apneas).[34] RDI was defined as the number of obstructive apneas, central apneas, mixed apneas, and hypopneas per hour of total sleep time. Diagnostic criteria for classification of SDB severity were based on current clinical practice and children were divided into 4 groups: (1) primary snoring—snoring and OAHI ≤ 1; (2) mild OSA—OAHI > 1–5; (3) moderate / severe OSA—OAHI > 5; and (4) control—OAHI < 1 and no symptoms of SDB.

The duration of each sleep stage and wake after sleep onset (WASO) was calculated as a percentage of the sleep period time (SPT), defined as the amount of time in minutes from sleep onset until lights on at the end of the study, including all periods of wake in between. WASO was defined as the amount of wake after sleep onset until lights on at the end of the study. Total sleep time (TST) was defined as SPT excluding all periods of wake. Other variables calculated included time in bed (TIB), sleep latency, REM latency, and sleep efficiency. TIB was defined as the time between lights off and lights on. Sleep latency was defined as the period from lights off to the first 3 consecutive epochs of stage 1 sleep or an epoch of any other stage. REM latency was defined as the period from sleep onset to the first epoch of REM sleep. Sleep efficiency was defined as the ratio of TST to TIB.

Power Spectral Analysis of EEG

Raw EEG signals were recorded with band-pass filters spanning 0.3 Hz to 100 Hz and sampled at a minimum of 500 Hz. The primary EEG channel (C4-A1) was exported in the European Data Format (EDF) and subsequently imported into Scan 4.3 (Neuroscan, Compumedics Ltd, Melbourne, Australia) for offline conditioning. The entire EEG time series was digitally band-pass filtered from 0.75 to 30 Hz to remove any low- or high-frequency artifacts, then divided into consecutive 5-s epochs irrespective of sleep stage or sleep-wake state. Data on changes in sleep stage were then exported as plain text from the Compumedics software and manually matched to spectral analysis data by synchronizing with the clinically scored sleep onset time.

All 5-s epochs containing major interference from an external source (i.e., impedance testing, signal drop-out due to mechanical disconnections) were removed from the analysis. These rejected epochs typically resulted in rejection of < 30 s of data per study. Due to the Fourier transformation requiring data segments of $n \times 10^2$ data points in length for spectral analysis, these epochs were re-sampled using a spline algorithm to 2048 points. Spectral analysis using fast Fourier transform (FFT) was run on the 5-s epochs over the entire recording with a Hanning window to avoid edge effects.[28]

Spectral power was calculated for all 5-s blocks from sleep onset until the end of the study, and epochs recorded prior to sleep onset were excluded from all analyses. The FFT output provided a total power for each 5-s block with a frequency resolution of 0.2 Hz between zero and 30 Hz. These 0.2 Hz frequency bins were subsequently summed within 5 frequency bands: δ (delta, 0.5-4 Hz); θ (theta, 4-8 Hz); α (alpha, 8-12 Hz); σ (sigma, 12-14 Hz); and β (beta, 14-30 Hz), producing a single power value for each band. In addition total power for each 5-s block was determined (0-30 Hz). The remaining EEG epochs from sleep onset until the end of the study were combined for 3 separate analyses:

1. Average 5-s power spectra for the whole SPT.
2. Average 5-s power spectra for each hour for the first 6 h of the SPT irrespective of sleep stage or sleep-wake state. This period was chosen, as 97.5% of children had ≥ 6 h SPT; whereas after this time, the number of children who remained asleep was significantly reduced.
3. Average 5-s power spectra for each sleep stage, as well as wake, for the whole night, with all blocks from sleep onset until study termination.

The 5-s epochs were recombined into 30-s blocks, and data for all 3 analyses were initially expressed as absolute power averaged over 30-s blocks. For the "whole night" analysis, the values for each frequency band were also expressed as relative power; the absolute power in each frequency band divided by the total absolute power. For the analyses "by hours" and "by stages," proportional values were calculated. The absolute 30-s values within each frequency band were determined for each hour or stage, summed over the 6 h or 6 stages, and the 30-s value in each hour or stage divided by the summed values. The computation of relative or proportional power removed individual differences in absolute power between subjects, and therefore reduced variability and avoided potential bias by individual subjects with high absolute power values.

Comparing EEG Spectral Power to Measures of SDB Severity

To further evaluate the relationship between childhood SDB severity and EEG spectral power distribution, OAHI and RDI were regressed with the whole night relative spectral power over all 120 subjects within each of the 5 frequency bands.

Statistical Analysis

Statistical analysis was performed using Sigma Stat 3.0 (SPSS Inc., Chicago, IL, USA). Data were first tested for normality and equal variance. One-way analysis of variance (ANOVA) was used for comparison between the 4 subject groups; control, primary snoring (PS), Mild OSA, and Moderate/Severe OSA, for conventional measures of sleep quality, with Student Newman-Keuls post hoc analyses. OAHI and RDI violated homogeneity of variance, thus a Kruskal-Wallis one-way ANOVA on ranks was used, with Dunn post hoc tests used to identify the source of any difference. Absolute and relative spectral power averaged over the whole night and proportional spectral power in each hour of the night, and in individual sleep stages in the different subject groups were analyzed with 2-way ANOVA. In cases where overall significance was identified by ANOVA, Student Newman-Keuls post hoc tests were performed to identify the source of the difference. Relative power for each of the 5 frequency bands was compared with regression analysis to OAHI and RDI. All results are presented as mean ± SEM. P values < 0.05 were considered statistically significant.

Table 1—Demographics and clinical scales of sleep disruption between subject groups

	Control n = 30	PS n = 50	Mild OSA n = 20	Mod/Sev OSA n = 20	Post hoc Comparisons
Gender	13M/17F	32M/18F	11M/9F	6M/14F	ns
Age (years)	9.8 ± 0.3	9.8 ± 0.2	9.0 ± 0.3	9.2 ± 0.4	ns
Height (cm)	137.9 ± 2.4	140.1 ± 1.8	135.1 ± 2.2	138.5 ± 2.1	ns
Weight (kg)	35.8 ± 1.8	38.7 ± 2.1	33.5 ± 2.6	43.4 ± 2.9	ns
BMI kg/m²	18.5 ± 0.5	19.4 ± 0.6	18.4 ± 0.9	22.7 ± 1.1	C, PS, M < MS, P < 0.01
BMI Z-score	0.5 ± 0.2	0.6 ± 0.2	0.4 ± 0.2	1.5 ± 0.2	C, PS, M < MS, P < 0.01
OAHI (hTST)	0.1 ± 0.0	0.3 ± 0.0	2.4 ± 0.2	16.8 ± 2.8	C, PS < M, MS, P < 0.01
RDI (hTST)	0.6 ± 0.1	0.9 ± 0.2	3.1 ± 0.3	17.6 ± 2.8	C, PS < M, MS, P < 0.01
CnAHI (hTST)	0.5 ± 0.1	0.5 ± 0.2	1.0 ± 0.3	0.9 ± 0.2	ns
ArI (Arousal/h)	11.4 ± 0.5	11.6 ± 0.6	14.8 ± 0.9	25.4 ± 2.5	C, PS, M < MS, P < 0.001

Values are expressed as mean ± SEM. C refers to control; PS, primary snoring; M, mild OSA; MS, moderate/ severe OSA. OAHI, RDI, and CnAHI are expressed the number of events per hour of total sleep time (hTST). ArI is expressed as arousals per hour.

RESULTS

Children were divided into 4 groups on the basis of OAHI: primary snoring (n = 50), mild OSA (n = 20), moderate/ severe OSA (n = 20), and controls (n = 30).

Demographics and Clinical Scales of Sleep Disruption (Table 1)

There were no significant differences between groups for age, height, or weight. However, both body mass index (BMI) and BMI z-score were significantly higher in the moderate/ severe OSA group (P < 0.01) than the other 3 groups. As expected on the basis of their clinical diagnosis, both the mild OSA and the moderate/ severe OSA group had a significantly higher OAHI, RDI, and total arousal index (ArI) in than both the control and PS groups (P < 0.001 for all).

Conventional Measures of Sleep Quality (Table 2)

Children in the SDB groups had more disturbed sleep, with reduced TST, reduced sleep efficiency, and increased % time spent in NREM1, with sleep disturbance being greater in the more severely affected children. Thus, TST showed a significant difference between subject groups, with the moderate/ severe group having a reduced TST compared to all other subject groups (P < 0.001). Similarly, SPT was also significantly reduced in the moderate/ severe group compared to the other groups (P < 0.01). All 3 SDB groups had a significantly longer

Table 2—Conventional measures of sleep quality between subject groups

	Controls n = 30	PS n = 50	Mild OSA n = 20	Mod/Sev OSA n = 20	Post hoc Comparisons
Time in bed (min)	479 ± 6	477 ± 5	484 ± 6	459 ± 9	ns
Total sleep time (min)	414 ± 9	397 ± 7	396 ± 10	357 ± 13	C, PS, M > MS, P < 0.001
Sleep period time (min)	463 ± 6	443 ± 6	452 ± 9	423 ± 11	C, PS, M > MS, P < 0.001
Sleep latency (min)	14 ± 2	30 ± 3	30 ± 6	35 ± 7	C < PS, M, MS, P < 0.01
REM latency (min)	158 ± 10	151 ± 6	132 ± 15	188 ± 18	PS, M < MS, P < 0.05
Sleep efficiency (%)	86.6 ± 1.5	83.7 ± 1.3	81.9 ± 1.9	77.8 ± 2.2	C, PS < MS, P < 0.05
NREM 1 (%)	7.9 ± 0.6	8.2 ± 0.4	10.2 ± 0.7	11.1 ± 1.1	C, PS < MS, P < 0.01
NREM 2 (%)	43.9 ± 1.4	43.8 ± 1	39.0 ± 1.7	38.4 ± 1.6	C, PS > MS, P < 0.01
NREM 3 (%)	4.5 ± 0.4	4.7 ± 0.2	4.7 ± 0.5	3.7 ± 0.3	ns
NREM 4 (%)	17.1 ± 0.8	17.3 ± 0.8	18.6 ± 1.1	17.0 ± 1	ns
REM (%)	16.1 ± 0.8	15.8 ± 0.6	15.3 ± 1	14.3 ± 1.5	ns
WASO (%)	10.5 ± 1.5	10.2 ± 1.1	12.2 ± 1.4	15.6 ± 2.2	ns

Values are expressed as mean ± SEM. C refers to control; PS, primary snoring; M, mild OSA; MS, moderate/ severe OSA

Table 3—Absolute and relative EEG spectral power over the whole night in each frequency band

	Freq. Band	Controls n = 30	PS n = 50	Mild OSA n = 20	Mod/Sev OSA n = 20	Post hoc Comparisons
Absolute Power	Delta (μv^2)	1514 ± 114	1600 ± 120	1672 ± 145	1806 ± 205	ns
	Theta (μv^2)	204 ± 14	253 ± 19	270 ± 20	270 ± 24	ns
	Alpha (μv^2)	54 ± 4	56 ± 3	67 ± 12	66 ± 7	ns
	Sigma (μv^2)	18 ± 2	21 ± 1	21 ± 2	20 ± 2	ns
	Beta (μv^2)	26 ± 2	25 ± 1	30 ± 4	31 ± 4	ns
	Total (μv^2)	1816 ± 123	1953 ± 134	2061 ± 166	2193 ± 226	ns
Proportional Power	Delta (%)	82 ± 1	80 ± 1	81 ± 1	81 ± 1	ns
	Theta (%)	12 ± 1	14 ± 1	14 ± 1	14 ± 1	ns
	Alpha (%)	3 ± 0.3	3 ± 0.2	3 ± 0.3	3 ± 0.3	ns
	Sigma (%)	1 ± 0.1	1 ± 0.1	1 ± 0.2	1 ± 0.1	ns
	Beta (%)	2 ± 0.1	2 ± 0.2	2 ± 0.1	2 ± 0.2	ns

Values are expressed as mean ± SEM.

sleep latency compared to controls ($P < 0.01$). REM latency was also significantly longer in the moderate/ severe OSA group than in the PS and mild OSA groups ($P < 0.05$). Sleep efficiency was significantly poorer in the moderate/ severe OSA group than all other subject groups ($P < 0.05$).

The moderate/ severe group spent more time in NREM1 than both the control and PS groups ($P < 0.01$ for both). A significant difference was also seen in the duration of NREM2, with the moderate/ severe OSA group spending significantly less time in NREM2 than both the control and PS groups ($P < 0.01$ for both). No significant differences were seen between groups in NREM3, NREM4, REM, or WASO.

EEG Spectral Power Distribution across the Night (Table 3)

Absolute EEG spectral power averaged over 30-s blocks for the entire night showed no significant differences between groups in any of the 5 frequency bands, or as summed power across all 5 bands. Similarly, relative EEG spectral power over the entire period of sleep (SPT), expressed for each frequency band as a percentage of total power, also showed no significant differences between groups in any of the frequency bands. As would be anticipated, power in the δ frequency band predominated in all subject groups, averaging ~80%, followed by θ (~15%), and α (~4%); σ and β power comprised only a small proportion (~2%) of the spectral distribution.

The relationship between childhood SDB severity and EEG spectral power distribution was evaluated by regressing the OAHI and RDI with the relative spectral power within each of the 5 frequency bands. No significant correlation was observed between either OAHI or RDI and EEG spectral power in any of the 5 frequency bands.

EEG Spectral Power Distribution as the Night Progresses (Figure 1A-E)

Proportional EEG spectral power is displayed across the first 6 h from sleep onset. Within each frequency band the absolute hourly averages (averaged over all 30-s blocks) were summed over the 6 h, and each hour expressed as a proportion of the sum. This representation indicated the distribution of power within each frequency band over the night. There was a fall in proportional spectral power as the night progressed, in the δ (Figure 1A), θ (Figure 1B) and α (Figure 1C) frequency bands with proportional power in Hour 1 being significantly greater in all 4 subject groups compared with all later hours ($P < 0.05$ for all). Delta proportional power in Hour 2 was also greater in the control, PS, and mild OSA groups compared to all later hours ($P < 0.05$). Sigma proportional power (Figure 1D) was greater in the control and mild OSA groups in Hour 1 compared with Hour 2 ($P < 0.05$). There were no differences in proportional power as the night progressed in the β frequency band (Figure 1E).

A significant difference was seen between subject groups in δ and θ power spectral distribution in the second hour, with a tendency for power in these frequency bands to shift later in the night. Thus, there was significantly lower proportional δ power in the moderate/ severe OSA group compared to all other groups in the second hour ($P < 0.05$ for all). The moderate/ severe OSA group also had a lower θ power than the other groups in the second hour, with this reaching statistical significance when compared to the mild OSA group ($P < 0.05$). A

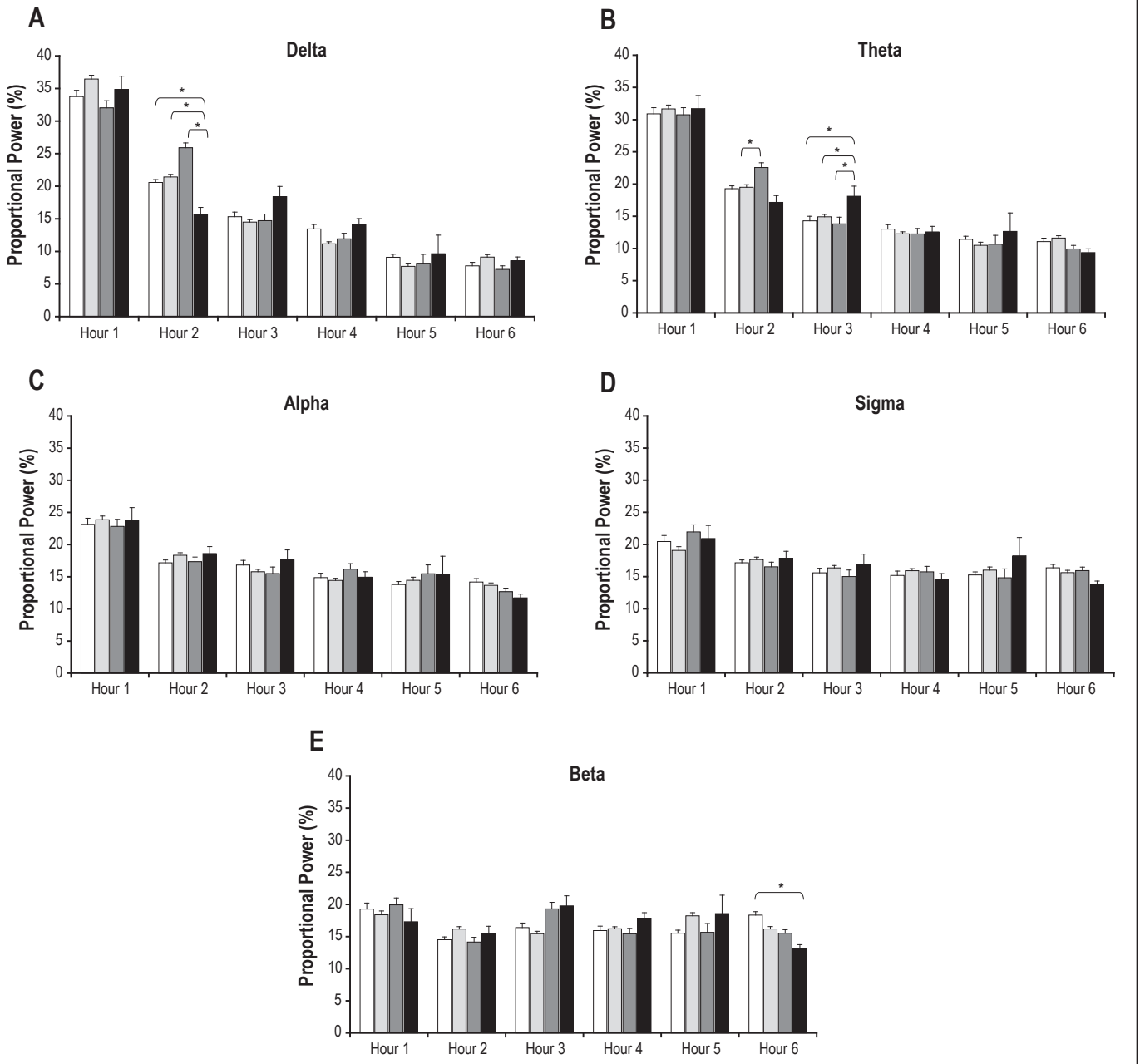

Figure 1A-E—Proportional spectral power distribution across the first 6 hours from clinically defined sleep onset, with power in each hour being expressed as a percentage of the sum of the 6 hours within each frequency band. (A) delta, (B) theta, (C) alpha, (D) sigma, (E) beta.
□ Controls ▒ PS ▒ Mild OSA ■ Mod/Sev OSA; *P < 0.05 between groups. (Statistical differences between Hours are not shown on graph)

significant difference was also seen during the third hour in the θ frequency band, with the moderate/ severe OSA group having significantly higher power than all 3 other subject groups ($P < 0.05$ for all). The β frequency band in the moderate/ severe OSA group displayed a significant decrease in spectral power during Hour 6 compared to the control group ($P < 0.05$). No significant differences were seen in α or σ EEG spectral power distribution in any of the 6 hourly blocks.

EEG Spectral Power Distribution in Sleep Stages (Figure 2A-E)

Within each frequency band, the proportion of EEG spectral power in each of the 5 sleep stages (NREM1-4 and REM) and WASO was determined by summing average absolute power in each stage, summing these values over the 6 stages and dividing the value for each stage by the sum (Figure 2). No significant difference in δ power (Figure 2A) was seen in any of the sleep stages between any of the 4 subject groups. Delta power predominated in NREM3 and NREM4, being significantly higher than in all other sleep and wake stages. Delta power was lowest in NREM1 and REM. Theta power (Figure 2B) in the moderate/ severe OSA group was significantly lower when compared to the PS group, in both NREM1 ($P < 0.05$) and NREM2 ($P < 0.05$). Theta power in NREM2 was also significantly lower in the moderate/ severe OSA group compared to the mild OSA group, ($P < 0.05$). Theta power predominated in NREM3 and NREM4, being significantly higher than in all other sleep and wake stages. Theta power was not different between NREM1 and REM. In the α frequency band (Figure 2C), the moderate/

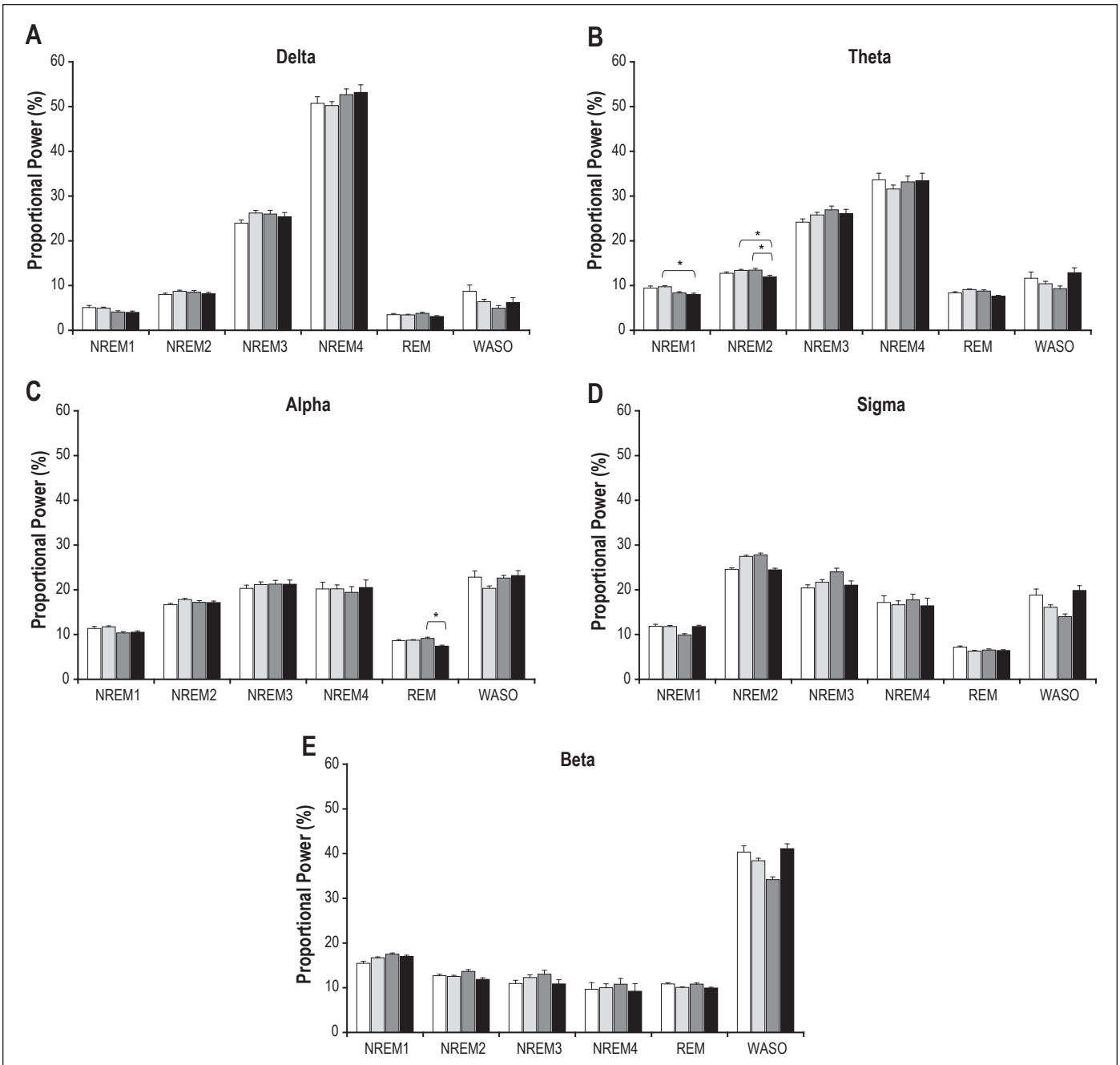

Figure 2A-E—Proportional spectral power distribution in each of the five sleep stages as well as in WASO periods as a percentage of (A) delta, (B) theta, (C) alpha, (D) sigma, (E) beta.
□ Controls ▒ PS ▒ Mild OSA ■ Mod/Sev OSA; *P < 0.05 between groups. (Statistical differences between Stages are not shown on graph)

severe OSA group displayed significantly lower power in REM compared to the mild OSA group ($P < 0.05$). No significant differences were seen between groups in any of the sleep stages in the σ or β frequency bands (Figures 2D, 2E).

DISCUSSION

To our knowledge, this is the first study to utilize spectral analysis of the EEG as an alternative measure of sleep quality for examining markers of sleep disruption in children with various severities of SDB and age-matched healthy children with no history of snoring. Using conventional measures of sleep quality we found evidence of relatively minor impairment of sleep quality in children with SDB compared to non-snoring control children. Spectral analysis of the EEG also did not identify any substantial disruption to sleep quality. We observed decreased TST, SPT, and NREM2 duration and decreased sleep efficiency, as well as increased NREM1 duration in the children with moderate/ severe OSA when they were compared to the other subject groups. However, no evidence of impaired sleep quality was seen in the duration of SWS or REM in any of the SDB groups.

The finding that conventional measures of sleep architecture of the children in this study were not substantially disrupted, regardless of SDB severity, is in agreement with previous research which has demonstrated that, unlike adults, serious disruptions of sleep architecture by cortical arousals causing

sleep fragmentation are uncommon in children suffering from OSA.[17,20,23,35,36] We found that children with moderate/ severe OSA did have reduced TST and SPT compared to all three other subject groups, with control children having on average 57 min longer TST and 40 min longer SPT compared to the moderate/ severe group. This decreased TST was largely due to the increased sleep latency in the SDB groups and a non significant increase in WASO across the SDB groups compared with the control group. We are unsure as to why the control group fell asleep more quickly; however, this may have been due to comparatively less anxiety compared with the children with SDB who were attending the clinic for investigation and would be unlikely to affect sleep quality. Reduced SPT in children with OSA compared to control children has been reported previously in one study[18]; however, other studies have reported no significant differences.[20,37] Importantly, the children in our study did not have a significantly increased time available for sleep (TIB) compared to the control group, removing the possibility that the significantly lower TST and SPT seen in the children with moderate/ severe OSA was due to a decreased opportunity for sleep. The reduced TST observed in the moderate/ severe group in this study is unlikely to be the sole causative factor for the previously reported neurocognitive deficits in OSA. Average nightly sleep reported by parents from sleep diaries was not different between the groups and averaged 9-10 h per night. In support of this view, past research has established that daytime functioning can be impaired even when TST was not significantly reduced.[8,38-41]

We observed increased time spent in NREM1 and decreased time spent in NREM2 in the moderate/ severe OSA group compared to both the control and PS groups. This may have been caused by more frequent awakenings or by difficulty falling asleep. Our findings are supported by a recent paper which compared sleep continuity in children with and without SDB which found that mean NREM2 duration was significantly reduced in children with SDB, even when overall sleep amount of NREM2 expressed as a percentage of total sleep time was not significantly different.[17] Chervin et al. suggest that the mean duration of NREM2, the stage that occupies ~50% of the night in children, could provide a more sensitive measure of the effects of SDB on sleep architecture than conventional measures, which typically record only the proportion of each sleep stage.[17] Findings of decreased sleep continuity across the night using survival curve analyses in adults with mild SBD compared to controls have also been reported, and this method has also been suggested as a more sensitive measure of sleep disruption than conventional measures.[42]

The lack of any significant differences between groups when comparing the duration of SWS and REM has been reported previously[20] and suggests that these essential stages of sleep are strongly protected in children with SDB.

Our EEG power spectral analysis supports the findings from conventional measures that children with OSA do not show substantial differences in sleep quality across the night. No differences were seen over the whole night in EEG spectral power within any of the five frequency bands between SDB severity groups when examined as either absolute or relative power. The main effect on sleep architecture seen from conventional measures was the significantly reduced sleep latency observed in the control children when compared to the three SDB subject groups. This however had a minimal effect on EEG spectral distribution results, as the sleep latency was not included in the analysis.

Our results exhibited the expected fall in proportional EEG spectral power as the night progressed within the delta and theta frequency bands. This has previously been reported to be due to satiation of sleep debt.[43-45] However, EEG spectral power distribution across the night did not identify an overall difference in the decrease in delta power across the night between the groups, as would have occurred if there were a different rate of satiation of sleep homeostatic mechanisms.[43-45] Our finding that the delta spectral power in the SDB severity groups was not significantly different from that of the control group in the first hour of the night is enlightening, as past research has indicated that delta and theta power reflect sleep intensity[45,46]; such that an absence of increased activity in these frequencies early in the night suggests that sleep debt was not higher in the SDB children.[29,46,47] However, the interpretation of the significant decrease in delta power within the second hour in the moderate/ severe OSA group compared to all other subject groups is unclear. It may reflect an interaction between sleep disruption and waning homeostatic drive in the severe group, although this is contradicted by higher levels of delta and theta activity during subsequent hours.

We found no specific sleep stage where a substantial difference in EEG spectral power was observed between SDB severity groups and the control group. When comparing control to moderate/ severe OSA children the present study showed no increase in delta or theta activity during REM sleep, or in any stage of NREM sleep. Had sleep quality been affected, a significant difference in both delta and theta EEG spectral power between non-snoring children and the moderate/ severe OSA children would have been observed due to differences in slow wave activity. Also, increased delta and theta activity was not observed during wakefulness after sleep onset, as would have been expected if there was increased sleep pressure. Importantly, the lack of any substantial difference between groups in delta EEG spectral power in any sleep stage indicates that there was little difference in the degree of sleep disruption occurring during any of the sleep stages in any of the subject groups. Thus, our findings suggest that sleep in childhood OSA is protected through the night regardless of sleep stage or time of night. This suggests that the overall sleep quality of a child with SDB, as reflected by spectral analysis, does not appear to be different from a normal child free of SDB, even in cases of severe OSA.

CONCLUSIONS

Our findings did not support our hypothesis that spectral analysis of the EEG would provide a more sensitive measure of sleep disruption in children with SDB. Rather, the study has demonstrated that neither conventional analysis of sleep architecture nor EEG spectral analysis demonstrate significant disruptions to sleep quality in children with SDB. Thus, our study validates current conventional measures used for assessing sleep quality, indicating that they are no less accurate in identifying sleep disruption in children with SDB. We speculate that children exhibit a stronger sleep drive than adults which preserves sleep quality even in severe SDB and suggest that the

reduced daytime functioning previously reported in children with SDB may not be due to impaired sleep quality caused by sleep fragmentation.

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DISCLOSURE STATEMENT

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