Normal Polysomnographic Respiratory Values in Children and Adolescents*

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Study objectives: **Polysomnography is important in the evaluation of children and adolescents with sleep-disordered breathing. Adult criteria for obstructive sleep apnea have been shown to be inapplicable to children. Nevertheless, very little data are available regarding normal respiratory parameters during sleep in healthy children and adolescents. The purpose of the study was to characterize normal polysomnography values in healthy children and adolescents and to establish respiratory reference values for pediatric polysomnography.**

Design, setting and participants: **Seventy healthy, normal children and adolescents were studied. Age ranged from 1 to 15 years (mean ± SD, 8.02 ± 4.57 years). All children underwent overnight polysomnography including EEG, electromyography, electrooculography, ECG, pulse oximetry arterial oxygen saturation (Spo_2), chest wall and abdomen motion, oral and nasal airflow, and end-tidal Pco_2 ($Petco_2$).**

Results: **Three children (4%) had a mean of 0.37 obstructive apneas (OAs) per hour of sleep (1 to 5 OAs per child per study), with mean apnea duration of 10.3 ± 2.1 s. This was not accompanied with oxygen desaturation. Twenty-six children had one to seven central apneas (CAs) per child, resulting in a mean of 0.4 CAs per hour of sleep (median, 0.33; 97.5 percentile, 0.9). Eleven of the 58 events of CA in six children coincided with oxygen desaturation to a minimum of 88% (nadir apnea desaturation range, 88 to 93%). The mean Spo_2 was 97.2 ± 0.8% with Spo_2 nadir of 94.6 ± 2.2%. $Petco_2$ > 45 mm Hg occurred for 1.6 ± 3.8% of total sleep time (TST) in 21 of 70 children (30%), with a distribution of 1.3 ± 3.03% in the range of 46 to 47 mm Hg; < 0.7% were within the range of 48 to 50 mm Hg; and in 0.29 ± 0.24% of TST, $Petco_2$ values were > 50 mm Hg.**

Conclusions: **Based on these data, the recommended limits for normal values are as follows: OA index, 1; CA index, 0.9; oxygen desaturation, 89%; baseline saturation, 92%; and $Petco_2$ > 45 mm Hg for < 10% of TST.** ***(CHEST 2004; 125:872–878)***

Key words: central sleep apnea; children; end-tidal CO_2; hemoglobin saturation; hypopnea; obstructive sleep apnea; polysomnography; sleep

Abbreviations: AI = apnea index; CA = central apnea; OA = obstructive apnea; OSA = obstructive sleep apnea; $Petco_2$ = end-tidal Pco_2; REM = rapid eye movement; SDB = sleep-disordered breathing; Spo_2 = pulse oximetry arterial oxygen saturation; TST = total sleep time

Polysomnography is important in the evaluation of children and adolescents with sleep-disordered breathing (SDB). Pediatric polysomnography guidelines for diagnosing SDB in infants and children have been published.[1] However, compared to adults, very little data are available regarding normal values of respiratory parameters during sleep in healthy children and adolescents. Adult criteria for obstructive sleep apnea (OSA) were not applicable to children.[2] Physiologic differences between adults and children, as well as differences in the epidemiology, pathophysiology, and clinical manifestation of SDB, require that interpretation of polysomnography in children be based on age-specific normal values, thus recognizing their uniqueness and the influence on development.[2,3] At present, only one study[4] presented normal polysomnography values for children and adolescents. Hence, pediatric sleep laboratories frequently use their own unpublished experience, which may depend on ethnic, geographic, and other

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influences. The purpose of this study was to characterize normal polysomnography values in healthy children and adolescents in order to establish reference values for pediatric polysomnography.

Patients and Methods

Study Population

Seventy, normal, healthy, white children and adolescents ranging in age from 1 to 15 years were included in the study. They were recruited haphazardly from schools and kindergartens and from families of hospital employees. Only children who were assessed as being normal subjects without SDB by clinical history were included. Children with the following history or findings were excluded from the study: snoring, labored breathing or chest retractions during sleep, sleep apneas, craniofacial anomalies, obesity (body mass index ≥ 25), chronic illness or requiring chronic medication, history of adenoidectomy, tonsillectomy, or other airway surgery.

Polysomnography

All children underwent overnight polysomnography for at least 6 h in a quiet, dark room. No sedation or sleep deprivation was used to induce sleep. All children were accompanied by at least one parent throughout the night. During polysomnography, the following variables were continuously measured and recorded by a computerized polysomnograph (Somnostar α Sleep Laboratory; SensorMedics; Yorba Linda, CA). Chest wall and abdominal motions were measured by mercury strain gauges and respiratory inductive plethysmography. Heart rate was measured by ECG. End-tidal P_{CO_2} (P_{ETCO_2}) and nasal airflow were measured by capnography (Datex Normocap; Datex-Ohmeda Instrumentarium; Helsinki, Finland) sampling at a rate of 60 mL/min. The capnograph was calibrated before each study. Mouth and nasal airflow was measured by a thermistor positioned at the mouth and nares. Pulse oximetry arterial oxygen saturation (S_{pO_2}) was recorded using a Nellcor N-200 pulse oximeter (Nellcor; Pleasanton, CA) set to use 2- to 3-s averaging (mode 2, fast). The pulse waveform was recorded in all cases on a different channel to differentiate between true desaturation episodes and artifacts. Sleep stages were measured using four EEG electrodes, located on the scalp, two on each side of the parietal and occipital areas (C3/A2, C4/A1, O1/A2, O2/A1), and by electrooculography. Submental electromyography was measured by two electrodes located at the point of chin and belly of the digastric muscle on each side of the chin.

Children were also monitored and recorded on audio/videotape using an infrared video camera. Each child was continuously observed by a technician trained in pediatric polysomnography who also recorded sleep behavior and respiratory events. The following variables were quantitated and analyzed.

Respiratory Variables

Obstructive apnea (OA) was defined as cessation of airflow at the nose and the mouth, as measured by the thermistor and the capnograph, while the respiratory effort continues (movements of the rib cage and the abdomen) for at least two breaths. The number and duration of OAs of any length were quantitated. The minimum saturation and sleep stage of every apnea were recorded.

Central apnea (CA) was defined as the absence of airflow at both the nose and mouth as measured by the thermistor and the capnograph, associated with absence of movement of the chest and abdominal walls. The number and length of CAs ≥ 10 s were quantitated. CAs of any length that were associated with desaturation > 4% from the average S_{pO_2} in the preceding epochs or ≤ 92% were quantitated separately. CAs occurring immediately following a sigh were counted separately. The sleep stage of each apnea was recorded.

Mixed apnea was defined as an apnea with both central and obstructive components, in any order, with the central component lasting ≥ 3 s. The number and duration of mixed apneas were measured.

Hypopnea was defined as a ≥ 50% decrease in the amplitude of the nasal/oral airflow signal, as measured by the thermistor, often accompanied by hypoxemia or arousal.[5] Hypopnea was further characterized as obstructive if the reduction in airflow was associated with paradoxical chest and abdominal movement, or central if associated with an in-phase reduction in the amplitude of the chest and abdominal signals. Apnea index (AI) and apnea-hypopnea index were calculated by dividing the number of apneas or hypopneas by the hours of sleep.

All hemoglobin desaturations defined as decreases > 4% from baseline S_{pO_2} or any S_{pO_2} values ≤ 92% were quantified. For each child, the total duration of desaturation was expressed as percentage of total sleep time (TST) and the stage of sleep at which it appeared. Measurements associated with poor pulse tracings were excluded.

High P_{ETCO_2} values were classified into three groups: 46 to 47 mm Hg, 48 to 50 mm Hg, and > 50 mm Hg. For each subject, the total duration of time during which P_{ETCO_2} was within these ranges was recorded and expressed as percentage of TST. The highest P_{ETCO_2} was also recorded.

Sleep Architecture

Sleep was staged according to standard criteria[6] using EEG, electrooculography, and electromyography. Sleep onset was defined as two consecutive 30-s epochs of stage I or one epoch of stage II or rapid eye movement (REM) sleep.[6] Arousals were defined as EEG arousal according to accepted definitions.[7] TST was defined as all sleep incurred from sleep onset until morning awakening. Sleep efficiency was defined as duration of TST to monitoring time. Respiratory parameters were expressed as a percentage of the TST.

The data were scored by one of two pediatricians with specific training in sleep medicine and who were senior staff members of the sleep center (R.T., M.G.). The scorers were unaware that the child was a "normal" subject or that the polysomnography was a part of a study.

Statistical Analysis

Data for variables that showed normal Gaussian distribution are expressed as mean ± SD. For data that were not normally distributed median, range and 97.5% percentile are used. The data were analyzed using BMDP statistical software (BMDP; Saugus, MA) [1993, chief editor: W.J. Dixon, University of California Press]. Continuous variables were compared across groups using analysis of variance. Pearson correlations were calculated between continuous variables. Fisher exact test (two tailed) was applied to find association between discrete variables.

Results

Subjects age ranged from 1 to 15 years (mean ± SD, 7.9 ± 4.4 years). Thirty subjects (43%) were male. The age and sex distributions are shown in Figure 1.

Sleep Architecture

The average TST was 6.46 ± 1.24 h, with mean sleep efficiency of 90.8±6.5%. The distribution of sleep stages as percentage of TST was as follows: stage I, 4.1 ± 4.1%; stage II, 48.9 ± 9.7%; slow-wave sleep, 25.2 ± 9.1%; and REM sleep, 17.4 ± 5.7%. The AI was 5.29 ± 3.49.

Apneas

A total of 65 apneas (not including apneas following a sigh) were observed in 29 of 70 children (41%). Fifty-eight of 65 apneas (89%) were CAs, with mean apnea length of 11.8 ± 3.0 s (range, 6 to 20 s). These apneas were observed in 26 children (37%; 10 male) with one to seven apneas per child and a mean CA index of 0.4 (median, 0.33; 97.5 percentile, 0.9). Six of 70 studied children had a total of 11 CA events (1 to 2 events per child) accompanied by oxygen desaturation to a minimum of 88% (range, 93 to 88%). Four of those 11 CAs that were accompanied by desaturation were short apneas lasting < 10 s (range, 6 to 9 s).

Children with CAs spent 18.1 ± 6.8% of TST in REM sleep. This was not different from the time duration spent in REM sleep by all the other 44 children in the study (16.9 ± 4.1%; p = 0.43). However, 41.1% of all the CAs occurred during REM sleep. Thus, CAs were more frequent during REM sleep (p < 0.01; Fig 2).

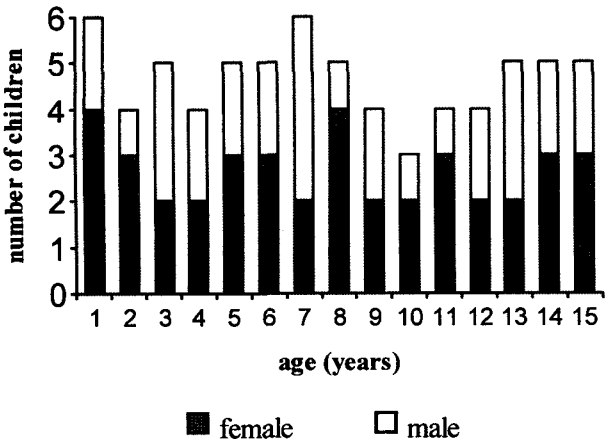

FIGURE 1. Distribution of the study population by age and gender.

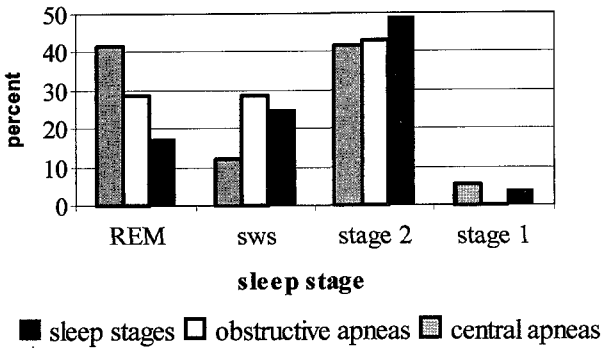

FIGURE 2. Distribution of CAs (as percentage of total CA) and OAs (as percentage of total OA) in relation to distribution of sleep stages. SWS = slow-wave sleep.

Seven of the 65 apneas (10%) were OAs that presented in 3 of 70 children (4%). Five of the OAs occurred in one subject, a 6-year-old girl. The average length was 10.3 ± 2.1 s (range, 7 to 13 s). None were associated with oxyhemoglobin desaturation.

The mean OA index for the whole study population was 0.016 (median, 0; 97.5 percentile, 0.1). When calculated only for children who had OAs, the mean index was 0.37 (median, 0.18; 97.5 percentile, 0.7). The distribution of the OAs did not show an association to a specific sleep stage. No mixed apneas were observed in any of the studied children.

Thirty-one children (44%) had 88 apneas preceded by a sigh (15 boys), with an average duration of 13.2 ± 2.8 s (range, 10 to 25 s). None were associated with desaturation. No correlation was found between the number, the duration, and the index of the apneas of any kind and age or gender.

There were six hypopnea events. Three children had one event of central hypopnea each, and all appeared during REM sleep stage. The mean duration was 21.0 ± 11.2 s (range, 14 to 34 s). Three other hypopneas were obstructive, all three observed in the same child. They lasted 13 to 21 s (mean, 17.6 s). None of these six events were accompanied by desaturation.

The mean hemoglobin saturation was 97.2 ± 0.85%, and the mean Spo_2 nadir was 94.6 ± 2.2%. Seven children exhibited desaturation events. Six of them had desaturations only in association with CAs (minimum, 88%), and one child had desaturation to a minimum of 89% (range, 92 to 89%) during 11% of TST (during 10%, the Spo_2 was 92%), but did not exhibit any apneas.

Twenty-one of 70 children in the study had $PETCO_2$ levels > 45 mm Hg, for a total of 1.6 ± 3.8% of TST (Table 1). $PETCO_2$ levels did not correlate with age or sex.

Table 1—*Percentage of Time Spent in P_{ETCO_2} Levels > 45 mm Hg of TST in Children With High P_{ETCO_2} Levels***

Variables	P_{ETCO_2}, mm Hg			
	46–47	48–50	> 50	Overall
% of TST	1.3 ± 3.0	0.67 ± 0.69	0.29 ± 0.24	1.6 ± 3.8
Range	0.11–13.96	0.14–2.29	0.11–0.57	0.11–15.85
No. of children/total (%)	21/70 (30)	10/70 (14)	3/70 (4)	21

*Data are presented ± SD unless otherwise indicated. A total of 21 children had P_{ETCO_2} levels > 45 mm Hg, 10 children also had levels in the range of 48–50 mm Hg, and 3 of the 10 children had values > 50 mm Hg.

Discussion

Although polysomnography is a common procedure in children, reference values are inadequate. Only one study[4] designed to establish normal values in children has been reported. A comparison of the present study to the previous report is presented in Table 2. The present study provides additional information on normal polysomnographic values in children.

Our population included only white children. It has been recently suggested that certain ethnic populations such as African Americans may be at higher risk for OSA compared to whites.[8] It is not clear, however, whether different normal values should be used in this ethnic group. Specific research is required to answer this question.

Table 2—*Comparison of Results*

Variables	Current Study	Marcus et al[4]
CA index		
Mean	0.4	
Median*	0.33	
97.5 percentile*	0.9	
CA duration, s		
Mean ± SD	11.8 ± 3.0	
Range	6–20	10–18
OA index		
Mean	0.37	
Median*	0.18	
97.5 percentile*	0.7	
Mean ± SD		0.56 ± 0.9†
OA duration, s		
Mean ± SD	10.3 ± 2.1	
Mean		≤ 10
Range	7–13	
Spo_2 nadir, %		
Mean ± SD	94.6 ± 2.2	96 ± 2†
P_{ETCO_2} levels > 45 mm Hg, % of TST ± SD	1.6 ± 3.8	6.9 ± 19.1†
TST, h		
Mean ± SD	6.5 ± 1.2	6.0 ± 1.6
Sleep efficiency, %		
Mean ± SD	90.8 ± 6.5	

*For parameters with nonnormal Gaussian distribution.
†No information is available regarding the distribution pattern of values.

Snoring children were excluded from the study population. Most snoring children are "habitual snorers," having "primary snoring" and at present are considered normal subjects. However, polysomnography may be required in order to reliably differentiate between primary snoring and OSA syndrome. Hence, including these subjects would have resulted in including children with OSA syndrome in a normal population. Children who usually do not snore but snore rarely during upper respiratory infections were not excluded. Children suspected of having any problem that might have affected normal values were not included. Hence, the study cohort more accurately represents the normal pediatric population.

This study shows that OSAs are uncommon events in the normal pediatric population. Only 3 of 70 children (4%) had a total of seven OAs; five of these seven OAs occurred in one child, and 2 children had only one episode each. None of the OAs were associated with desaturation or arousal. Marcus and colleagues[4] reported a somewhat higher rate of 9 of 50 subjects (18%) in a comparable group. They reported similar results in one child who had 12 OAs per night while all the other children with OAs had only 1 to 2 OAs per night. It is possible that the only child with multiple OAs (1.4%) in our population as well as the single subject in the study by Marcus et al[4] (2%) represent true cases of mild OSA who filtered into the study population despite the exclusion criteria. The rate of 2% is well in accordance with the frequency of OSA syndrome in children.[9] Also, the length of OA in our study was similar to the previous report (7 to 13 s vs ≤ 10 s, respectively). The OA index calculated for the entire study population (97.5 percentile, 0.1) was similar to the value previously reported (0.1 ± 0.5).[4] The ranges were also similar: 0 to 0.3 except for the one subject in each study with multiple OAs per night. Defining reference values for AI by the mean + 2 SD or by the 97.5 percentile from the entire study populations in both studies is irrelevant because the distribution of OA significantly differed from normal distribution. Moreover, because the vast majority of children

in both studies (67 of 70 subjects and 41 of 50 subjects, respectively) did not exhibit any OA, the definition of normal AI values based on the mean and SD or the 97.5 percentile for the entire study cohort may introduce a significant bias by lowering the reference values significantly and, therefore, misleading the diagnoses of normal children as abnormal. The mean OA index calculated only for children with OA was 0.37 (97.5 percentile, 0.7) in our subjects compared to 0.56 ± 0.9 in the study by Marcus et al[4] (calculated from their data). Acebo et al[10] reported mean total AIs of 1 and 0.9 in 23 boys and 22 girls, respectively. They showed that normal older boys and girls (age range, 13.3 ± 2.1 years) could have up to four apneas per hour of sleep. They provided, however, a combined value for obstructive and central apneas together. Hence, a slightly higher value is not surprising. Because the present normal value for AI (< 0.8) is very close to the value previously recommended (≤ 1), we recommend that for practical reasons the normal values for OA index be ≤ 1. These findings differ significantly from adult normal values[2,11,12] that define normal reference values as five or more complete OA events per hour of sleep, and emphasize the importance of using pediatric reference values. In patients with OSA, OAs were found to be associated with REM sleep. Such an association was not found in our subjects. Although this may be explained by the low rate of OAs that precludes association of OAs to any sleep stage, the explanation may be that this tendency was observed in children with OSA syndrome, while the present population comprised of normal children in whom the occurrence of OA is rare.

CAs lasting > 10 s were common in the present study at all ages and consisted 82% of all apneas. Only 11 of 58 CAs (19%) were accompanied by mild desaturation.

The tendency of CAs to occur during REM sleep has been shown in adults and in patients with chronic lung disease. CAs occurring during REM sleep are also more frequently associated with desaturation. The present results show that this is also the situation in normal children. It may be explained by the decrease in functional residual capacity during REM sleep.[13]

Previous observations have shown that CAs of various lengths were not uncommon in children[14,15]; however, these reports involved smaller groups (22 adolescents and 9 adolescents, respectively), and references values could not be defined. In the study by Marcus et al[4] of normal 1- to 17-year-old children, 30% had CAs that lasted 10 to 18 s and only one CA was accompanied by mild desaturation. Normal values for references were not provided. Our data allow to recommend the upper limit for the rate of CA in children > 1 year old to be less than one CA per hour of sleep (97.5 percentile), and the longest duration 20 s (means + 2 SD). In view of the common occurrence of CA, the most important feature is whether the CA is associated with desaturation. We suggest that any CA accompanied with hemoglobin desaturation to < 89% will be considered as abnormal. In addition, the American Thoracic Society guidelines based on other reports defined that a drop > 4% in Spo_2 should also be considered a desaturation event.[1]

It is interesting that desaturations were associated with CAs but not with OAs. However, each type of apnea should be looked at separately. Only six children had a total of 11 CA events accompanied by desaturation. The desaturation was relatively mild to a nadir of 93 to 88%. It is possible that some of these events were preceded by mild central hypoventilation, and the CA that followed resulted with desaturation. Only three subjects had a total of seven OAs (two children experienced one OA each). Hence, this may be considered a relatively rare and mild event in normal children, and the risk for accompanying desaturation cannot, therefore, be concluded. Marcus and colleagues[4] also did not show an association of OA and desaturation. It should be noted that the only other study that was specifically designed to establish normal values (Marcus et al[4]) did not include hypopneas. This study also did not use EEG to define sleep.

Because episodes of complete airway obstruction are relatively uncommon in children with sleep-related upper airway obstruction,[1,2,15-17] and OSA syndrome in children may manifest mainly as hypopneas and continuous hypoventilation with only partial cessation of airflow, it might be difficult to diagnose OSA in some children. Hence, monitoring carbon dioxide levels becomes of major importance in the evaluation of gas exchange abnormalities during sleep. Brouillette and colleagues[18] suggested that the maximal pediatric normal value for $PETCO_2$ during sleep is 45 mm Hg, and that higher values should be considered abnormal. However, Marcus et al[4] in their normal healthy pediatric population found that the normal sleep duration with $PETCO_2$ > 45 mm Hg was 6.9 ± 19.1% (range, 0 to 90.5%) of TST and > 50 mm Hg 0.5 ± 4% of TST (range, 0 to 25%). Based on these results, they recommended that a peak $PETCO_2$ > 53 mm Hg or $PETCO_2$ > 45 mm Hg for > 60% of the TST should be considered abnormal. Our results disagree with the latter recommendations. $PETCO_2$ in our study was > 45 mm Hg for only 1.6 ± 3.8% of TST and > 50 mm Hg for 0.29 ± 0.24% of TST. In another study by Marcus and associates,[19] hypercapneic arousal response was investigated in seven healthy children aged 4.4 ± 1.1

Table 3—*Recommendations for Normal Polysomnographic Respiratory Values*

	Current Study	Marcus et al[4]
CA	Lasting> 20s Any length when associated with Spo_2 < 89% or Spo_2 drop > 4%†	NR* Any length when associated with Spo_2 < 90%
CA index	≤ 1.0	NR*
OA index	≤ 1.0	≤ 1.0
Spo_2 nadir, %	> 92	> 92
$PETCO_2$ levels > 45 mm Hg, % of TST	< 10	< 60

*No recommendations (NR) were given. Nevertheless, extrapolation from their data shows very similar results for CA (lasting > 18 s and any apnea associated with Spo_2 < 90%, and CA index < 1.0).

†Spo_2 drop > 14% is recommended by other sources and does not result from the present study (see text).

years. The challenge was maintained until arousal occurred or for a maximum of 3 min. They found that all children aroused in response to hypercapnia when $PETCO_2$ values reached 49 to 52 mm Hg. Hence, if these $PETCO_2$ values cause arousal it is unlikely that normal children spend up to 60% of TST with these values and a significant time with $PETCO_2$ > 50 mm Hg. It should also be noted that $PETCO_2$ is lower than arterial PCO_2 by 2 to 4 mm Hg. Data from Gozal et al[20] support our results. They studied six normal healthy children aged 7 to 14 years and found that $PETCO_2$ stayed > 45 mm Hg for only 2.0 ± 0.3% (range, 0 to 5.1%) of TST with a peak $PETCO_2$ of 46.1 ± 2.2 mm Hg (range, 42 to 51 mm Hg). In another review based on the published literature and his own experience, Gozal[21] suggests that the $PETCO_2$ > 45 mm Hg up to 20% of TST is the normal range employed by most pediatric sleep laboratories. We suggest that $PETCO_2$ > 45 mm Hg for > 10% (mean + 2 SD) of the TST or any $PETCO_2$ > 50 mm Hg be considered abnormal.

Capnography tracings are subject to technical difficulties that result mainly from displacement and blocked sampling catheters and from interference with the nasal pressure prongs. This may result in low CO_2 tracings and loss of the plateau phase during parts of the polysomnography, a condition that is prerequisite for estimation of end-tidal CO_2. Great caution should, therefore, be used when analyzing $PETCO_2$ values and reporting normal values by the percentage of time the $PETCO_2$ is above a certain value.

The present data on normal values for nocturnal hemoglobin saturation (97.2 ± 0.8%) is well in accordance with previous reports. An older study[15] found a minimum value of 96.1 ± 0.6% and average of 97.1 ± 0.3% (n = 9). Acebo and colleagues[10] reported Spo_2 nadir values of 93.5 ± 2.7% (adolescent boys) and 94.3 ± 1.7% (adolescent girls), and Marcus et al[4] published a nadir value of 96 ± 2% (n = 50) for a similar population as the present study. In the study by Gries and Brooks[22] of 180 children (age range, 1 to 10 years) and 46 older subjects (age range, 10 to 20 years), the nadir Spo_2 values were 90.1 ± 3.6% and 90.4 ± 2%, respectively.[22] These values are lower compared to those found in our investigation and in some of the studies cited above. The subjects in their study were not recruited from the general population, but were referred to the laboratory because of suspected sleep disorders and were considered normal subjects based on normal polysomnography results (respiratory disturbance index ≤ 5). Hence, they may not represent a completely normal population. A recent study[23] of third-grade children performed at home showed that almost all children spent most of the night at Spo_2 ≥ 98% and intermittent falls of ≥ 4% were frequent, but Spo_2 levels ≤ 90% were rare. This study was done on 90 children, but only 58 of them did not have respiratory complaints. Snoring children were not excluded and children were not continuously observed by a technician. We, therefore, recommend Spo_2 of 92% as the lower limit in normal boys and in girls.

It should be noted that the present study was performed at sea level. We recommend that this variable be adjusted according to altitude above sea level especially in areas of high altitude.

This study may contain a theoretical limitation in that the TST was relatively short. This may explain the relative paucity of REM sleep, which is a little below normal range. However, all sleep studies began at the child's usual bedtime. All studies contained at least two REM periods. This criteria is in accordance with the recommendations.[1]

The relation of normal values to clinical significance in pediatric SDB is not always clear. Indications for surgery are not universal. Some clinician may require at least five OAs per hour before referring a child to surgery.[24,25] Others would refer to surgery any child with abnormal polysomnography findings. The issue of the short-term and long-term results of SDB in children as well as the reversibility of damages and long-term developmental, behav-

ioral, and neurocognitive sequelae has been a very debated topic during recent years. Recent data suggest that mild SDB and even primary snoring are risk factors for neurocognitive deficits.[26–28] A significant amount of research is underway to elucidate this issue. The present study provides reference values for pediatric polysomnography.

In conclusion, this study provides information on normal reference values for respiratory variables during sleep in children and adolescents (Table 3). Most recommendations are in agreement with previously published reports and thus add to the reliability of the already published normal values. This study suggests new upper limits for normal P_{ETCO_2} levels.

References

1 American Thoracic Society. Standards and indications for cardiopulmonary sleep studies in children. Am J Respir Crit Care Med 1996; 153:866–878

2 Rosen CL, D'Andrea L, Haddad GG. Adult criteria for observation sleep apnea do not identify children with serious obstruction. Am Rev Respir Dis 1992; 146:1231–1234

3 Carrol JL, McColley SA, Marcus CL, et al. Reported symptoms of childhood obstructive sleep apnea syndrome (OSA) vs primary snoring [abstract]. Am Rev Respir Dis 1992; 145: A177

4 Marcus CL, Omlin KJ, Basinski DJ, et al. Normal polysomnographic values for children and adolescents. Am Rev Respir Dis 1992; 146:1235–1239

5 Moser NJ, Phillips BR, Berry DTR, et al. What is hypopnea, anyway? Chest 1994; 105:426–428

6 Rechtschffen A, Kales A, eds. A manual of standardized terminology, techniques and scoring for sleep stages of human subjects. Washington, DC: National Institute of Health, 1968; publication No. 204

7 Bonnet M, Careley D, Carskadon M, et al. EEG arousals: scoring rules and examples; a preliminary report from the Sleep Disorders Atlas Task Force of the American Sleep Disorders Association. Sleep 1992; 15:173–184

8 Meetze K, Gillespie MB, Lee FS. Obstructive sleep apnea: a comparison of black and white subjects. Laryngoscope 2002; 112:1271–1274

9 Ali NJ, Pitson DJ, Stradling JR. Snoring, sleep disturbance and behavior in 4–5 year olds. Arch Dis Child 1993; 68:360–366

10 Acebo C, Millman RP, Rosenberg C, et al. Sleep, breathing, and cephalometrics in older children and young adults: part I; normative values. Chest 1996; 109:664–672

11 Berry DTR, Webb WB, Block AJ. Sleep apnea syndrome: a critical review of the apnea index as a diagnostic criterion. Chest 1984; 86:529–531

12 Guilleminault C, Van den Hoed J, Mitler M. Clinical overview of the sleep apnea syndromes. In: Guilleminault C, Dement W, eds. Sleep apnea syndrome. New York, NY: Alan R. Liss, 1978; 1–2

13 Muller NL, Francis PW, Gurwitz D, et al. Mechanism of hemoglobin desaturation during rapid eye movement sleep in normal subjects and in patients with cystic fibrosis. Am Rev Respir Dis 1980; 121:463–469

14 Carskadon MA, Harvey K, Dement WC, et al. Respiration during sleep in children. West J Med 1978; 128:477–481

15 Tabachnick E, Muller NL, Bryan AC, et al. Changes in ventilation and chest wall mechanics during sleep in normal adolescents. J Appl Physiol 1981; 51:557–564

16 Brouillette RT, Fernbach SK, Hunt CE. Obstructive sleep apnea in infants and children. J Pediatr 1982; 100:31–40

17 Marcus CL, McColley SA, Carroll JL, et al. Upper airway collapsibility in children with obstructive sleep apnea syndrome. J Appl Physiol 1994; 77:918–924

18 Brouillette RT, Weese-Mayer DE, Hunt CE. Disorders of breathing during sleep in the pediatric population. Semin Respir Med 1998; 9:594–606

19 Marcus CL, Bautista DB, Amihyia A, et al. Hypercapneic arousal responses in children with congenital central hypoventilation syndrome. Pediatrics 1991; 88:993–998

20 Gozal D, Arens R, Omlin KJ, et al. Ventilatory response to consecutive short hypercapnic challenge in children with obstructive sleep apnea. J Appl Physiol 1995; 79:1608–1614

21 Gozal D. Obstructive sleep apnea in children. Minerva Pediatr 2000; 52:629–639

22 Gries RE, Brooks LJ. Normal oxyhemoglobin saturation during sleep. Chest 1996; 110:1489–1492

23 Urschitz MS, Wolff J, von Einem W, et al. Reference values for nocturnal home pulse oximetry during sleep in primary school children. Chest 2003; 123:96–101

24 Jain A, Sahni JK. Polysomnographic studies in children undergoing adenoidectomy and/or tonsillectomy. J Laryngol Otol 2002; 116:711–715

25 Suen JS, Arnold JE, Brooks LJ. Adenotonsillectomy for treatment of obstructive sleep apnea in children. Arch Otolaryngol Head Neck Surg 1995; 121:525–530

26 O'Brien LM, Mervis CB, Holbrook CR, et al. Is primary snoring in children really benign [abstract]? Am J Respir Crit Care Med 2003; 167:A18

27 Urschitz MS, Guenther A, Eggebrecht E, et al. Snoring, intermittent hypoxia and academic performance in primary school children. Am J Respir Crit Care Med 2003; 168:464–468

28 Schechter MS. American Thoracic Society, Section on Pediatric Pulmonology, Subcommittee on Obstructive Sleep Apnea Syndrome. Technical report: diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics 2002; 109:e69