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Paroxysmal activity and seizures associated with sleep breathing disorder in children: A possible overlap between diurnal and nocturnal symptoms

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ABSTRACT

Purpose: Sleep breathing disorders (SBD) can trigger paroxysmal events. We recently found a higher percentage of paroxysmal activity (PA) in a sample of Italian children with obstructive sleep apnea syndrome (OSAS) and no history of epilepsy. The signs of nocturnal seizures can overlap with sleep respiratory events. The aim of this study was to confirm the high frequency of PA or interictal epileptiform discharges (IEDs) during sleep in a cohort of Spanish children who underwent polysomonography (PSG) for suspected SBD and to ascertain the eventual presence of seizures by means of a video-PSG with an extended electroencephalogram (EEG).

Methods: PSG was performed in a population of children with no previous history of epileptic seizures recruited prospectively for suspected OSAS from January to December 2007. Recordings included at least 13 EEG channels.

Results: In total, 25 children (mean age, 6.6 ± 3.8 years, 14 males) were diagnosed with SBD, and 4/25 (16%) children met the criteria for OSAS and epilepsy, with IEDs and/or seizures during sleep. We diagnosed benign epilepsy with centro-temporal spikes in 2 cases, partial symptomatic epilepsy in one, and nocturnal frontal lobe epilepsy in another, while we found PA in 2 patients. The body mass index and the apnea–hypopnea index were significantly higher in children without IEDs/PA.

Conclusions: Our study demonstrated a close relationship between pediatric SBD, PA during sleep, and epilepsy that may aggravate the prognosis of SBD. Due to the possibility of an overlap of symptoms, a video-PSG with extended EEG montage is necessary.

1. Introduction

Sleep respiratory events can trigger seizures and interictal epileptiform discharges (IEDs) in patients with epilepsy, and epilepsy has been shown to improve significantly with the resolution of sleep breathing disorder (SBD).[1–8] The exacerbation of seizures could be related to sleep fragmentation and instability due to sleep respiratory events or/and hypoxemia.[9] It has been demonstrated that the association between PA/IEDs, seizures and sleep instability is significantly related to the occurrence of arousals, or of the cyclic alternating pattern (CAP) A phases, which serve as a promoting gate for seizures and PA in several forms of epilepsies.[9–13] It has also been reported that sleep respiratory events may trigger parasomnias in children by means of an increase of sleep instability, mediated by arousal or by burst of delta activity, supporting the hypothesis that parasomnias and seizures share the same trait of arousal activated phenomena.[11,13]

We recently reported a higher percentage of paroxysmal activity (PA) in the polysomnographic recordings (PSG) of an Italian cohort of non-epileptic children with obstructive sleep apnea syndrome (OSAS) and no history of epileptic seizures.[14] We found PA in 14.2%, although none of the children with primary snoring experienced PA during sleep. Children with PA and OSAS showed some clinical differences compared to those without PA: they were older, and had a longer disease history, a lower percentage of adenotonsillar hypertrophy, and slightly higher occurrence of perinatal injuries. Since the percentage of PA found in the full-night PSG of healthy children was 1.43%,[15] the percentage found in children with OSAS (14.2%) was higher than expected.[14]

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Several authors show that the occurrence of IEDs during sleep might be associated with cognitive and behavioral dysfunction in children who suffer from benign epilepsy with centro-temporal spikes (BECTS), in children with attention deficit hyperactivity disorder, in autistic children, or in children with language disorders.[16–21] It has recently been reported that in children with BECTS, the presence of IEDs for more than 1 year and early onset of seizures (before 8 years of age) are relevant markers potential learning difficulties.[22]

The demonstration of PA in children with OSAS may explain the neurocognitive dysfunction observed in these patients. In our study,[14] we used an extended electroencephalographic (EEG) recording that may be considered excessive for standard polysomnography, but insufficient for an epilepsy work-up. In our sample of children with OSAS, PA mostly occurred over the centro-temporal regions, suggesting some similarities with the IEDs in BECTS, even if we did not find seizures or ictal epileptic activity.[14]

The signs of nocturnal seizures can overlap with sleep respiratory events such as apnea, tachypnea, bradycardia, coughing, vocalizations, bruxism, and body or limb movements which may be part of a simple obstructive sleep apnea event or may represent autonomic and motor signs of a seizure.[23]

The differential diagnosis is challenging and requires a video-EEG-PSG recording (extended EEG, electrocardiogram, respiratory channels, electromyograms to provide information on motor activity and paroxysmal events during sleep).

The aim of this study was to confirm the high frequency of PA/IEDs during sleep in a cohort of Spanish children who underwent a PSG recording for a suspected SBD. We also show the value of video-PSG with an extended EEG montage when determining the eventual presence of seizures.

2. Methods

We recruited prospectively children for suspected OSAS at the Sleep Unit of the Clinical Neurophysiology Department of the University Hospital Gregorio Marañón in Madrid, Spain, because of their habitual snoring or apnea, or restless sleep, witnessed by parents. All patients were referred either from specialists (otolaryngologists, child neurologists, child pulmunologists) or from primary care providers. The diagnosis of OSAS was confirmed by a full-night video-EEG-PSG recording showing an obstructive apnea/hypopnea index >1, according to the criteria of the American Academy of Sleep Medicine.[24] Habitual snores with and an apnea/hypopnea index <1 were diagnosed with primary snoring.

Patients with a history of epilepsy, or seizures, or with a history of previous treatment of OSAS (including tonsillectomy and adenoidectomy), of acute or chronic cardiorespiratory or neuromuscolar diseases, dysmorphism, major craniophacial abnormalities or associated chromosomic syndromes, were excluded. Recruitment was done between January 2007 and December 2007.

2.1. Patients

A detailed personal and family history was obtained for all participants, together with a general clinical examination. The patients completed the Pediatric Daytime Sleepiness Scale (PDSS).[25] All patients also underwent an ENT examination to grade adenotonsillar hypertrophy. Tonsillar hypertrophy was graded according to a standardized scale ranging from 0 to 4.[26] Adenoid hypertrophy was graded according to Greenfeld et al.[27]

Patients routinely underwent a video-EEG-polygraphic daytime nap recording at our sleep center as screening for obstructive respiration. If the nap recording was negative, then we performed a full-night PSG recording, which demonstrated the presence of obstructive sleep respiratory events.

The local ethics committee approved the study protocol and all the children's parents gave their informed consent for the procedures.

2.2. Polysomnography and sleep stage scoring

Standard overnight video-PSG recordings were obtained with a computerized sleep recorder (Deltamed System). The video was analog, and visualized synchronized with the digital recording. The patients underwent the following recordings: a 13-channel EEG – electrodes: Fp (pre-frontal), Fz (frontal), C (central), T (mid-temporal), O (occipital), Pz (parietal), and Cz (vertex) with bipolar montages (see figures) – with the International "10–20" system to place electrodes in standardized scalp locations, an electrooculogram (EOG) (1 channel), a submental electromyogram (EMG), and an electrocardiogram. Thoracic and abdominal movements were recorded by inductance plethysmography (in most cases a bipolar montage which showed the sum of both movements in 1 channel), and airflow pressure by an oro-nasal cannula. Oxygen saturation was recorded continuously from a transcutaneous sensor (pulse-oximetry). A tibialis anterior electromyogram and/or deltoid electromyogram were also recorded.Sleep was subdivided into 30-s epochs, and sleep stages were scored according to the standard criteria of Rechtschaffen and Kales.[28] We evaluated the following parameters of sleep architecture: sleep period time (SPT defined as time from sleep onset to the end of the final sleep epoch minus wake time during sleep); sleep efficiency (defined as the percentage ratio between time from sleep onset to the end of the final sleep and time in bed); sleep-onset latency (time from lights out to sleep onset, which was further defined as the first of 2 consecutive epochs of stage 1 sleep or 1 epoch of any other stage, in minutes); REM latency (time from sleep onset to the first epoch of REM sleep); wakefulness after sleep onset (WASO, time spent awake between sleep onset and end of sleep, in minutes); percentage of SPT in stage 1, stage 2, and slow wave sleep (SWS, defined as the sum of the stages 3 and 4 percentage); percentage of REM sleep; and number of stage shifts per hour.

Sleep scoring was performed by one of the investigators (RPA).

2.3. Interictal epileptiform discharges/PA

The EEG was reviewed blind by one of the authors (RPA), and each screen contained 15 s of recording. The presence of spikes (transient, clearly distinguishable from background activity lasting 20–70 ms) and sharp waves (same as spikes, but lasting 70–200 ms), either alone or accompanied by slow waves (the slow wave being of a higher amplitude than the spike or the sharp wave) occurring in isolation or in bursts was considered as representing IEDs/PA, according to the definitions of the International Federation of Societies for Clinical Neurophysiology.[29] In order to consider a subject as having IEDs/PA, at least 10 spikes and/or sharp waves had to be present.

2.4. Arousal analysis

Arousals were visually detected on EEG and marked by one of the investigators (SM), who was blind to the children's group, age, and sex, following the criteria reported in the American Academy of Sleep Medicine manual for the scoring of sleep and associated events, and each epoch contained 30 s of recording.[25] EEG frequency shifts longer than 3 s were analyzed, associated in REM sleep with an increase in submental EMG amplitude. We also estimated as an 'activation' burts of rhythmic theta-activity (4–7 Hz), taking age into account. A minimum of 10 s of continuous sleep interval was needed to score a new arousal.

2.5. Sleep respiration analysis

Central, obstructive, and mixed apnea events were counted according to the criteria established by the American Academy of Sleep Medicine.[30] Obstructive apnea was scored as a >90% drop in the signal amplitude of airflow for >90% of the entire event, compared with the pre-event baseline amplitude, with continued chest wall and abdominal movement, for a duration of at least 2 breaths. Central apnea was defined as the absence of airflow, with the cessation of respiratory effort lasting more than 20 s or at least 2 missed breaths (or the duration of 2 baseline breaths), and was associated with an arousal, an awakening, or a >3% desaturation; central apnea occurring after gross body movements or after sighs was not considered a pathologic finding. Mixed apnea was defined as apnea that usually began as central apnea and ended as obstructive apnea, according to changes in the chest, abdominal, and flow traces. An event was scored as hypopnea if there was a >50% drop in airflow signal amplitude compared with the pre-event baseline amplitude for at least 90% of the duration of the event; the event must have lasted at least 2 missed breaths and have been associated with an arousal, awakening, or a >3% desaturation. The apnea–hypopnea index was defined as the average number of apneas and hypopneas and arousals related to respiratory events per hour of sleep.

2.6. Study design

We defined 2 separate groups on the basis of the presence or absence of PA, IEDs, or ictal activity. Some children underwent a nap video-polygraphic recording only and were excluded from the statistical analysis of sleep parameters.

2.7. Statistical analyses

Data are expressed as means ± SD. The Mann–Whitney U or χ^2 test was used as appropriate to compare data. p values less than 0.05 were considered statistically significant.

3. Results

We studied 126 consecutive pediatric patients during the period January to December 2007. All of them had a nap EEG-polygraphic recording as screening for obstructive respiration followed by a full-night PSG with an extended EEG recording (routinely used in our Sleep Unit for suspected pediatric sleep disorder). Twenty-five patients (mean age, 6.6 ± 3.8 years, 14 males) met the criteria for a diagnosis of SBD, and 6 met the criteria for SBD and also had PA/IEDs and/or seizures. More specifically, 4/25 (16%) met the criteria for OSAS and epilepsy, with IEDs and seizures during sleep. In one patient, the video-PSG showed many IEDs with minor motor events, mostly during SWS, and a diagnosis of nocturnal frontal lobe epilepsy, was established and confirmed by magnetic resonance imaging (MRI) that disclosed signs of focal and minimal cortical dysplasia involving the right Sylvian fissure. A further 2 cases met the criteria for BECTS, with partial motor seizures during sleep. The video-EEG-PSG showed left, right, or bilateral interictal centro-temporal and rolandic spikes and partial motor seizures during sleep. The last case (number 2), received a diagnosis of focal symptomatic epilepsy, the MRI performed after the PSG study, revealed the presence of a malacic area on the left subcortical white matter, and the neurologic exam a mild right hemiparesis.

Figs. 1 and 2 are examples of interictal activity during sleep.

The partial motor seizures were associated with tachycardia and tachypnea (lasting about 20–30 s), and rhythmic slow activity on the EEG (theta and delta rhythms) (patients number 1–3). The partial motor seizures were characterized from the clinical point of view by the dystonic posture of the legs, arms, and trunk in 2 cases and right jerks in the other case. In patient number 1, we also found an ictal episode lasting about 20 s, with spike activity and rhythmic slow waves over the right centro-temporal and rolandic regions, occurring concomitantly with central apnea, and bradycardia. The episode was followed by an arousal and SaO_2 desaturation, and appeared during the REM stage (see Fig. 3). All seizures were followed by stage shifts or, more rarely, by an awakening.

In 2 cases (2/25) (8%), we found only PA, mostly represented by centro-temporal spikes. One of the cases met the criteria for primary snoring, whereas the other met the criteria for OSAS.

Table 1 shows the clinical and anthropometric parameters of children with PA/IEDS and seizures: we observed snoring (6/6), mouth breathing (6/6), daytime sleepiness (2/6), allergic diseases (2/6), poor growth (2/6), parasomnias (bruxism and enuresis) (4/6), and a family history of epilepsy (1/6). None of the patients had a history of atypical parasomnia.

Among children with only a diagnosis of OSAS or primary snoring (19 patients), 9 did not undergo a full-night PSG recording and met the criteria for primary snoring (5 had flow limitation, as detected by the nasal cannula, with sleep breathing effort as expressed by paradoxical breathing, for most of the nap recordings). Of the children with only SBD who underwent a full-night video-EEG-PSG recording (10 patients), 7 met the criteria for OSAS and 3 for snoring (1 with flow limitation in the nasal cannula and sleep breathing effort for most of the night). Among children with PA/IEds and or seizures (6 subjects), one (patient number 6) underwent only a nap recording and met the criteria for snoring plus PA.

Table 2 shows the clinical and anthropometric features of patients with IEDs or PA or seizures (6/25) compared to those with

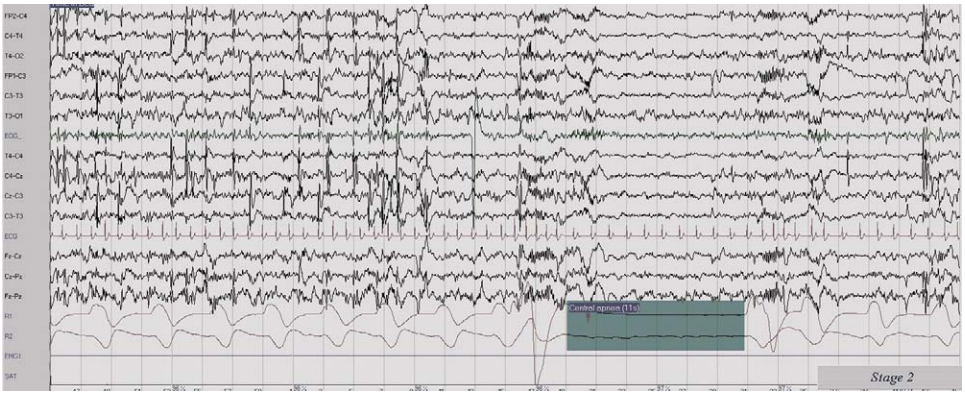

Fig. 1. Patient 4, 6.6 years. Polysomnographic recording showing the presence of increased bilateral interictal centro-temporal and rolandic spikes during stage 2 NREM. Central apnea after a sigh occurred was observed in the respiratory channels (R1, oro-nasal cannula; R2, thoracic–abdominal movements). 30-s sleep epoch, 20 μV.

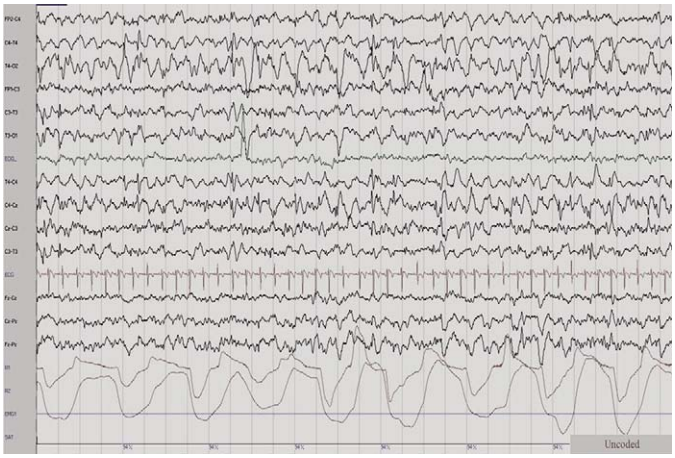

Fig. 2. Patient 1, 5.4 years. Polysomnographic recording showing the presence of interictal rhythmic slow waves over the right centro-temporal and rolandic regions. Snoring and flow limitations at the oro-nasal cannula (R1) during stage REM. 30-s sleep epoch, 20 μV

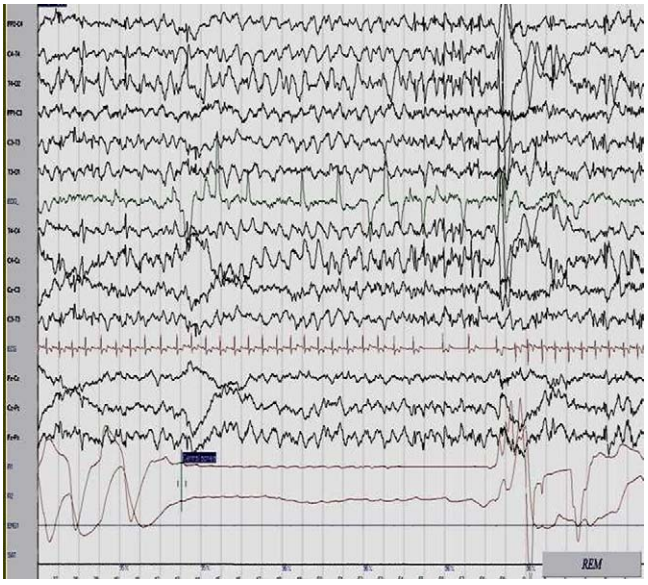

Fig. 3. Patient 1, 5.4 years. Ictal spiky activity + rhythmic slow waves over the right centro-temporal and rolandic regions + central apnea with bradycardia and O_2 desaturation followed by an arousal during REM stage. 30-s sleep epoch, 20 μV.

only a diagnosis of SBD (19/25). The body mass index was significantly higher in children without IEDs/PA.

Table 3 shows the sleep and respiratory parameters of children with SBD and IEDs/PA and or seizures (6/25) compared to those with only SBD who underwent full-night PSG (10/25). There were no statistical differences between the 2 groups, but the body mass index and the apnea–hypopnea index were significantly higher in children without IEDs/PA.

4. Discussion

Our study confirmed a close relationship between pediatric SBD and paroxysmal activity during sleep, as recently observed.[14] We also reported a close relationship between SBD and epilepsy: 4 out of 25 patient with suspected OSAS also had epilepsy. We diagnosed BECTS in 2 cases, partial symptomatic epilepsy in one, and nocturnal frontal lobe epilepsy in another, whereas we found PA in 2 patients.

Considering that population based studies in developed countries reported a prevalence rates of 3.6–4.2/1000 for childhood epilepsy, and that PA has been found in 1.43% of healthy children studied by PSG, we might assume that the frequency of IEDs/PA and of epilepsy in our children with SDB is higher than that expected in healthy children or in general population.[31–33]

Moreover, in this small cohort of Spanish children with SBD, the percentage of children with only PA was lower than that found in our previous study (8% of patients compared to 14.2%), in which we did not observe seizures.[14] This feature is interesting, and can be explained by the more extended EEG montage that we routinely use in children, which allowed us to better detect the IEDs and ictal activity, as well as the analog video with a full image of the patient (no top sheet) for a better definition of motor activity during sleep.

We found few differences between the group of children with comorbid SBD and epilepsy and those without, although this result might be influenced by the small sample size. We found a higher body mass index in children with OSAS or primary snoring without PA/IEDs and/or seizures, and in patients with comorbid conditions we found MRI alterations in 2 cases out of 6. In our previous study,[14] children with PA were older, predominantly boys, and had

Table 1
Clinical and anthropometric characteristics of patients with SBD and IEDs with or without seizures.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6
Sex	Female	Male	Male	Female	Male	Male
Age (years)	5.4	7.3	6.8	4.9	8.1	6
Pediatric Daytime Sleepiness Scale	16	21	10	11	14	15
Body mass index (kg/m^2)	15.8	15.4	13.3	15.9	18.9	15.4
Adenotonsillar hypertrophy	Yes/yes	Yes/yes	Yes/no	Yes/no	No	Yes/yes
Allergies	No	Cow milk	Pollen	No	No	No
Parasomnias	No	Bruxism	Enuresis	Enuresis and bruxism	Enuresis	No
Symptoms of hyperactivity and attention deficit	Yes	No	Yes	No	Yes (plus learning difficulties)	No
Interictal epileptiform discharges (centro-temporal, and rolandic spikes)	Right + left	Left	Right + left	Left	Paroxysmal arousals	Right
Seizures (total number recorded during nap + overnight recordings)	Partial motor, right jerks, cough (3)	Partial motor, dystonic posture and cough (5)	Partial motor and sometimes dystonic posture with automatisms (12)	No	Minor motor events (right jerks), automatisms, final dystonic extension of the limbs (12)	No
Antiepileptic treatment	Valproic acid	No	Valproic acid	No	No	No

Table 2
Clinical and anthropometric characteristics of patients with SBD and IEDs/PA and/or seizures (Group 1) compared to those without (Group 2). Data are expressed as mean ± standard deviation or number.

	Group 1 ($n=6$)	Group 2 ($n=19$)
Male	4	10
Age (years)	6.41 ± 1.2	6.63 ± 1.1
Pediatric Daytime Sleepiness Scales	14.5 ± 3.9	12.73 ± 2.8
Body mass index (kg/m^2)*	15.78 ± 1.8	21.76 ± 5.5
Adenotonsillar hypertrophy	5	12
Allergies	2	7
Parasomnias	4	6
Symptoms of hyperactivity and attention deficit	3	13
Apnea–hypopnea index (ev/h)	1.33 ± 1.02	2.82 ± 6.8
Mean overnight oxygen saturation (%)	96.16 ± 1.5	95.76 ± 2.4

* $p < 0.05$ (Mann–Whitney test).

Table 3
Sleep polysomnographic parameters of patients who underwent full-night polysomnography. The table compares patients suffering from sleep breathing disorder and interctical epileptiform discharges/paroxysmal activity and/or seizures (Group 3) with those who did not (Group 4). Data are expressed as mean ± standard deviation, or number.

	Group 3 ($n=5$)	Group 4 ($n=10$)
Age (years)	6.5 ± 1.32	7.93 ± 4.2
Male	3	3
Pediatric Daytime Sleepiness Scale	14.4 ± 4.4	12.50 ± 2.6
Body mass index (kg/m^2)*	15.8 ± 2	22.6 ± 4.2
Sleep architecture parameters		
Sleep period time (min)	426 ± 29.3	394.2 ± 37.6
Sleep-onset latency (min)	18.1 ± 2.8	27.65 ± 23.4
First REM latency (min)	111.4 ± 42.9	139.1 ± 28.3
Wakefulness after sleep onset (min)	29.2 ± 29.1	36.7 ± 36.1
Stage shifts/h	13.64 ± 3.7	15.43 ± 4.3
Sleep efficiency (%)	89.44 ± 6.2	95.04 ± 8
Stage 1%	12.32 ± 6	11 ± 4.9
Stage 2%	28.82 ± 5.5	27.07 ± 4.8
Slow wave sleep (stage 3 + stage 4) (%)	42.04 ± 7.1	47.17 ± 8.2
REM (%)	16.68 ± 2.1	14.79 ± 4.9
Arousal index/h	20.07 ± 6.9	17.13 ± 6.9
Sleep respiratory parameters		
Apnea–hypopnea index (ev/h)**	1.60 ± 0.9	5.36 ± 8.8
Mean overnight oxygen saturation (%)	96.4 ± 1.5	96.6 ± 1.2

* $p < 0.01$ (Mann–Whitney test).
** $p < 0.05$ (Mann–Whitney test).

a longer history of OSAS, a lower percentage of adenotonsillar hypertrophy, and a slightly higher percentage of perinatal injuries than children without PA. These results suggest that children with SBD and neurological conditions may have an additional risk of developing PA/IEDs and/or epilepsy. Future video-PSG-EEG-based research in different populations of children with SBD is needed to confirm the high percentage of PA/IEDs and/or epilepsy in pediatric OSAS in order to better define the phenotype of this subpopulation of children with SBD. Unfortunately, we are unable to explain why we found a higher body mass index in children without PA/IEDs, and if they really belong to a different subpopulation compared to those with PA/IEDs and/or epilepsy.

In the present study, we found a mild form of OSAS or primary snoring in children with IEDs/PA and/or seizures, while in the previous study all children were diagnosed with OSAS and PA.[14] We are unable to explain this discrepancy, due to the small sample size and the absence of data in the literature. We found nocturnal epileptic seizures in most of the cases with IEDs/PA, suggesting that some symptoms of SBD (e.g., snoring and/or apnea, vocalizations, coughing, tachypnea, tachycardia, bruxism, salivation, and head, body or limb movements during sleep) can mimic OSAS, thus leading to confusion between symptoms of epileptic seizures and obstructive sleep respiratory events.[23] This could lead pediatricians trained in sleep disorders to overestimate nocturnal respiratory symptoms in children and eventually misdiagnose paroxysmal nocturnal events not clinically suspected, as not prior seizures or nighttime events had been recognized. However, we were unable to make a differential diagnosis between the 2 subgroups of children with SBD (with or without IEDs/PA, and/or epilepsy), because of the lack of data in the literature, and we strongly recommend considering the possibility of an overlap syndrome. Furthermore, we suggest that physicians bear in mind an overlap syndrome in those children who report symptoms of SBD and no signs (obesity and/or adenotonsillar hypertrophy), even if we cannot define a phenotype.

We think that our study is limited by the lack of an objective method to identify IEDs/PS; in addition, we did not use horizontal dipoles to better differentiate the EEG activity from an artefact.

A further 10/25 patients did not undergo a full-night PSG, and this may significantly limit the accuracy of the diagnosis of SBD and of the detection of EEG interictal and ictal activity, due to the protocol followed by the Sleep Unit, used to screen children with suspected SDB, in order to limit the waiting list for polysomnography.

Moreover, we diagnosed BECTS in two cases with epilepsy and SBD, and we had already found some EEG similarities with BECTS in our previous study.[14] The present study may reinforce the hypothesis of a relationship between BECTS and pediatric SBD.

It has recently been questioned whether "benign" rolandic epilepsy is associated with long-term cognitive outcome,[34] considering that that IEDs may disrupt cognitive functions and impair learning abilities and memory.[35] The neuropsychological assessment in children affected by BECTS revealed disorders in visuospatial short-term memory, attention span, cognitive flexibility, verbal fluency, phonological awareness, visuoperceptual skills, and academic performance, with a significant improvement after the remission of IEDs during sleep.[20,22] Many children with SBD show similar neurobehavioral complications. Intermittent hypoxemia and subtle changes in sleep architecture might be involved in the pathogenesis of neurocognitive deficits in pediatric OSAS,[36] and the association between BECTS and SBD may aggravate the prognosis of neuropsychological deficits.

Due to the possibility of an overlap between diurnal and nocturnal symptoms in this population, a nap EEG-polygraphic recording or a video-PSG (with extended EEG leads and montage) is necessary for an accurate diagnosis. Future studies should thoroughly analyze the neuropsychological features of children with SBD and PA/IEDs and/or epilepsy.

Conflict of interest statement

None of the authors have any conflicts of interest to declare.

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