Is a 2-Night Polysomnographic Study Necessary in Childhood Sleep-Related Disordered Breathing?*

Albert M. Li, MRCP; Yun Kwok Wing, MRCPsych; Agnes Cheung; Dorothy Chan, MRCP; Crover Ho, RPSGT; Stephen Hui, MRCP; and Tai Fai Fok, MD, FRCP

Background and objectives: **There are limited data on the night-to-night variability of childhood sleep-related disordered breathing (SDB). We aim to assess for the presence of first-night effect (FNE) and to examine whether a single-night sleep study is adequate in the assessment of childhood SDB.**
Design: **In a case-control study investigating whether obesity is a risk factor for childhood SDB, the night-to-night variability of sleep and respiratory variables were studied.**
Participants and setting: **Forty-six obese children from a pediatric obesity clinic and 44 age- and sex-matched normal weight control subjects from local schools.**
Interventions: **All subjects underwent two consecutive overnight polysomnographic studies. An obstructive apnea index (OAI) ≥ 1/h was considered diagnostic of SDB.**
Results: **The mean age of the children was 11.21 years (SD 2.21). Forty-four obese children and 43 control subjects completed the 2-night study. Based on the criterion of the worst OAI over the 2 nights, 13 subjects were found to have SDB, 12 subjects were primary snorers, and 62 were normal subjects. In all subjects, the sleep efficiency improved and sleep-onset latency was reduced on the second night. While there was a rebound of rapid eye movement sleep with the associated worsening of respiratory indexes (mainly accounted for by an increase in central apneas and hypopneas) evident in normal subjects, there was a significant improvement of respiratory disturbances in the SDB group on the second night. The first-night polysomnography would have correctly identified 84.6% of cases as defined by the criteria of the worst OAI over the 2 nights. All cases missed by the first-night study had only borderline OAI.**
Conclusions: **The phenomenon of FNE in children was well demonstrated in our study. We proposed that a single-night sleep study is adequate and more cost-effective in assessing for childhood SDB.** ***(CHEST 2004; 126:1467–1472)***

Key words: children; first-night effect; sleep-related disordered breathing

Abbreviations: AHI = apnea-hypopnea index; $ETCO_2$ = end-tidal carbon dioxide; FNE = first-night effect; OAI = obstructive apnea index; ODI = oxygen desaturation index; REM = rapid eye movement; SDB = sleep-related disordered breathing; SWS = slow-wave sleep; UARS = upper airway resistance syndrome

Childhood sleep-related disordered breathing (SDB) is characterized by recurrent events of partial or complete upper airway obstruction during sleep, resulting in the disruption of normal ventilation and sleep patterns.[1] The condition is increasingly being recognized, and studies[2–4] have found the prevalence to be between 0.7% and 10.3% among the pediatric population. Overnight polysomnography remains the "gold standard" in the diagnosis of SDB.[5–8] However, very little is known regarding the night-to-night variability of polysomnographic findings in children.[7] It is still uncertain whether the severity and frequency of the obstructive episodes in childhood SDB remains the same from 1 night to the other. In adults, incorrectly diagnosing SDB as primary snor-

*From the Departments of Paediatrics (Drs. Li, Chan, Hui, and Fok) and Psychiatry (Dr. Wing, Ms. Cheung, and Mr. Ho), Prince of Wales and Shatin Hospital, The Chinese University of Hong Kong, Shatin, Hong Kong, SAR.
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Correspondence to: Yun Kwok Wing, MRCPsych, Department of Psychiatry, Room 7010, Seventh Floor, Shatin Hospital, Shatin, The Chinese University of Hong Kong, Hong Kong; e-mail: ykwing@cuhk.edu.hk

ing after a single-night recording has been reported in some studies.[9–11] A disturbed sleep pattern, which is known as the *first-night effect* (FNE) caused by

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artificial sleep laboratory environment and continuous visual surveillance, is well described and may influence the result reliability of a single-night study.[11–14] The FNE is known to reduce the amount of rapid eye movement (REM) sleep; with childhood SDB being more severe during REM sleep, single-night polysomnography may be expected to generate a high rate of false-negative findings. The aim of this study was to assess the night-to-night variability of sleep architecture, and the presence and severity of SDB in children as measured by polysomnography performed on 2 consecutive nights. This would provide important data on the feasibility of a single-night study in the assessment of SDB in children.

Materials and Methods

Subject Selection

From 1998 to 1999, we recruited consecutive children aged 7 to 15 years from the pediatric obesity clinic at our university hospital. They were all referrals from primary care physicians for healthy living and dietary advice. Normal weight-, age-, and sex-matched control subjects were randomly selected from local schools. Children with known clinical syndromes such as Down syndrome and Prader-Willi syndrome, neuromuscular disease, laryngomalacia, or upper airway surgery were excluded. Obese children were defined as those with actual weights $\geq$ 120% of the ideal weight for height, whereas normal-weight control subjects had an ideal weight for height from 80 to 120%.[15] A detailed description of the methodology can be found in our previous publication.[16] The university ethics committee approved the study, and all the subjects and their parents gave written informed consent.

Physical Examination and Sleep Questionnaires

All children and their parents completed a questionnaire pertaining to symptoms of sleep-disordered breathing as well as sleep habits and other sleep disorders. The height and weight of each child were measured by the standard standiometer and the digital floor scale (Detector model 6029; SECA; Hamburg, Germany), respectively.

Polysomnography and Main Respiratory Outcome Measures

All children underwent two consecutive standard overnight polysomnographic studies to be followed by a daytime multiple sleep latency test at the third day with a CNS 1000P polygraph (CNS; Chanhassen, MN). For a detailed description of the standard polysomnography procedure and scoring criteria for the respiratory disturbances, please refer to our previous article.[16] Briefly, standard overnight polysomnography recorded the following parameters: EEG, electro-oculogram, electromyogram of mentalis activity and bilateral anterior tibialis, and respiratory movements of the rib cage and abdomen. The position of the subject, respiratory airflow (thermistor signals), arterial oxyhemoglobin saturation, and end-tidal carbon dioxide ($ETCO_2$) were also measured. All computerized sleep data were further visually edited. The terminology and main scoring criteria for the respiratory outcomes were reported previously, and closely followed the recommendation of American Thoracic Society on the polysomnography scoring.[1,16] Briefly, obstructive apnea was defined as absence of airflow with persistent respiratory effort lasting longer than two baseline breaths, irrespective of arterial oxyhemoglobin saturation changes. Central apnea was defined as absence of respiratory effort associated with absence of airflow. Those of $>$ 20 s with or without oxygen desaturation, and those of any duration but associated with oxygen desaturation of at least 4% were quantified. The oxygen desaturation index (ODI) was defined as the total number of dips in arterial oxygen saturation $>$ 4% per hour of sleep. The apnea-hypopnea index (AHI) is the total number of apneas and hypopneas per hour of actual sleep time. The obstructive apnea index (OAI) was defined as the total number of obstructive apneas per hour of actual sleep time. The controversy of the diagnostic criterion for childhood SDB has been discussed.[5–7] As there is no widely accepted diagnostic criterion for classic SDB in children, we chose a more conventional and well-accepted criterion, OAI $\geq$ 1, as the diagnostic cut-off of SDB in the current study. The concept of childhood SDB is quickly evolving, and is often seen as a spectrum from normal upper airway to resistance and obstruction.[7] However, controversies existed on the diagnostic concept and standard of upper airway resistance syndrome (UARS) in children.[7] The 2002 American Academy of Pediatrics Clinical practice guideline for diagnosis and management of childhood obstructive sleep apnea did not explicitly acknowledge the existence of UARS and the differentiation of UARS from primary snoring.[5–7] In view of the controversy and lack of a standard guideline, we did not further differentiate UARS from primary snoring in the current study. We classified an individual with a normal OAI but with nocturnal snoring for $>$ 4 nights per week as suffering from primary snoring.[5–6] A normal control subject has a normal OAI and has nocturnal snoring for $\leq$ 4 nights per week.

Statistical Analysis

The statistical analysis was performed by using SPSS for Windows (Release 10.0; SPSS; Chicago, IL). The descriptive statistics were expressed as mean and SD. The variables were compared by paired-samples t test and one-way analysis of variance for more than two groups. χ^2 test was used to test for the categorical variables. κ coefficient was used to compare the night-to-night variability. The level of significance was set at 5% for all comparisons.

Results

Forty-six obese children and 44 normal-weight control subjects were studied (mean age, 11.21 years; SD 2.21), were studied. All except three subjects underwent 2 consecutive nights of sleep assessment. Two of these subjects were obese children who were found to have very severe SDB (OAI $>$ 15) on the first night, and required immediate treatment with continuous positive airway pressure on the following night. The third subject (control) had a signaling

Table 1—*Demographic Characteristics of the Subjects

Characteristics	SDB	Primary Snorer	Normal	p Value
Total subjects, No.	13	12	62	
Age, yr	10.5 (2.6)	11.83 (1.8)	11.19 (2.23)	NS
Boys/girls, No.	13/0	8/4	38/24	$p < 0.05$
Body weight, kg	65.75 (21.9)	56.59 (20.61)	48.52 (18.1)	SDB > normal†
Body height, m	1.5 (0.17)	1.55 (0.13)	1.48 (0.13)	NS
Body mass index	28.86 (5.73)	22.96 (5.81)	21.61 (5.62)	SDB > primary snorer, normal†
Ideal body weight for height,‡ %	165.81 (34.07)	126.23 (27.74)	124.18 (30.56)	SDB > primary snorer, normal†

*Data are presented as mean (SD) unless otherwise indicated. NS = not significant.
†One-way analysis of variance *post hoc* test, least significant difference. Statistically significant, $p < 0.05$.
‡(Actual body weight/ideal body weight) × 100%.

problem on the second night. The results from the three subjects were excluded in the final analysis.

If based on the worst OAI data obtained on any single night, 13 children (12 obese and 1 normal-weight children) were found to have an OAI ≥ 1 (SDB group). Twelve subjects had primary snoring, and the rest (n = 62) were normal control subjects (non-SDB group). The demographic data of the three groups are shown in Table 1. The SDB group was significantly heavier than the non-SDB group, as most SDB cases were obese children.

The sleep architecture and respiratory indexes of the SDB subjects over the 2 nights were shown in Tables 2, 3. The actual sleep time, sleep efficiency, and sleep-onset latency improved significantly on the second night. There was a trend toward reduction in stage 2 sleep and increase in slow-wave sleep (SWS) among the SDB cases, but did not reach statistical significance. Both AHI and ODI showed improvement on the second night.

The sleep architecture and respiratory indexes of the non-SDB subjects are shown in Tables 4–7. Similar to that seen in the SDB group, the actual sleep time, sleep efficiency, and sleep-onset latency improved significantly on the second night. The phenomenon of REM sleep rebound was evident on the second night only in the normal control subjects. There was no significant difference in respiratory indexes over the 2 nights among the primary snorers. There was, however, statistically significant increase in AHI and hypopnea index in the normal control subjects, accountable for by mainly an increase in central respiratory disturbances (for example, central apnea index increased from 0.24 (SD 0.36) to 0.36 (SD 0.4), $p < 0.005$) [Table 7].

The first-night polysomnography would have correctly identified 11 of the 13 cases (84.6%) if the worst OAI over any single night was used as the criterion ($\kappa = 0.9$). The cases missed by the single first-night polysomnography had only borderline OAI. In order to ascertain the potential influence of night-to-night variation on sleep position, the percentage of total sleep time spent in the four different sleep positions (prone, right lateral, left lateral, and supine) of SDB and non-SDB cases over the 2 nights were assessed (Table 8). There was no significant difference between the 2 nights.

Table 2—*Sleep Architecture of SDB Cases (n = 13) Cases

Variables	Night 1	Night 2	p Value
Total time in bed, min	543.7 (17.95)	544.54 (13.43)	NS
Actual sleep time, min	436.46 (51.26)	487.88 (41.47)	< 0.001
Sleep efficiency, %	80.27 (8.9)	89.53 (6.4)	< 0.001
Sleep-onset latency, min	23.96 (20.09)	13.23 (14.95)	< 0.05
REM onset latency, min	128.77 (43.5)	138.73 (45.62)	NS
Stage 1 sleep, %	5.37 (2.14)	5.17 (3.82)	NS
Stage 2 sleep, %	43.21 (10.96)	41.95 (7.8)	NS
Stage 3 sleep, %	7.45 (3.4)	8.63 (3.57)	NS
Stage 4 sleep, %	22.14 (6.29)	23.86 (5.57)	NS
SWS,† %	29.59 (7.79)	32.49 (7.25)	NS
REM sleep stage, %	21.82 (6.64)	20.4 (3.63)	NS

*Data are presented as mean (SD); see Table 1 for expansion of abbreviation.
†Stage 3 and stage 4 sleep.

Discussion

In this study that compared 2 consecutive nights of polysomnography recordings, a typical FNE was

Table 3—*Respiratory Indexes of SDB Cases (n = 13)

Variables	Night 1	Night 2	p Value
Central apnea index	1.36 (3.13)	0.42 (0.57)	NS
OAI	5.58 (5.75)	5.22 (5.53)	NS
Apnea index	6.94 (7.81)	5.64 (5.85)	NS
AHI	16.65 (11.58)	12.32 (10.57)	< 0.05
Hypopnea index	9.71 (5.55)	6.68 (5.74)	< 0.05
ODI	17.72 (15.26)	13.27 (10.66)	< 0.05
Peak $ETCO_2$,† mm Hg	50.73 (6.21)	52.73 (18.53)	NS
Arousal index	8.66 (5.92)	9.44 (6.43)	NS

*Data are presented as mean (SD); see Table 1 for expansion of abbreviation.
†Only 10 subjects had $ETCO_2$ data on both nights.

Table 4—*Sleep Architecture of Primary Snorers (n = 12)

Variables	Night 1	Night 2	p Value
Total time in bed, min	546.13 (7.16)	539.88 (28.95)	NS
Actual sleep time, min	378.96 (108.77)	447.88 (57.84)	< 0.05
Sleep efficiency, %	69.39 (19.95)	83.03 (10.17)	< 0.05
Sleep-onset latency, min	48.67 (31.8)	27.46 (27.09)	< 0.05
REM-onset latency, min	120.79 (49.18)	125.67 (55.58)	NS
Stage 1 sleep, %	8.91 (4.27)	7.06 (3.93)	< 0.05
Stage 2 sleep, %	46.79 (8.03)	47.02 (7.31)	NS
Stage 3 sleep, %	6.43 (1.91)	6.16 (2.09)	NS
Stage 4 sleep, %	22.27 (8.27)	21.05 (6.88)	NS
REM sleep stage, %	15.6 (6.51)	18.89 (4.23)	NS

*Data are presented as mean (SD); see Table 1 for expansion of abbreviation.

Table 6—*Sleep Architecture of Normal Subjects (n = 62)

Variables	Night 1	Night 2	p Value
Total time in bed, min	542.32 (13.51)	542.31 (28.61)	NS
Actual sleep time, min	447.45 (55.26)	489.66 (32.82)	< 0.001
Sleep efficiency, %	82.54 (10.27)	90.31 (4.24)	< 0.001
Sleep-onset latency, min	26.25 (23.62)	16.54 (14.43)	< 0.001
REM-onset latency, min	129.65 (55.43)	115.81 (40.36)	< 0.05
Stage 1 sleep, %	5.79 (2.8)	4.98 (2.54)	< 0.05
Stage 2 sleep, %	51.02 (7.42)	48.41 (6.57)	< 0.001
Stage 3 sleep, %	7.1 (3.38)	6.73 (3.1)	NS
Stage 4 sleep, %	17.51 (7.7)	18.44 (7.11)	NS
REM sleep stage, %	18.57 (4.04)	21.62 (4.02)	< 0.001

*Data are presented as mean (SD); see Table 1 for expansion of abbreviation.

demonstrated. A single-night sleep study would have correctly identified 84.6% of the cases if the worst OAI of the 2 nights was used as the diagnostic criterion of SDB.

The phenomenon of FNE has been well reported in the adult population, and it has been suggested that age plays a significant part in its etiology, with evidence showing older subjects experiencing greater FNE.[17] But our findings and that of several other studies[12,18] would argue against that. To our knowledge, data assessing the night-to-night variability of childhood SDB severity and sleep architecture is very limited. Rebuffat et al[19] studied the night-to-night variability in sleep characteristics and frequency of apneas in healthy infants and found no significant variability between 2 consecutive nights. However, a habituation nap that preceded the actual recordings could have affected the result. Katz et al[20] performed polysomnography on 30 snoring children with a mean age of 4.1 years, and found that the clinical diagnosis of either SDB or primary snoring remained the same in all subjects tested. No statistically significant differences in sleep or respiratory variables were observed between the nights except for the percentage of total sleep time in stage 2 sleep, which was slightly higher on the second night. However, the sleep assessments were not performed on consecutive nights, and in fact they were done 7 to 27 days apart; thus, both nights could be considered as a first night in the laboratory. In our study, FNE was evident in all three groups of subjects tested, namely SDB subjects, primary snorers, and normal control subjects. The effect was most marked in the normal control group, with statistically significant decrease in stage 1 and 2 sleep on the second night. REM sleep rebound was only seen in the normal subjects, whereas a nonsignificant increase in SWS rebound was seen in the SDB group. Why there should be a discrepancy between the two groups in terms of FNE is unclear. This illustrates the complex relationship between sleep, respiratory function, and FNE.

Contrary to our initial hypothesis that there might be worsening of SDB indexes on the second night, we found that there was actual significant improvement in AHI of the SDB subjects on the second night. We initially thought that the improvement was because the subjects were more at ease on the

Table 5—*Respiratory Indexes of Primary Snorers (Night 1 vs Night 2) [n = 12]

Variables	Night 1	Night 2	p Value
Central apnea index	0.2 (0.26)	0.5 (0.9)	NS
OAI	0.19 (0.26)	0.2 (0.33)	NS
Apnea index	0.39 (0.33)	0.71 (0.92)	NS
AHI	2.34 (1.46)	2.46 (1.26)	NS
Hypopnea index	1.95 (1.35)	1.76 (0.95)	NS
ODI	1.49 (1.46)	1.52 (2.08)	NS
Peak $ETCO_2$,† mm Hg	48.99 (2.52)	49.88 (3.65)	NS
Arousal index	4.29 (2.02)	4.78 (1.87)	NS

*Data are presented as mean (SD); see Table 1 for expansion of abbreviation.

†Only nine subjects had $ETCO_2$ data on both nights.

Table 7—*Respiratory Indices of Normal Subjects (Night 1 vs Night 2) [n = 62]

Variables	Night 1	Night 2	p Value
Central apnea index	0.24 (0.36)	0.36 (0.4)	< 0.005
OAI	0.12 (0.21)	0.15 (0.21)	NS
Apnea index	0.36 (0.4)	0.5 (0.46)	< 0.005
AHI	1.56 (1.7)	2.09 (1.85)	< 0.001
Hypopnea index	1.2 (1.52)	1.59 (1.71)	< 0.005
ODI	1.34 (1.57)	1.67 (1.82)	NS
Peak $ETCO_2$,† mm Hg	49.59 (9.68)	51.14 (13.16)	NS
Arousal index	4.36 (2.57)	4.56 (2.47)	NS

*Data are presented as mean (SD); see Table 1 for expansion of abbreviation.

†Only 52 subjects had $ETCO_2$ data on both nights.

Table 8—*Percentage of Total Sleep Time in Different Sleep Positions

Positions	SDB Cases (n = 13)	Non-SDB Cases (n = 74)	p Value
Prone			
Night 1	3.78 (8.8)	1.06 (3.33)	NS
Night 2	1.81 (4.7)	1.07 (3.04)	NS
Right lateral			
Night 1	22.58 (28.23)	15.82 (18.51)	NS
Night 2	19.96 (23.88)	17.81 (20.59)	NS
Left lateral			
Night 1	16.76 (15.49)	23.63 (25.05)	NS
Night 2	21.74 (21.18)	26.35 (25.27)	NS
Supine			
Night 1	56.88 (30.32)	59.5 (26.06)	NS
Night 2	56.49 (22.79)	54.77 (25.12)	NS

*Data are presented as mean (SD); see Table 1 for expansion of abbreviation.

second night. As a result, they were more willing to turn to positions where they were most comfortable, for example, the lateral position that would reduce the degree of airway resistance with fewer apneas and hypopneas. However, the sleep position data did not support our initial hypothesis. There were no significant differences between the various positions over the 2 nights. Instead, this improvement of the severity of respiratory indexes may be most likely explained by the possible increase in the percentage of SWS among the SDB subjects on the second night. It has been reported by Goh et al[21] that SWS is actually protective against childhood SDB, and the underlying mechanism may be partly related to an increase in genioglossus muscle tone activity.[22] Nevertheless, the childhood data were in sharp contrast to the adult data, which demonstrated an exacerbation of SDB when sleep quality improved with both REM and SWS rebound on the second night.[12] The discrepancy between the results obtained in childhood and adult SDB certainly require further study, and may suggest that childhood SDB is more sensitive to SWS rebound rather than REM sleep rebound. It may also be hypothesized that the relative attrition of SWS in adult may dampen any potential of the rebound effect of SWS on the respiratory activity. If the potential protective value of SWS on childhood SDB is confirmed, it could suggest the potential use and development of pharmacologic agent in enhancing SWS in childhood SDB.[23]

As discussed above, there is no universally accepted polysomnographic parameter for diagnosing SDB in children, and one of the most commonly used cutoffs is an OAI ≥ 1.[6–8,24] The alternative was to use various levels of AHI as the cutoff criteria.[6–8] The cutoff level of using AHI ≥ 5 had the best correlation with OAI ≥ 1 ($\kappa = 0.71$), while the correlation of OAI ≥ 1 and AHI ≥ 2 or 3 had low κ values of 0.22 and 0.31, respectively. In fact, if we used AHI ≥ 5, there was a similar finding of the improvement of AHI in the SDB group in association with a even more clear-cut and statistically significant SWS rebound but worsening of central respiratory disturbances in association of REM sleep rebound in the normal children. Nevertheless, as clearly suggested by the American Thoracic Society workshop summary, the diagnostic cutoff of OAI ≥ 1 only suggested a statistical significance but more studies will be needed to find out the level of the indexes that will predict a clinical significance.[7,8] In this study, we used the worst OAI of the 2 nights as a standard. An alternative approach could be to use the mean OAI from the 2 nights, but currently there are no evidences as to which approach would be best correlated with clinical outcome. Using the worst OAI of 2 nights as the criterion, the first-night study would correctly identify 84.6% of SDB cases. The cases missed by the single-night study belonged to the borderline range. The cost implication of our study findings is significant, as an additional night of polysomnography will mean extra manpower, resources, and lengthening of the waiting list.

There are certain limitations to our study, including our small sample size, the exclusion of two most severely affected cases for trial of continuous positive airway pressure on the second night, and the use of thermistor signal that may limit the detection of subtle airflow changes. However, as we were measuring the night-to-night variability of SDB by using the same measurement technique, we believed that this would not alter our result significantly. It has been suggested that FNE may last for > 1 night, and 3 consecutive nights might yield the most reliable representation of sleep, allowing for a FNE on night 1, rebound sleep on night 2, and usual sleep on night 3.[18,25] The rationale being that the second night could be affected by partial REM sleep deprivation that occurred in the first night.[26] However, a study[27] investigated the FNE phenomenon across multiple sleep laboratory sessions with polysomnographic data from three different periods of 4 consecutive nights suggested that the FNE was present only in the "very first night" of the first period. We assessed obesity as the cause of SDB in our cohort of children, whether night-to-night variability also occurs in other causes of SDB will need further study.

In summary, there were significant differences between 2 consecutive nights in the sleep architecture and respiratory indexes in our cohort of children with the demonstration of the FNE. A single-night study is able to identify 84.6% of cases with SDB with the remaining having borderline OAI. In the current climate of financial constraints and long

waiting times for sleep assessment, we proposed that a single-night sleep study was adequate and cost-effective in assessing for SDB in children.

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