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CHEST Postgraduate Education Corner

CONTEMPORARY REVIEWS IN SLEEP MEDICINE

Childhood Obstructive Sleep-Disordered Breathing*

A Clinical Update and Discussion of Technological Innovations and Challenges

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Childhood sleep-disordered breathing (SDB) has been known to be associated with health and cognitive impacts for more than a century, and yet our understanding of this disorder is in its infancy. Neuropsychological consequences in children with snoring or subtle breathing disturbances not meeting the traditional definition of sleep apnea suggest that "benign, or primary snoring" may be clinically significant, and that the true prevalence of SDB might be underestimated. There is no standard definition of SDB in children. The polysomnographic technology used in many sleep laboratories may be inadequate to diagnose serious but subtle forms of clinically important airflow limitation. In the last several years, advances in digital technology as well as new observational studies of respiratory and arousal patterns in large populations of healthy children have led to alternative views of what constitutes sleep-related breathing and arousal abnormalities that may refine our diagnostic criteria. This article reviews our knowledge of childhood SDB, highlights recent advances in technology, and discusses diagnostic and treatment strategies that will advance the management of children with pediatric SDB.

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Key words: biomedical engineering; noninvasive techniques; pediatrics; sleep apnea

Abbreviations: AHI = apnea-hypopnea index; CAP = cyclic alternating pattern; CPAP = continuous positive airway pressure; $Etco_2$ = end-tidal carbon dioxide; ICSD-2 = International Classification of Sleep Disorders 2nd edition; OSA = obstructive sleep apnea; PAT = peripheral arterial tonometry; PTT = pulse transit time; RERA = respiratory effort-related arousal; RIP = respiratory inductive plethysmography; SDB = sleep-disordered breathing; T&A = adenotonsillectomy; UARS = upper airway resistance syndrome

Childhood sleep-disordered breathing (SDB) has been known to be associated with health and cognitive impacts for more than a century,[1–3] and yet our understanding of this disorder is in its infancy. Children with SDB have behavior problems,[4] intelligence quotient deficits,[5–7] deficits of executive function,[5,8,9] school performance problems,[10] a high prevalence of abnormal neuropsychological diagnoses,[11] poor quality of life,[12,13] impaired growth,[14,15] cardiovascular insults,[16,17] and a 2.6-fold increase in health-care utilization.[18] However, due to a lack of standard diagnostic or therapeutic strategies,[19] and changes in a child's anatomy and physiology from infancy to adolescence, the true prevalence and the long-term social impact of this disorder are not understood.

With advances over the past few years in the recognition of subtle forms of sleep disruption affecting health,[20–23] guidelines for the treatment of pediatric SDB are needed. In the last several years,

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studies[24,25] of normal respiratory and arousal patterns in nonsnoring children have refined the view of what might be considered abnormal sleep-related breathing events. Childhood SDB has been traditionally defined with adult criteria, summing up the total number of upper airway obstructions per hour as the apnea index,[26] or including partial obstructions as the apnea-hypopnea index (AHI). Studies[4,27,28] of this accepted definition of obstructive sleep apnea (OSA) performed in school-aged children suggest a prevalence of 2 to 3%. However, neuropsychological consequences in children with snoring or subtle breathing disturbances not meeting the traditional definition of OSA[29–32] suggest that this prevalence is underestimated. Habitual snoring, which is a result of partial airway obstruction, is noted to exist in school-aged children with a prevalence ranging between 12% and 20%.[33–36] Therefore, the common assumption that "benign, or primary snoring" may not need therapeutic attention[37] is under debate.[38] Few large population studies have determined the prevalence of SDB in infants or toddlers, who have risk factors that differ from those of school-aged children and adolescents due to different craniofacial structure, fat deposition, hormonal influences, environmental factors, and sleep architecture.

Recommended Definition of Pediatric SDB

The International Classification of Sleep Disorders, 2nd edition (ICSD-2),[39] improves the diagnostic criteria for childhood SDB compared to traditional definitions based on adult studies. However, specific thresholds for respiratory-related events that define disease or required treatment are not standardized; therefore, sleep laboratories generally define their own thresholds, often using or modifying standard adult sleep-scoring techniques[40] or suggested pediatric criteria from the American Thoracic Society.[41]

OSA is an absence or reduction in airflow in the upper airway despite ongoing respiratory effort, frequently in combination with paradoxical breathing efforts and/or snoring.[37] The respiratory rates in children vary significantly with age (ranging from nearly 60 breaths/min in a premature infant to a rate of 12 breaths/min in the adolescent). Thus, defining an apnea in terms of time in seconds as is done in adults would likely lead to an underestimated respiratory disturbance index; therefore, the ICSD-2 defines apnea as a cessation of airflow over two or more attempted respiratory cycles.[39] The definition of hypopnea is in greater need of standardization.[42] A hypopnea is defined as a reduction (either qualitative or quantitative depending on the laboratory) in airflow over two or more respiratory cycles, accompanied by a 3% (or 4%) fall in oxygen saturation, and/or terminated by an arousal. The application of these criteria, however, varies significantly between pediatric sleep centers,[42] and some centers require the presence of both an arousal and oxygen desaturation to define a hypopnea. Moreover, the definition of apnea or hypopnea assumes discreet events. Children are known to have prolonged periods of partial airflow obstruction during sleep, often without arousal or oxygen saturation abnormalities, but frequently with elevations of carbon dioxide[43] (sometimes termed *obstructive hypoventilation*) or with increased work of breathing as measured by an esophageal pressure manometer that Guilleminault et al[44] have described as "upper airway resistance syndrome" (UARS) in adults and children.[45] Partial upper airway obstruction that results in UARS, however, is underestimated when using only the AHI to diagnose SDB.[43,45] The consideration of additional measures of ventilation would likely enhance our ability to identify children with SDB. Intermittent hypoxemia, even brief episodes of mild decreases in saturation from the baseline, is hypothesized to be a possible cause of neuropsychological dysfunction,[29,46–48] and yet the time spent with intermittent oxygen desaturation is not yet included in most definitions of SDB. Additionally, carbon dioxide levels are commonly measured in pediatric sleep laboratories, and the time spent with hypercapnia during sleep may be a very important marker of partial obstruction or abnormal respiratory compensation.[24]

The arousal index (*ie*, the number of arousal events occurring per hour of sleep) is one measure of sleep fragmentation. The definition of an arousal originated from EEG findings that were easily detected on older polysomnography equipment, and are defined as an abrupt shift in EEG frequency, which may include θ, α, or frequencies of > 16 Hz, but not spindles, for ≥ 3 s in duration.[49] Frequent arousals have been linked to alterations of attention and vigilance.[25,50] Advances in digital technology have led to research using spectral analysis of the EEG[51–53] or cardiovascular changes[23,54] during arousal events, which have afforded alternative methods of detecting important respiratory-related events during SDB, including "subcortical" arousals not necessarily detected on the epoch-by-epoch visually evaluated EEG.[54] Respiratory effort-related arousals (RERAs) are specific to SDB[21,22] and may be important to consider in the definitions of childhood SDB. The morbidity from each separate respiratory parameter (*ie*, AHI, intermittent hypoxemia, hypercapnia, or sleep fragmentation from arousals), however, is unknown. New technologies designed to improve the detection of these parameters and future studies to understand their significance as they relate to SDB are

needed before these tools are incorporated into standard diagnostic equipment.

What Is Normal?

In the last several years, large observational studies[24,25,55] of healthy children have increased our knowledge of normal respiratory parameters during sleep. This information adds to our knowledge of respiratory or arousal abnormalities noted in children with subtle SDB, such as those occurring without discreet gas exchange abnormalities, and may contribute to recommendations for treatment. An AHI of greater than one event per hour is out of the normal range in children,[39,55,56] but, as discussed in the previous section, the AHI should also be used in context with other respiratory or arousal parameters since the clinical significance of a mildly abnormal AHI is unknown. Hypercapnia time, measured with end-tidal or transcutaneous CO_2 levels of >50 mm Hg in healthy children, has been reported to be <10 to 20% of total sleep time in healthy children depending on the device used for measuring CO_2[24,25,56] and the strictness of the exclusion criteria (excluding children who snored associated with lower expected CO_2 values as in the study by Uliel et al[24]). Oxygen saturation in nonsnoring healthy children, measured by pulse oximetry, rarely drops to $<95\%$ from obstructive events in children,[24] although occasionally drops into the 89% range can occur normally with central apnea.[24] In another study[57] that included 180 children aged 1 to 10 years (including snoring children or those with an AHI of <5), the saturation nadir was 90.1%, with $>90\%$ of time spent at a saturation of $>95.1\%$. However, the amount of time with intermittent desaturations between 90% and 95% might be clinically significant in these children.[25] The expected arousal index (*ie*, the number of arousals per hour) in children has been not been standardized; however, a recent population study[25] of preschool children aged 3 to 7 years suggests that the average arousal index over total sleep time was less than eight arousals per hour; and less than one respiratory-related arousal (associated with apnea, hypopnea, or snore) per hour was noted. Arousal indexes in healthy children in the sleep laboratory tend to be <14.[25,58] Respiratory-related arousals are not recorded as standard diagnostic criteria in many clinical laboratories, but this value may improve the accuracy of the arousal index as a characteristic of SDB.

Pathophysiology

Subjects with obstructive airflow limitation have collapsible upper airways[59,60] or resistance along the upper airway.[61] Gold et al[62] have demonstrated that healthy children have an ability to oppose airway collapse due to the preservation of upper airway neuromotor responses during sleep, but those dynamic upper airway responses are diminished in children with OSA.

Bao and Guilleminault[45] and Guilleminault et al[63] have speculated that alterations of the neurons and musculature involved in pharyngeal tone during inspiration are the cause of airway collapse; this is supported by the results of pathologic investigations[64] of pharyngeal biopsy specimens from OSA patients demonstrating fascicular atrophy. Brain-imaging studies[65,66] of adults with sleep apnea have demonstrated morphologic changes in gray matter, including areas such as the cerebellum, which is important in inspiratory initiation.[67] A study[5] published recently has demonstrated alterations of resting neuronal metabolites of the hippocampus and frontal cortex in children with sleep apnea, also in association with neuropsychological deficits, indicating a possible neuronal injury associated with sleep apnea. The improvement of airway responses after the treatment of childhood OSA[68] indirectly supports the concept that neuronal alterations were caused by OSA, rather than predating the development of airway collapse. If these concepts are validated, the early recognition and treatment of childhood OSA would protect neuronal function.

Nasal obstruction is an often overlooked cause of upper airway resistance.[61,69] Nasal obstruction may be especially important in infants with "obligate nose breathing,"[70] where life-threatening breathing obstruction can occur, as seen with choanal stenosis at birth. Nasal resistance can lead to airflow limitation without affecting the collapsibility of the airway.[71] Airflow limitation impacted by nasal obstruction has been implicated in symptomatic cases of UARS.[72]

Symptoms of SDB in Children

Children with airflow limitation often snore, but may demonstrate other symptoms such as labored breathing efforts (*ie*, paradoxical efforts between the chest and abdomen), gasping, gagging, choking, noisy breathing, witnessed apnea, or excessive sweating.[8,19] Children with severe nasal obstruction may open their mouths and hyperextend their necks to breath, minimizing snoring noises. Infants with severe OSA may not snore but frequently have stridor, especially if the cause of the airway obstruction is fixed at the level of the epiglottis or vocal cords due to laryngomalacia or vocal cord paralysis, where daytime symptoms may be worse. Airway dynamics are different in infants compared to those in children

and adults,[73] which may in part be due to the anatomy of the infant airway where the epiglottis closely approximates the soft-palate tissues, thus reducing the size of the oropharynx[70] where snoring occurs.

Insomnia has been reported to be a symptom of SDB,[72] and somatic complaints are now being recognized[74] among other daytime symptoms such as sleepiness. Younger children with sleepiness may present with irritability or hyperactivity, and are often seen in sleep clinics with a diagnosis of attention deficit hyperactivity disorder.[11] The clinical presentation of children with SDB includes adenotonsillar hypertrophy,[37] nasal obstruction,[70] allergic symptoms, and craniofacial abnormalities causing reduced airway diameter or patency.[75] Other symptoms of sleep walking, enuresis, and parasomnias have also been seen in association with childhood SDB.[76]

Advances in Polysomnographic Diagnostic Technology

Measuring Airflow

The measurement of airflow is part of the diagnostic criteria for apneas and hypopneas, as well as for respiratory-related arousals. Many new devices designed for airflow measurement have become available for use during polysomnography. However, the clinical usefulness of more sensitive measures of airflow limitation remains to be determined especially in regard to correlation with daytime symptoms or treatment outcomes. Inspiratory flow limitation during sleep is defined by a decreasing (more negative) intrathoracic pressure without a corresponding increase in airway flow rate.[77] Most sensors designed to measure airflow actually measure the presence of airflow, not the quantitative measurement or volume of airflow. A pneumotachometer provides a quantitative measurement and is the "gold standard" for the measurement of airflow.[77] Until recently, the use of a pneumotachometer was precluded during sleep due to the excessive weight of the devices.

A common commercially available airflow measuring device is the thermistor, which is standard equipment in many sleep laboratories, but is notable for its limitations. A thermistor provides a qualitative signal that detects fluctuations in temperature with respiration, and therefore, does not actually detect flow and actually does not correlate well with decreases in airflow.[78–80] Thus, the thermistor lacks the reliability to detect changes in airflow that are necessary to meet the criteria for hypopneas, RERAs, and apneas. However, when combined with other airflow-measuring devices, this device is valuable, especially with its ability to detect oral airflow, which is important in mouth breathers.

A recommended device for monitoring airflow is the pressure manometer. A nasal cannula attached to a pressure transducer provides a semi-quantitative airflow signal. The result is the ability to detect inspiratory airflow limitation, and an increased sensitivity for the detection of hypopneas and RERAs.[79,80] Nasal pressure transducers that have improved the diagnostic sensitivity of the polysomnogram in terms of airflow limitation can be used in children as young as 2 years old for at least half of the night,[79] and our laboratory uses them in infants. The notable problem with the cannula in these studies is the significant amount of time in children during which there is no signal because it has been pulled off or dislodged. Artifacts are caused by mouth breathing, nasal obstruction, and obstruction of the cannula from secretions, all of which are particularly common in children. To ensure a consistent signal throughout the night, diligence is required on the part of the technician. Our research laboratory utilizes a nasal pressure transducer in conjunction with an oral thermistor to detect mouth breathing and to minimize artifact.

A variety of noninvasive methods have been used to detect upper airway flow limitation, including analysis of the systolic BP profile,[81] pulse transit time (PTT),[54] upper airway impedance using forced oscillatory flow,[82] respiratory inductance plethysmography,[83] and inspiratory flow contour.[84] Of these methods, inspiratory flow contour analysis has been shown[84] to accurately identify changes in upper airway resistance. The shape of a normal inspiratory flow vs time signal is rounded or sinusoidal. A flattening or plateau of this morphology implies flow limitation. These flow signals can be obtained by the pressure transducer cannula discussed in the previous paragraph. Care should be taken to avoid filters on the flow signal since flow changes may be disguised.

End-tidal carbon dioxide ($Et\overline{CO}_2$) is another qualitative assessment of airflow. Hypopneas and apneas will often result in a reduction or absence of the $Et\overline{CO}_2$ signal, respectively. Expired CO_2 measurement by the pressure transducer cannula, however, has not been assessed for reliability in the detection of respiratory events, as it becomes easily clogged with secretions that are common in children and, thus, likely underestimates the occurrence of hypopneas and other subtle respiratory events. However, this device is important as the time spent with hypercapnia above certain thresholds should be taken into consideration for the detection of obstructed airflow or hypoventilation.[24]

A lightweight quantitative pneumotachometer that attaches to a small, tight-fitting, nasal mask is being

assessed in our research laboratory (modified Pitot Tube; Key Technologies; Baltimore, MD). This device, which is light on the nose, has not had an apparent effect on sleep in initial studies in children. It is presently investigational but will be commercially available soon.

Pulse Oximetry

Until recently, movement artifacts in infants and children have impaired the diagnostic usefulness of pulse oximetry during sleep. New technology that decreases the number of these false-negative events is now available. The Masimo oximeter (Irvine, CA) was compared with the Nellcor 200 (Pleasanton, CA) and was found to be superior in event detection during movement.[85] When the averaging time on the pulse oximeter was reduced to 2 s, more short saturation declines were detected. The averaging time of the pulse oximeter should be taken into consideration in the pediatric sleep laboratory. Short apneas of < 10 s in rapidly breathing infants can cause significant drops in oxygen saturation that are missed with pulse oximeters that average the signal over a long period of 15 to 20 s.

Carbon Dioxide Measurements

$Et\mathrm{CO}_2$ monitoring is the noninvasive measurement of exhaled CO_2, which is standard practice in most pediatric sleep laboratories. Caution should be used when measuring $Et\mathrm{CO}_2$ at the nose with a cannula, since adding too many monitors to the nose may cause iatrogenic obstruction. Additionally, $Et\mathrm{CO}_2$ measurements are underestimated during low-tidal-volume tachypnea in infants,[86] or when performed simultaneously with positive-pressure delivery devices or oxygen, due to the addition of high flow near the site of measurement.

Transcutaneous monitors offer a noninvasive method of measuring carbon dioxide levels without obstructing nasal airflow.[86,87] The monitors heat the local tissue to improve capillary flow and estimate $Pa\mathrm{CO}_2$ levels. The transcutaneous CO_2 monitor has been found to be more accurate than end-tidal monitors,[87] but accuracy is reduced in patients with thick skin or peripheral edema, and in poorly perfused areas. The limitation of the transcutaneous monitor is the slow reaction time and the inability to detect breath-by-breath CO_2 changes, as well as the risk of burn. When heated to 43°C and sampled at 100 Hz, response time is much improved; however, at that temperature, burns are more likely on sensitive skin, and the probe site should be changed more often than every 4 h (some clinicians have suggested changing it every 2 h).

Measuring Arousals

An investigational device used in some sleep laboratories, PTT, potentially aids in the recognition of arousals, which normally depend on the accuracy of EEG scoring.[23,54] Upper airway obstructive events in sleeping children may terminate without visible EEG arousal.[88] The PTT is a noninvasive marker of BP and, therefore, of subcortical arousal.[54,89] The PTT is the interval between the R-wave of the ECG and the arrival of the photoplethysmographic pulse at the finger. BP elevation, which is associated with respiratory arousal from sleep, results in a drop in the PTT. Although the usefulness of the monitor was decreased when used alone, and is limited during movement, in conjunction with other respiratory indexes on the polysomnogram, PTT improved the detection of hypopneas that were missed by scoring EEG arousals.[23]

Another investigational measurement of arousal activity is the spectral analysis of the EEG signal throughout the night and in specific sleep stages. This spectral analysis is now available by digital acquisition of the EEG signal on the polysomnogram, offering new ways to interpret normal vs pathologic arousal patterns that might contribute to daytime symptoms of SDB. Cyclic alternating patterns (CAPs) allow a longer term evaluation of sleep where brief and frequent arousals appear as a prominent feature (Fig 1).[90] A CAP is a periodic EEG activity of non-rapid eye movement sleep that is characterized by repeated spontaneous sequences of transient events (phase A) that differ from the background rhythm sleep stage with an abrupt frequency and/or amplitude variation, recurring at intervals up to 1 min long.[91] Respiratory events are noted to affect the pattern of arousal, and this pattern, when analyzed with spectral analysis, may add to the sensitivity of polysomnography to correlate respiratory events with symptoms.[92] The clinical utility and correlation of EEG spectral analysis with treatment outcomes remains to be determined.

SDB is accompanied by autonomous nervous system changes in sympathetic activity, BP, and peripheral vascular resistance.[93] Peripheral arterial tonometry (PAT) determines the peripheral arterial vascular tone using a noninvasive plethysmographic manometer[94] or a watch combined with oximetry and actigraphy.[95] Studies assessing the use of PAT (Watch PAT 100; Itamar Medical Ltd; Caesarea, Israel) in the home setting of adult subjects, compared to unattended[96] and attended polysomnography,[97] have demonstrated that PAT is able to identify SDB events and might be a useful tool to screen for OSA. PAT has also been shown to detect arousals reliably in both adults[95]

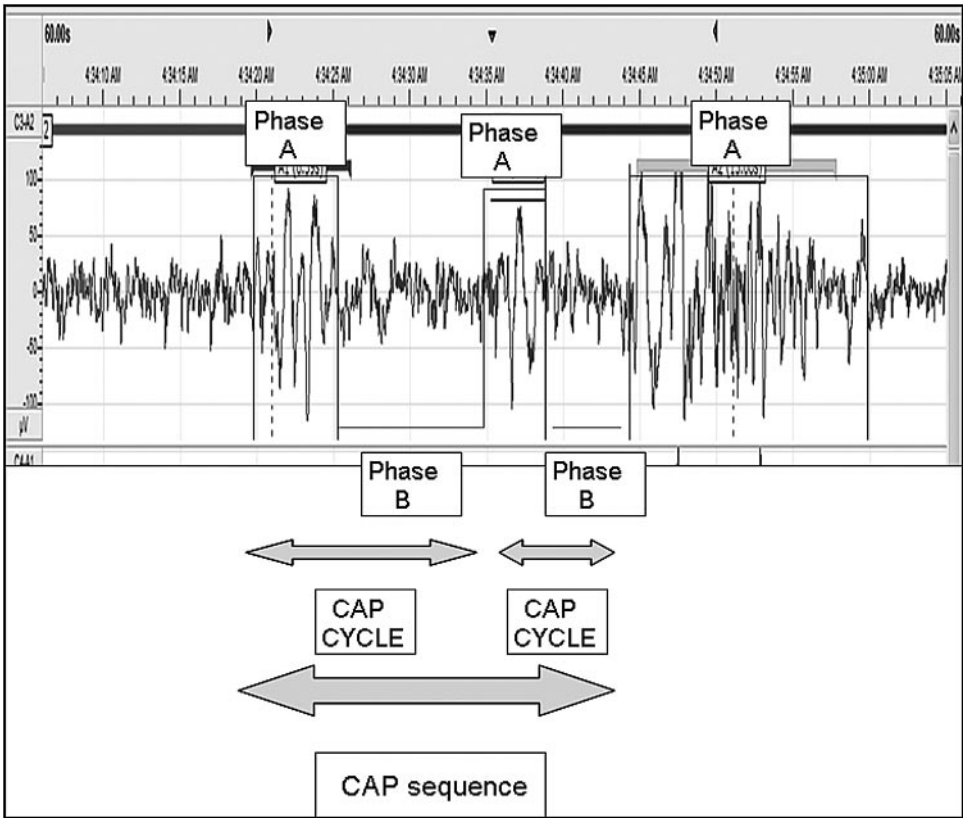

FIGURE 1. CAP phase A is considered to be a periodic EEG activity during non-rapid eye movement sleep. It is an activation phase that includes high-voltage slow waves (synchronization) or low-voltage fast waves (desynchronization). CAP phase B is described as the interval between two phase A intervals, corresponding to the stage-related background activity. CAP cycles are defined as the sum of A and B phases, and each CAP sequence consists of at least two consecutive CAP cycles. The figure was provided by M. Cecilia Lopes, MD, Stanford University Sleep Medicine Program, Stanford, CA.

and children, although in children a significant number of events identified by PAT were not accompanied by EEG arousals.[94] Thus, PAT might be useful for screening children for OSA, and might also aid in prospective studies to evaluate the current arousal criteria or respiratory effort-related events in children.

Measuring Work of Breathing

Esophageal pressure manometry remains the "gold standard" for detecting increased respiratory effort, and the use of this device is suggested to improve the diagnosis of RERAs by the ICSD-2, but a survey reported at the recent annual Sleep Disorders in Infancy and Childhood Conference (for general information, go to: http://www.5starmeded.org/sleepdisorders/overview.html) suggest that very few pediatric sleep laboratories utilize these devices. The use of a pediatric feeding catheter instead of the esophageal balloon has made the procedure more tolerable in both adults and children; Virkkula and colleagues[98] showed that esophageal pressure is well tolerated, adds much diagnostic information, and can lead to cost savings if used instead of polysomnography, although some children or parents may continue to have anxiety about the invasiveness of the probe.[99]

PTT, which was discussed in the previous section in relation to the measurement of subcortical arousals, is also a noninvasive indirect measurement of work of breathing by virtue of the swings in BP associated with inspiration against resistance.[89] The use of this device may add to the diagnosis of RERAs, but is limited by movement artifacts.

Respiratory inductive plethysmography (RIP) has been used for both a qualitative assessment of respiratory effort and, when the plethysmograph has been properly calibrated, as a noninvasive quantification of lung volume.[100] Grigg-Damberger et al[58] evaluated the use of both RIP and Piezo crystal belts as qualitative signals for respiratory effort, and found increased identification of paradoxical thoracic and abdominal movement with the use of the Piezo crystal belts. Previous studies[101] evaluating RIP have verified that it effectively measures lung volume

changes when the plethysmograph is properly calibrated. There is concern, however, about the validity of measures of lung volume after changes in body position without recalibration, which could be difficult to maintain in children due to frequent movement during sleep. Thus, while RIP is a promising diagnostic tool, future prospective studies should be encouraged to evaluate the utility of RIP as both a qualitative measure of respiratory effort, which would benefit from comparison to the results of esophageal manometry, and its utility as a noninvasive quantification of lung volume, including recalibration techniques after changes in body position.

Treatment Considerations

Although individual sleep laboratories define their own treatment criteria, there is still a lack of consensus on the level of severity of SDB that justifies treatment in children. Recently, Chervin and colleagues[11] demonstrated that children with symptoms of SDB who were treated with adenotonsillectomy (T&A) improved in assessments of hyperactivity, inattention, and sleepiness, and even in the diagnosis of attention deficit-hyperactivity disorder after a 1-year follow-up. Importantly, polysomnographic parameters did not predict which children would have neuropsychological problems or which children would improve, but children were referred for T&A for clinical symptoms regardless of the diagnosis of apnea obtained by polysomnogram. These findings demonstrate the need for alternative technologies to detect SDB and the need to define treatment criteria in children.

Surgical Treatment of SDB

T&A is generally considered to be the standard treatment of childhood sleep apnea in children with normal craniofacial features and uncomplicated medical status. T&A is curative of sleep apnea in the majority of pediatric cases.[102] The success rate of T&A was recently analyzed in a metaanalysis[102] of 14 studies evaluating polysomnographic cure rates before and after surgery. While the definition of success varied (AHI range, 0.5 to 5 events per hour), the overall cure rate was 82.9% (95% confidence interval, 76.2 to 89.5%; $p < 0.001$). For those studies that defined success as an AHI of 1, the cure rate ranged from 53 to 100%. It is important to note that the method of T&A was not addressed in this study, and the T&A technique may have confounded these results as the use of a partial vs total tonsillectomy or tonsillar pillar manipulation may affect success and cure rates.

The surgical treatment of SDB focuses on relieving airway obstruction in the retroglossal region, the nose, and/or the retropalatal region. T&A potentially addresses all three areas of obstruction. T&A was traditionally performed with snare or cold knife techniques. New techniques such as monopolar and bipolar electrocautery, harmonic scalpel, microdebrider, and coblation have all been reported to be equivalent with respect to efficacy, postoperative pain, and hemorrhage rates (and they have the advantage of causing less intraoperative blood loss than traditional techniques).[103–106] The addition of uvulopharyngopalatoplasty to T&A, which addresses retropalatal obstruction, has been be advocated in neurologically impaired children such as those with cerebral palsy and Down syndrome.[107,108]

Nasal obstruction has traditionally been addressed with inferior turbinate reduction, septoplasty, and nasal valve surgery. More recently, the rapid maxillary expansion technique has been advocated to increase nasal cavity volume and therefore decrease nasal resistance.[109] A study of 31 children with a normal body mass index and a lack of adenotonsillar hypertrophy showed a significant reduction in the mean AHI for all children (12.2 events per hour preoperatively compared to 0.4 postoperatively). An older prospective study[110] showed that improvement in nasal airflow and resistance occurred in a 45% of 16 pediatric patients. Concerns about facial growth tend to limit extensive nasal surgery in young children, although turbinate reduction appears to be less problematic.

Surgeries to address retroglossal obstruction include tongue reduction, genioglossal advancement, hyoid myotomy, and suspension, all of which are infrequently performed in the pediatric population. However, recent studies[111] using MRI compared the presence or absence of lingual tonsils in children and found that they were absent in 100% of control subjects but present in 33% of children with SDB (even after T&A), and in 50% of children with SDB and Down syndrome, suggesting that tongue base procedures may be underutilized in this population. For patients with life-threatening OSA, and especially those with craniofacial anomalies, mandibular distraction, maxillomandibular advancement, and/or tracheotomy may be necessary.

Medical Treatment of OSA

A number of medical therapies have been investigated for the treatment of OSA. Adenotonsillar tissue reveals the presence of glucocorticoid and leukotriene receptors in children with OSA more so than those found in children with recurrent tonsillar infection as demonstrated by polymerase chain reac-

tion.[112,113] Based on these findings, the use of systemic steroids was evaluated in nine patients with polysomnography-confirmed OSA with no improvement noted after a 5-day course of oral prednisone.[114] Further studies[115] using nasal steroids have demonstrated moderate improvement, but not cure, in patients with OSA either by reduction in adenotonsillar tissue size or due to independent factors. A 16-week course of leukotriene inhibitor treatment led to moderate reductions in OSA along with a reduction in adenoid size in 24 children with mild OSA.[116] The combination of nasal steroid therapy plus leukotriene inhibition will reduce snoring and mild SDB, and may be an important additional therapy to consider in children who snore without significant gas-exchange abnormalities.

Follow-up from therapy should include an assessment of treatment efficacy. Since significant SDB symptoms or respiratory parameters fail to improve in about 20% of children, perhaps more attention should be paid to follow-up evaluation than is presently practiced.[117] T&A is a first-line surgical therapy for SDB in children, but identification of the site of airway resistance and alternative surgeries should be given consideration in those children who demonstrate ongoing symptoms of obstructed airflow.[45]

Positive Airway Pressure Treatment

Continuous positive airway pressure (CPAP) is first-line therapy for adult sleep apnea and is considered second-line therapy in children since surgical therapy is generally curative. The American Academy of Pediatrics 2002 Clinical Practice Guideline[37] recommends CPAP as an option for the treatment of children who are not candidates for surgery or do not respond to surgery. CPAP has been studied in children,[118,119] has been determined to be effective for the treatment of OSA[120] and other hypoventilation syndromes,[121–123] is less invasive than surgery,[122] and offers temporary treatment for conditions such as postoperative airway obstruction.[120,123] CPAP has been approved by the US Food and Drug Administration for use in children >7 years of age in the United States.

Studies have shown that CPAP therapy is effective in children younger than 7 years of age, as it has been used in children ranging from the preterm infant[124] to the adolescent[118,125] including more than 60 articles on the use of positive-pressure noninvasive ventilation in the neonate as a noninvasive alternative to mechanical ventilation with an endotracheal tube or tracheostomy. Despite data in support of CPAP use in younger children, at the time of this publication there are no US Food and Drug Administration-approved nasal interfaces that are available for infants or toddlers for home treatment of SDB in the United States. The devices are widely available in most other industrialized countries, even by device companies based in the United States. The lack of safe, available pediatric medical devices, studied in and designed for children, such as nasal interfaces for CPAP is due to the decreased market value of pediatric devices compared to the relatively large profit margin afforded by the development and marketing of adult devices.[126] This situation puts US health-care workers in the uncomfortable position of being unable to treat children in whom SDB has been diagnosed. Many health-care workers resort to physically altering equipment that is designed for use in adults,[121] making homemade nasal interfaces,[122] prescribing inadequate therapies such as oxygen treatment, or sending the patient for invasive surgical therapies such as tracheotomy.[122] Significant action and communication between advocates of respiratory medical care and device companies will need to take place in order to bring standard-of-care therapy for SDB to children in the United States.[126]

Conclusion

SDB in children is common. The impact of SDB on the growth and development of a child may have detrimental effects on health, neuropsychological development, quality of life, and economic potential; therefore, SDB in children should be recognized as a public health problem as it is in the adult population. Polysomnographic technology used in the majority of sleep laboratories may be inadequate to diagnose subtle forms of clinically important airflow limitation that are associated with daytime dysfunction, but new diagnostic technologies are on the horizon. In the meantime, the combined use of multiple diagnostic sensors, and the consideration of the use of the "gold standard" equipment such as the esophageal pressure manometer will improve diagnostic sensitivity. Studies to determine the diagnostic monitoring systems that best correlate with SDB symptoms and with treatment efficacy are needed to improve the standardization of diagnostic criteria. It behooves the device manufacturers to work closely with health-care providers to update and improve the sensitivity of diagnostic equipment, and to improve the availability of much needed therapeutic devices for children.

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