320

American Thoracic Society
MEDICAL SECTION OF THE AMERICAN LUNG ASSOCIATION

Standards and Indications for Cardiopulmonary Sleep Studies in Children

THIS OFFICIAL STATEMENT OF THE AMERICAN THORACIC SOCIETY WAS ADOPTED BY THE ATS BOARD OF DIRECTORS, JULY 1995

CONTENTS

Sleep induces changes in the function and control of the respiratory system. These changes may result in clinically significant abnormalities in upper airway function and gas exchange in both normal children and those with underlying respiratory or central nervous system disease (1, 2, 3, 4).

To clarify the state of the art for pediatric breathing and sleep disorders and to develop recommendations about standards and indications for cardiopulmonary sleep studies in children, the Pediatric Assembly of the American Thoracic Society convened a consensus conference of individuals with expertise in pediatric pulmonology, otolaryngology, neonatology, plastic surgery, neurology, and developmental respiratory physiology. The format of the conference was similar to that used to develop standards for adults (5). This summary has been reviewed and revised extensively by the committee and members of the pediatric assembly.

The conference had several goals:

- to define the indications for evaluating breathing during sleep in children and adolescents.
- to develop standards for the indications, techniques, and interpretation of polysomnography (PSG) for evaluating breathing disorders during sleep in children and adolescents.
- to identify areas where the knowledge base is lacking and will require research in order to establish recommendations.

RESPIRATORY INDICATIONS FOR POLYSOMNOGRAPHY IN CHILDREN

Obstructive Sleep Apnea Syndrome

Background. Obstructive Sleep Apnea Syndrome (OSAS) in children is a disorder of breathing during sleep characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction (obstructive apnea) that disrupt normal ventilation during sleep and normal sleep patterns (6, 7). It has been estimated that approximately 7 to 9% of children snore regularly (8, 9, 10), with an estimated prevalence of OSAS at 0.7% in 4- to 5-yr-old children (9). Most affected children breathe normally while awake. However, a minority with marked upper airway obstruction also have noisy, mildly labored breathing when awake. Clinical manifestations of OSAS in children include chronic mouth breathing, snoring, and restlessness during sleep, with or without frequent awakenings (6, 7). Loud snoring that disturbs and concerns parents is a common indication for evaluation. However, clinical experience suggests that some infants with clinically significant OSAS may have little or no snoring. Less frequently, daytime hypersomnolence, failure to thrive, and cor pulmonale may be seen (6, 7, 11–14). The frequency of behavioral, personality, and learning problems in children with OSAS is unknown, but such problems may prove to be common manifestations as more experience is accumulated (6, 11). An association between OSAS and enuresis has been suggested (15, 16). Systemic hypertension secondary to OSAS is uncommon in children (17).

Major risk factors for OSAS in children include hypertrophy of the tonsils and adenoids (6, 7, 15), neuromuscular disease including conditions associated with both muscular hypotonia and hypertonia (18, 19), obesity (20), and genetic syndromes, especially those associated with midface hypoplasia, small nasopharynx, or micrognathia, such as Down syndrome and Pierre Robin sequence (21–23). Less common risk factors for OSAS are laryngomalacia (24), pharyngeal flap surgery (25), sickle cell disease (26), structural malformations of the brainstem (27), and certain metabolic and genetic disorders (28). Viral respiratory infections and allergic rhinitis are not primary risk factors for OSAS, but they may exacerbate existing OSAS in affected children (29).

Clinical experience suggests that the pathophysiology, clinical manifestations, diagnosis, and management of children with suspected obstructive sleep apnea are different than for adults (30, 31).

Consensus recommendations.

- Polysomnography is recommended to differentiate benign or primary snoring, i.e., snoring not associated with apnea, hypoventilation, or evidence of cardiovascular or central nervous system effects, for which treatment is rarely indicated, from pathologic snoring (OSAS), i.e., snoring associated with either partial or complete airway obstruction, hypoxemia, and sleep disruption (32). A history of loud snoring alone has not been shown consistently to have sufficient diagnostic sensitivity upon which to base a recommendation for surgery, whether adenotonsillectomy, uvulopalatopharyngoplasty (UPPP), or tracheostomy (33–35). Polysomnography findings suggestive of abnormality are included in the section on interpreting PSG data.
- Polysomnography is indicated for evaluating the child with disturbed sleep patterns, excessive daytime sleepiness, cor pulmonale, failure to thrive, or polycythemia unexplained by other factors or conditions, especially if the child also snores.
- In the child who has clinically significant airway obstruction (apnea, retractions, paradoxical respiration) during sleep as

Am J Respir Crit Care Med Vol 153. pp 866–878, 1996
Revised 4/1/96

observed by medical personnel, or documented by audiovideo recording, a PSG to confirm the clinical diagnosis may be deferred in order to proceed with therapy expeditiously. Polysomnography is recommended if the physician is uncertain whether the clinical observation of obstructed breathing is sufficient to warrant surgery or if a child needs intensive postoperative monitoring following adenotonsillectomy or other pharyngeal surgery (36). Risk factors for postoperative complications include age less than 2 yr, those with more than 10 obstructive events per hour of sleep, those with Sa_{O_2} less than 70%, and those with underlying neuromuscular disease or craniofacial abnormalities, specifically those associated with midface hypoplasia and retro- or micrognathia (21, 25, 36, 37).

- Polysomnography is recommended in children with laryngomalacia whose symptoms (stridor and work of breathing) are worse asleep than awake or who have failure to thrive or cor pulmonale (24).
- Although obesity is a risk factor for OSAS in children (20, 38), its presence alone is not an indication for PSG unless the child also exhibits unexplained awake hypercapnia, chronic snoring, increased work of breathing during sleep, disturbed sleep, daytime hypersomnolence, polycythemia, or cor pulmonale (20, 38).
- Polysomnography is recommended during evaluation of the child with sickle cell disease who has either the typical signs and symptoms of OSAS or frequent veno-occlusive crises occurring during sleep (26, 39). Oxygen saturation data from pulse oximetry must be interpreted cautiously in this population, because sickle hemoglobin may adversely affect the accuracy of oximetry (40).
- Repeat PSG is recommended for children previously diagnosed with obstructive sleep apnea who exhibit persistent snoring or other symptoms of sleep-disordered breathing. If possible, based on the child's clinical condition, this study should be deferred until at least four weeks postsurgery to allow for resolution of postoperative edema.
- If continuous positive airway pressure (CPAP) is used in the management of OSAS or other respiratory conditions, PSG should be used to titrate the level of CPAP and to reevaluate periodically the appropriateness of the settings.
- When weight loss is the primary therapy for OSAS in an obese child, PSG should be repeated to determine if the weight loss program has decreased the severity of OSAS.
- Children with mild to moderate OSAS who have complete resolution of snoring and disturbed sleep patterns after therapy do not need a follow-up PSG. However, in a child under 1 yr or a child with severe OSAS based on clinical symptoms, the number of obstructive events, or severe desaturation episodes, follow-up PSG should be considered to assure resolution of clinically significant abnormalities (41). Whether or not a follow-up study is ordered, parents and primary care providers should be taught the signs and symptoms of a recurrence of OSAS. The child should also have routine clinical follow-up to ensure early detection of a recurrence of abnormal breathing during sleep.
- If excessive daytime sleepiness is found not to be due to OSAS based on the results of nocturnal PSG, or if excessive daytime sleepiness persists after treatment, a multiple sleep latency test (MSLT) can be used to quantitate excessive daytime sleepiness and determine if it is secondary to narcolepsy (42). The MSLT can also be used to monitor the response to treatment of OSAS or narcolepsy. It should be performed on the day following a nocturnal PSG, so that nocturnal factors that may contribute to daytime sleepiness can be controlled and eliminated. Age-appropriate reference values should be used (43, 44). A sleep diary can also be used to confirm adequacy of the sleep periods.
- Assessment of sleep and breathing in the home using video and cardiorespiratory recordings with extended oximetry appears promising, but recommendations regarding their use require further clinical trials (45, 46).
- Extended oximetry recording alone can be used to identify hypoxemia during sleep in patients with loud snoring. However, the absence of hypoxemia during sleep does not preclude clinically significant OSAS. Conversely, the presence of hypoxemia during sleep is not in itself diagnostic of OSAS, as it may be caused by other respiratory conditions.

Bronchopulmonary Dysplasia

Background. Bronchopulmonary dysplasia (BPD) is a form of chronic lung disease that follows an acute lung injury in the neonatal period. It is characterized by persistent signs of respiratory distress (tachypnea and dyspnea), the need for supplemental oxygen beyond the first month of life to treat hypoxemia, and characteristic radiographic findings (47, 48).

Some infants and children with BPD have been shown to have prolonged episodes of hypoxemia during sleep, despite the presence of adequate oxygenation while awake (3, 49–54). In addition, infants with BPD, who are hypoxemic while awake, may experience worsening of hypoxemia while asleep. Abnormal duration of rib-cage paradox during inspiration while in rapid eye movement (REM) sleep and abnormal rib-cage paradox during non-REM sleep have also been reported (51-53). During REM sleep, intercostal muscles and other accessory muscles of inspiration are inhibited. In infants with BPD, the combination of abnormal pulmonary mechanics and REM-related loss of inspiratory muscle activity results in rib-cage paradox and hypoxemia (52-54). These episodes are not always predicted by awake blood gas measurements and may not be detected by monitoring oxygenation during daytime naps, direct observation, or by recording techniques with sluggish response time, such as transcutaneous Po_2 monitoring (50,55).

Consensus recommendations.

- Assessment of Sa_{O_2} when the infant is awake may not accurately predict hypoxemia during feeding and sleep (56). Oxygen saturation during feeding and sleep should be measured for an extended period (hours) in these groups: (*1*) infants and children with BPD who are on oxygen, to know that they have enough oxygen to keep saturation values above 92% during these periods, and (*2*) infants and children with BPD who have recently been weaned to room air when awake. The latter patients need to have saturation measured for an extended period during sleep to know that they no longer need oxygen.
- Under certain circumstances, patients with BPD who have had supplemental oxygen discontinued should undergo continuous documentation of Sa_{O_2} during sleep. These patients include infants who develop, after supplemental oxygen has been discontinued, polycythemia, cor pulmonale, failure to thrive (unexplained by other factors such as nutrition or other metabolic condition), disturbed sleep patterns, or apnea and bradycardia during sleep.
- If bradycardia without apnea is documented by impedance monitoring in infants with BPD, PSG may be indicated to detect upper airway obstruction during sleep. Similarly, if the infant with BPD is suspected of having airway obstruction during sleep based on observation of snoring, PSG is required.
- An infant who is receiving supplemental oxygen therapy and develops any of the above complications should have, in addition to measurements of Sa_{O_2} while awake, continuous documentation of Sa_{O_2} overnight, while asleep, to determine the adequacy of supplemental oxygen being delivered. Recording of the plethysmographic waveform or the pulse amplitude

modulation signals, in addition to Sao_2, is necessary to distinguish true drops in saturation from apparent drops due to movement artifact or a weak pulse signal.
- Polysomnography can be used with esophageal pH monitoring to document the temporal relationship between gastroesophageal reflux and respiratory events such as obstructive apnea, cough, or hypoxemia.

Cystic Fibrosis

Background. Episodes of desaturation unrelated to apnea have been observed in some patients with cystic fibrosis (57–60). Patients with an awake $Pa_{O_2} < 60$ mm Hg spend > 80% of sleep time with O_2 saturations < 90%, while those with an awake $Pa_{O_2} > 70$ mm Hg spend < 20% of their time asleep with Sa_{O_2} < 90% (61). In an individual patient, clinical scores, awake oxygenation status, pulmonary function, and the response to exercise have not been shown to predict hypoxic events during sleep (60, 61).

Consensus recommendations.

- Patients with cystic fibrosis who have an awake $Pa_{O_2} < 70$ mm Hg or an equivalent Sa_{O_2} (< 95%) during a period of disease stability are at risk for worsening hypoxemia during sleep and should have continuous documentation of Sa_{O_2} during nocturnal sleep. This study should be performed during a period of disease stability (generally at least 2 wk following treatment for an acute pulmonary exacerbation) in order to determine the extent and severity of sleep-associated hypoxemia and confirm the adequacy of prescribed supplemental oxygen. Random, brief checks of saturation (< 5 min recording) or visual observation of the patient during sleep are not adequate to identify desaturation episodes and will not detect OSAS (62).
- Patients with cystic fibrosis who develop polycythemia, cor pulmonale unexplained by awake blood gas or Sa_{O_2} measurements, or who complain of headaches upon awakening, excessive daytime sleepiness, or disturbed sleep patterns should also undergo continuous documentation of Sa_{O_2} for at least 8 h overnight during sleep.
- Patients with cystic fibrosis receiving supplemental oxygen may require PSG to rule out OSAS, if there is a history of snoring, desaturation episodes during sleep, cor pulmonale, polycythemia, or disturbed sleep. Polysomnography should also be considered for assessing the potential adverse effects of supplemental oxygen during sleep in patients with advanced lung disease who are hypercapnic when awake.

Asthma

Background. Early morning worsening of asthma is seen in children and adolescents (63, 64). The circadian variation in airway caliber seen in normal children is amplified in patients with asthma and may produce as much as a 50% decrease in peak flow rate (64). These changes in flow rates do not appear to be caused by sleep *per se*, but rather to be due to diurnal variations in pulmonary function (64, 65). Patients with asthma, including those suboptimally controlled, tend to have small changes in Sa_{O_2} during sleep (66, 67).

Consensus recommendations.

- In children with asthma symptoms during sleep, a thorough clinical investigation including an assessment of the environment and the appropriateness of nocturnal therapy is indicated to identify factors that may contribute to these symptoms. If there is concern about the presence of gastroesophageal reflux during sleep as a trigger for nocturnal symptoms, PSG with esophageal pH monitoring should be considered. Continuous documentation of oxygenation during sleep is infrequently needed in routine management of children with asthma, but should be considered for children with poorly controlled nocturnal asthma who exhibit disturbed sleep, morning headaches, or cor pulmonale.

Neuromuscular Disease

Background. Children and young adults with a variety of pediatric neuromuscular disorders are at risk of developing both central and obstructive apnea/hypoventilation during sleep. These disorders include Duchenne muscular dystrophy (68), myotonic dystrophy (69), spinal muscle atrophy (70), diaphragmatic paralysis (71), cerebral palsy, poliomyelitis (72), and congenital muscle diseases (73-76). Abnormal breathing during sleep in these disorders is often not predicted by awake pulmonary function testing, arterial blood gases, or the degree of muscle involvement (68, 70, 75-85).

Children with cerebral palsy and other static encephalopathies often experience bulbar involvement and glottic muscle dysfunction. If pharyngeal muscles are more severely affected than the diaphragm or other inspiratory muscles, the usual reduction of upper airway muscle tone that occurs during sleep can precipitate obstructive sleep apnea (71). This vulnerability to respiratory abnormalities is most pronounced during REM sleep, when inhibition of accessory muscles of respiration requires the diaphragm to assume more of the work of breathing. Children with central nervous system disease and evidence of pharyngeal dysfunction should be considered at risk for obstructive sleep apnea and obstructive hypoventilation (86, 87).

Consensus recommendations.

- Polysomnography, including either end-tidal or transcutaneous CO_2 monitoring, is indicated in evaluating children with neuromuscular disease who demonstrate impaired respiratory muscle function evidenced by a FVC < 40%, a peak inspiratory pressure < 15 cm H_2O, and/or phyaryngeal dysfunction (snoring or swallowing abnormalities).
- Polysomnography, including some measurement of CO_2, is indicated in children with neuromuscular disease who develop snoring, cor pulmonale, morning headaches, personality or behavioral changes, failure to thrive, or developmental delay disproportionate to the degree of neurologic impairment or the typical course of the disease.
- Polysomnography with CO_2 monitoring is indicated for planning and implementing elective nocturnal assisted ventilation for patients with ventilatory muscle weakness.
- Polysomnography with CO_2 monitoring is indicated in children with neuromuscular disease to assess the adequacy of ongoing home respiratory support, including supplemental oxygen, CPAP, or assisted ventilation. Periodic reassessment should be scheduled according to the child's growth rate and degree of clinical stability, but generally it should be at least annually (88).
- Polysomnography with CO_2 monitoring should be used during preoperative and postoperative assessment of children with neuromuscular disease before major upper airway, thoracic, abdominal, or orthopedic surgery, to detect unsuspected hypoventilation that could be aggravated by sedation, analgesia, and anesthetics.

Alveolar Hypoventilation Syndromes

Background. Alveolar hypoventilation in the absence of underlying primary pulmonary disease or respiratory muscle dysfunction is usually the result of abnormal central integration of chemoreceptor signals. This process may be primary, as in idiopathic congenital central hypoventilation syndrome, or it may be secondary to diseases of the spinal cord or brainstem, such

as Arnold-Chiari malformation. The respiratory deficit is typically more severe during sleep as is characterized by alveolar hypoventilation resulting is hypoxemia dn hypercarbia (89-92).

Consensus recommendations.

- Polysomnography including assessment of CO_2 is indicated to determine the nature and severity of the ventilatory deficit in children with alveolar hypoventilation syndrome.
- Polysomnography is indicated periodically to determine the adequacy of ventilation and oxygenation provided by artificial ventilatory support, including diaphragmatic pacing (92). The frequency of follow-up study will vary depending on the clinical stability of the child, but such studies should occur at least annually. Polysomnography should be used to determine the effects of any pharmacologic trials.
- Polysomnography is recommended for any patient with a clinically stable central hypoventilation syndrome who develops cor pulmonale, polycythemia, morning headaches, deterioration in mental status, or altered growth patterns.

Apnea/Bradycardia in Infants

Background. Infants, especially those born prematurely, frequently experience apnea or bradycardia during sleep and feeding during the first several weeks of life (93). Virtually all infants under 1000 g will experience apnea (94). Apnea may be mixed, central, or obstructive (94, 95).

An apparent life-threatening event (ALTE) is an episode of apnea, color change (pallor, cyanosis, or erythema), and hypotonia that the observer believes to be life-threatening to the infant and for which some intervention (stimulation, shaking, and/or cardiopulmonary resuscitation) is felt to be required. The major concerns following one of these episodes relate to the risk of recurrent events and death. Because this topic was discussed in detail at a National Institutes of Health–sponsored consensus conference (96), this ATS committee decided to limit present discussion of this topic to how PSG should be used for evaluating these patients.

Consensus recommendations.

- Polysomnography is not indicated for routine evaluation of infants with an uncomplicated ALTE.
- Polysomnography may be helpful in defining the frequency and type of apnea and the extent of cardiac, blood gas, and sleep alterations in certain infants with apnea or ALTE. These patients include infants with suspected obstructive apnea, those with recurrent isolated bradycardia without central apnea, and those suspected of having abnormal respiratory control.

GUIDELINES FOR PERFORMING POLYSOMNOGRAPHY IN CHILDREN

Pediatric sleep and breathing disorders affect patients from infancy through adolescence. Consequently, the pediatric sleep laboratory must be able to accommodate a wide range of physical, developmental, and behavioral challenges. Children may be easily frightened by sleeping in a strange environment, attached to a variety of monitoring devices. Success in studying children requires a comprehensive approach including recognition of the unique needs of children. Polysomnography for cardiopulmonary indications simultaneously records physiologic variables including sleep state, respiration, cardiac rhythm, muscle activity, gas exchange, and snoring. Behavioral aspects of sleep, such as the quality of the child's sleep, may also be assessed. This assessment should be accomplished in a manner that is minimally invasive or disruptive to the child's usual sleep patterns.

Laboratory Conditions

Supervision of the laboratory. The individual responsible for overall supervision of a laboratory whose primary activity is performing PSG in infants and children with cardiorespiratory disorders should be a pediatrician with training and experience in pediatric respiratory disorders and/or sleep medicine.

We recognize that due to limited availability of PSG resources, pediatric patients may be studied in facilities that predominantly study adults. In this setting, it is strongly recommended that a pediatrician with expertise in pediatric pulmonology, neonatology, neurology, or pediatric sleep medicine oversee laboratory operations related to children. The pediatric specialist can assure that the PSG is performed, scored, and interpreted appropriately for the age and condition of the child.

If such personnel are not actively involved in the daily supervision of the laboratory, formal consultation should be obtained from physicians with expertise in pediatric pulmonology, otolaryngology, cardiology, neonatology, neurology, and behavioral medicine.

Setting. Children should be studied in a dedicated pediatric facility with a laboratory decor that is both age-appropriate and nonthreatening. If a separate pediatric laboratory is not available, an area should be designated for children. The setting should accommodate a parent comfortably during the study. A place for the parent or guardian to sleep near the child while the study is in progress is recommended. Immediate parental access to the child is often necessary to reduce fear and anxiety, especially in the younger child, and to provide ordinary child care.

Personnel. Staffing by personnel skilled in dealing with infants, children, and adolescents is required. All clinical personnel should be certified in pediatric cardiopulmonary resuscitation. They should also demonstrate knowledge of childhood behavior and the ability to deal with children of varying ages and developmental stages. Procedures should be explained to the patient and parent or guardian by someone skilled in presenting medical information to children. Audiovisual aids may be useful.

Timing of the Study

Background. Polysomnography for evaluating respiratory disorders should be performed under conditions that most closely approximate the child's usual sleep habits. Overnight sleep studies should begin at the child's usual bedtime. Although there are limited data on the usefulness of nap studies for diagnosing abnormal breathing during sleep in adults (97, 98), one study in children demonstrated that a positive nap study for obstructive sleep apnea correlated well with the findings of an overnight study (99). However, a negative nap study did not exclude the presence of obstructive apnea occurring during a nocturnal study (99). Nap studies are limited in that daytime sleep periods are shorter than overnight sleep periods, may not include REM periods, do not incorporate circadian variability, and are unusual daytime behaviors for children older than 4 yrs. In many instances, the only way nap studies can be accomplished reliably is by sleep deprivation or the use of sedatives. Both measures may increase the amount of obstructive apnea (100-104). The use of sedation can be associated with severe worsening of obstructive sleep apnea and its use is contraindicated (104). Polysomnography (including a nap study) cannot be considered normal unless it assesses breathing during at least one REM period. There was also concern, based on clinical experience, that a single REM episode may not be sufficient, since it is not clear that all REM episodes are equivalent. More data are needed to answer this question.

Consensus recommendations.

- Whenever possible, PSG should be performed as an overnight study. It should be performed without the use of sedatives or sleep deprivation.
- Daytime nap studies may be useful as a screening technique to identify disordered breathing during sleep. To be considered reliable, the nap must last at least 2 h and include at least one period of REM sleep. A normal nap study is not sufficient to exclude a diagnosis of obstructive sleep apnea in a patient with clinical manifestations suggesting OSAS or to exclude abnormal ventilation and oxygenation during nocturnal sleep in patients with obstructive lung disease.

Number of Studies

Background. Sleep during the first night in the sleep laboratory may differ from sleep on subsequent nights. Data from children and young adults demonstrates the so-called "first-night effect," marked by decreased total sleep time and percent of REM time (105–107). However, this first-night effect is not believed to alter the respiratory patterns in adults with clinically significant sleep-disordered breathing (5, 107). The consequences of the first-night effect in children with mild OSAS are not known. The night-to-night variability of respiratory patterns is also not felt to be clinically significant in adults with OSAS (108); thus, a single-night study is thought sufficient to exclude clinically important sleep-disordered breathing in adults (5). However, in children, data on the magnitude and nature of the first-night effect on respiratory patterns is limited, and at this time, there are no data on the reproducibility, sensitivity, and specificity of a single-night PSG for children of different ages.

Consensus recommendations. Until more data are gathered, for most indications, a single-night study is believed sufficient to rule out a clinically significant disorder of respiration during sleep. However, if the child does not experience at least one REM period or the parents report that what was observed during the study did not reflect a typical night's sleep or the chief complaint, consideration should be given to repeating the study. If the first study was clinically indicated and it is found to be technically inadequate, then it should be repeated.

Measurement Techniques

Respiratory variables. These measurements are obtained to assess the adequacy of ventilation, to identify and differentiate between central and obstructive apnea, and to evaluate the severity and physiologic consequences of the breathing disturbance. Respiratory parameters recorded include movements of the chest wall and abdomen, detection of airflow at the nose and mouth, and assessment of the effectiveness of respiration using oxygenation and CO_2 retention measures (109, 110). Currently, there are no noninvasive techniques that provide a comprehensive quantification of breathing during sleep. However, noninvasive qualitative or semiquantitative techniques are adequate for most clinical purposes.

Respiratory movements. A cardiopulmonary sleep study should permit the clinician to distinguish between normal respiratory effort; decreased respiratory effort, as in central hypoventilation; and increased respiratory effort, as in OSAS. In older children, paradoxical inspiratory movement of chest and abdomen is a sensitive indicator of increased airway resistance. It can be recorded using strain gauges (111, 112), respiratory inductive plethysmography (113–116), or magnetometers (117, 118). In infants, paradoxical movements are also seen during normal REM sleep, and the presence of paradoxical breathing does not necessarily indicate abnormality (119). Electromyography of the diaphragm and accessory muscles of respiration can provide additional information about activity of specific muscle groups (diaphragm, upper airway dilators, expiratory muscles, etc.). Esophageal pressure recording provides the most accurate, quantitative measurement of respiratory effort, but routine use of this invasive technique is not necessary for most clinical purposes (120–123).

Airflow measurements. Airflow can be measured by a pneumotachograph connected to nasal prongs, an oronasal mask, or tracheostomy tube. Quantitative measurements of airflow and tidal volume are most useful in research settings and assessment of central hypoventilation. Such measurements have little if any role in routine clinical studies because the mask may be uncomfortable, may frighten the child, and may alter the pattern of breathing (124). Measurement of airflow can be accomplished in a variety of ways. Oronasal thermistors and/or nasal CO_2 catheters are used most commonly in clinical laboratories. They provide qualitative airflow signals and require attachment to the face (109, 125). Thermistors may not reliably detect episodes of partial airway obstruction with reduced tidal volume (hypopneas). The laryngeal microphone has been recommended to detect airflow or its absence in patients with OSAS (126, 127). However, this technique is limited because it can only detect complete obstruction. Recording sound will only yield information on the degree and quality of snoring, neither of which has been shown to correlate with the severity of the ventilatory disturbance (33, 34).

Respiratory inductive plethysmography (RIP) provides both an assessment of the chest/abdominal asynchrony and a semiquantitative measurement of airflow and tidal volume (113–116). The method uses bands around the chest and abdomen and may detect airway obstruction without the need to attach thermistors or end-tidal CO_2 sampling catheters to the face. Calibration can be performed automatically to set the abdominal and thoracic sum signal proportional to tidal volume. This system worked well for detecting obstructive apnea as well as partial airway obstruction in children and adults (128-130) and in measuring tidal volume in infants (114).

Caution is warranted when using RIP in infants and in children with increased work of breathing. In infants, especially preterm neonates, chest/abdominal asynchrony is common as REM-sleep-related hypotopia permits inward chest motion during inspiration (119). To avoid overdiagnosing apnea/hypopnea when using REP inn infants, confirmatory channels such as thermistors or end tidal CO2 readings should be used. In patients with increased work of breathing, use of accessory muscles of breathing may lead to complex chest/abdomen movements, violating the assumption underlying RIP that the respiratory system moves only two degrees of freedom (111). As with other methods, careful attention to positioning of the RIP bands is mandatory.

Measurement of ventilation. In patients with normal lungs and unobstructed breathing, the CO_2 value measured at the nose or mouth over the last one-fifth of expiration is presumed to reflect alveolar CO_2. This value is thought to be a reliable estimate of arterial CO_2 and thus of alveolar ventilation (125, 131). Careful positioning of probes at the mouth and nose is essential to obtaining reliable recordings from both thermistors and CO_2 monitors. Although not an absolute guarantee, an end-tidal plateau of the CO_2 value usually indicates a good signal for CO_2 readings. Caution must be advised when using end-tidal CO_2 tracings. Underestimation of the actual alveolar CO_2 value can be seen in patients with obstructive lung disease or rapid respiratory rates. This error should be obvious from inspection of the end-tidal tracing. End-tidal CO_2 recording is effective in detecting apnea and prolonged hypoventilation (131, 132). The technician's vigilance is essential to ensuring that the catheter is posi-

tioned properly, that it is kept patent, and that the waveform is appropriate.

At times, the nasal sampling catheter required for the CO_2 recording may be difficult to maintain, especially in young children. In these situations, transcutaneous P_{CO_2} (Ptc_{CO_2}) monitoring may be useful (132, 133). These measurements are more reliable when corrected to the Pa_{CO_2} values (132). The Ptc_{CO_2} measurements will not reflect transient changes in Pa_{CO_2}, but only a trend (132, 133). In older or obese patients, Ptc_{CO_2} does not necessarily reflect arterial values, although the difference between the transcutaneous value and the arterial value is usually stable.

Measurement of oxygenation. Blood oxygen levels can be measured by pulse oximetry (131, 134–136) or by transcutaneous oxygen electrodes (54, 55, 137). Pulse oximetry has a rapid response time, but the result is affected by the lung-to-probe circulation time and the averaging algorithm used by the equipment. It uses a comfortable sensor that can be left on the patient for extended periods. The Sa_{O_2} values obtained with pulse oximetry have been shown to correlate well with measured arterial oxygenation values > 70%, as long as there is an adequate arterial pulse waveform and absence of motion artifact (131, 134–136). The accuracy and reliability of continuously documented oximetry can be improved by also recording the pulse amplitude signal (56). Individuals using pulse oximetry should be familiar with factors that affect the accuracy and reliability of its readings. Understanding the relationship between saturation and partial pressure of oxygen, as well as the importance of oxygen delivery is essential to using these measurements effectively (131, 135).

Transcutaneous oxygen tension measurements (Ptc_{O_2}) are less useful because the response time of these electrodes to changes in Pa_{O_2} is often too slow to follow the rapid and transient changes in oxygenation that may occur following apnea (54, 55, 131, 133). Furthermore, patient age and the temperature of the probe require repositioning the heated probe to prevent skin damage in infants and young children (138). These factors make these monitors less desirable for studies designed to be minimally disruptive to natural sleep. If transcutaneous measurements are used, the location of the sensor should be changed approximately every 4 h to prevent skin damage. Depending on the temperature of the probe and the characteristics of the equipment, this interval can be extended in older patients, but it may need to be shorter in premature infants. Alternatively, several sensor rings should be placed at the beginning of the study and the sampling location rotated every 4 h. This technique minimizes disturbances during the study.

Nonrespiratory variables.

Sleep staging. Staging sleep involves the combined measurement of the electroencephalogram (EEG), electrooculogram (EOG) to record rapid eye movements, and the electromyogram (EMG) to record submental and tibial muscle activity. Well-defined sleep stages similar to those in adults are easily identifiable in children > 6 mo old, although differences in the characteristics of the voltage and waveforms of the EEG occur with maturation beyond this age (139, 140). Special criteria have been used to define sleep states in infants < 6 mo old (141, 142).

Electrocardiogram (ECG). Monitoring cardiac rate and rhythm is useful in assessing consequences of the breathing disturbance.

Tibialis muscle activity. Monitoring of peripheral muscle tone is useful in documenting excessive movements and arousals during sleep (143). Although rare in pediatrics, the diagnosis of periodic leg movement can be detected with anterior tibialis EMG (143). Motion sensors for the extremities can also be used to detect excessive leg movements.

Esophageal pH. The measurement of esophageal pH using standard methodology (144) in conjunction with PSG can be used to document the presence and cardiorespiratory consequences of gastroesophageal reflux.

Audiovisual recording. Because the physician is not present overnight to observe the child during sleep, videotaping using infrared or low-light cameras can provide invaluable information on sleep behavior, snoring, respiratory effort, and sleep positions associated with a particular respiratory pattern. Preliminary work has suggested that videotape analysis may prove useful as a noninvasive approach to discriminate sleep and wakefulness and to help assess movement arousals (45, 46, 145).

Consensus recommendations. Comprehensive evaluation of respiration during sleep requires a combination of measurements that at a minimum should include the following techniques:

- Both respiratory effort and airflow should be assessed. Simultaneous recording of movement of the chest wall and abdomen is required to detect paradoxical inspiratory rib cage movement and identify obstructive apnea and/or hypoventilation. Magnetometers, strain gauges, or RIP may be used. Intercostal EMG recordings cannot be used alone to monitor respiration, since the signal may be reduced or absent during REM sleep. Calibrated RIP can detect both apnea and hypopnea (128, 129, 130). Esophageal pressure catheters are not required for routine clinical studies, because placement of the catheters can be upsetting to both the child and parent. The presence of the catheter may contribute to sleep disruption, leading to a less-than-optimal study.

  Airflow at the nose and mouth can be measured either by thermistors or by capnography. It is important to identify mouth and nasal breathing separately, because absence of nasal airflow or exclusive mouth breathing are both associated with clinical abnormalities (146, 147). Capnography is recommended because it can assess both airflow and ventilation simultaneously.

- Measurement of Sa_{O_2} by pulse oximetry should be performed in all studies. In addition, we strongly recommend that the oximeter's pulse waveform be recorded on a separate channel adjacent to the electrocardiogram (ECG) signal, so that the accuracy of the saturation reading can be determined and artifacts due to movement or low signal strength easily identified. Although the software used to calculate saturation may vary, the algorithm calculating the saturation value should use the mode with the shortest averaging time.
- An ECG is recorded using a standard three-lead placement.
- An EMG is recorded from electrodes placed over the anterior tibial region. Motion sensors can also be used. Recording movements of an extremity is recommended in studies focused on excessive leg movements or in children with excessive daytime sleepiness; it can help quantitate movement arousals during PSG assessment of cardiopulmonary function.
- Electrode placement for sleep staging in children is based on the International 10–20 system and is similar to that used in adults (148). Electrodes are placed at A1, A2, C3, and C4, and sleep stage is determined by the monopolar derivation C3/A2 or $C3/A_1$ or C4/A1 or $C4/A_2$. The EOG is recorded by placing electrodes adjacent to the outer canthus of each eye. The right electrode is placed 1 cm above the horizontal axis while the left is placed 1 cm below. An EMG is recorded by placing one electrode in the center of the chin and two electrodes on opposite sides under the chin. Only two of these are required for recording purposes; the third is for backup. If a paper polygraph is used, the data should be recorded at a rate of 10 mm/s. Although this may reduce the sensitivity of EEG patterns, it has been found to be sufficient for accurate sleep staging and assessing breathing patterns.
- Audiovideo recording during sleep is recommended but not

required. Infrared or low-light equipment should be used so that ambient light can be minimized. If available, simultaneous recording of the patient and PSG variables can be helpful in correlating physiologic disturbances with clinical or behavioral findings.

- Supervision by a trained technician is required to assure the quality of the study. This individual should make notations regarding unusual events or behavior during the study. Although unattended PSG in children appears promising, its role in either the laboratory or home setting has not been fully established (45, 46). Additional research is needed to determine reliability and limitations of this approach before it is adopted for routine use in children.

SCORING AND REPORTING POLYSOMNOGRAPHY DATA

Respiratory Variables

Physiologic differences between adults and children, as well as differences in the clinical manifestations of sleep-related upper airway obstruction, demand that interpretation of PSG in children recognize their uniqueness and the influences of development (30, 33).

The presence or absence of snoring should be noted. Although limited data suggest a clinically significant correlation between the quality or loudness of snoring and severity of upper airway obstruction (149, 150), there are no studies quantifying snoring on a PSG in children, nor are there widely accepted and validated scales for assessing the quality and severity of snoring in children.

The number of obstructive events (complete or partial) of any duration should be scored in all studies. Obstructive apnea is defined as cessation of airflow at the nose and mouth associated with out-of-phase movements of the rib cage and abdomen. Several investigators have demonstrated that children with OSAS may present fewer and generally shorter episodes of complete obstruction, but prolonged periods of partial upper airway obstruction (7, 30, 31).

Partial airway obstruction has traditionally been described by the term "hypopnea" (breathing that is shallower or slower than normal). Hypopnea is defined by a 50% or greater decrease in the amplitude of the nasal/oral airflow signal, often accompanied by hypoxemia or arousal (128, 129, 153). If a calibrated RIP is used, hypopnea can be identified by a decrease in the RIP sum signal (128–130). However, in young infants, rib-cage paradox characteristic of REM sleep can produce a similar decrease in the signal. If RIP is used in children < 1 yr who may normally experience paradoxical inspiratory rib-cage movements during REM sleep, then hypopnea should be defined using other supportive data such as changes in nasal/oral flow, oxygenation, or end-tidal CO_2. Hypopnea may be further characterized as either obstructive, if the reduction in airflow is associated with paradoxical chest and abdominal movements, or central, if associated with an in-phase reduction in the amplitude of the chest and abdominal signals.

Defining and identifying hypopnea consistently by this approach may sometimes be difficult (153). Scoring hypopnea as discrete events also may be difficult, because the degree of partial obstruction often changes continuously, without clear points of onset or termination. An alternative approach based on measuring end-tidal P_{CO_2} was considered by many committee members to be a more sensitive method for assessing partial airway obstruction. If end-tidal CO_2 data is used, the effects of extended partial airway obstruction or reduced respiratory effort can be assessed by measuring changes in end-tidal CO_2 associated with going to sleep, as well as with respiratory events. By using end-tidal P_{CO_2} data, hypoventilation can be identified as obstructive or central based on the associated changes in chest and abdominal wall movements. Reference data on the range of normal end-tidal CO_2 values is limited to a recent study of 50 children, 1–17 yr old (154). This study demonstrated that no normal child had an end-tidal $CO_2 > 45$ mm Hg for > 60% of total sleep time, end-tidal $CO_2 > 50$ mm Hg for > 8% of total sleep time, or peak end-tidal CO_2 (on two consecutive breaths during or after upper airway obstruction) > 53 mm Hg (154). The same study showed that the increases in $P_{ET CO_2}$ rarely exceeded 13 mm Hg during overnight PSG. The committee believed that these data may be used conservatively to identify patients with elevation of CO_2 during sleep, assuming that a high-quality CO_2 reading is obtained.

Limited data are available on the occurrence of paradoxical inspiratory rib-cage movements in normal children. In infants up to 6 mo old, these movements occur throughout REM time (155). From 7 mo to 3 yr there are no paradoxical inspiratory rib-cage movements during non-REM sleep, and the duration of paradoxical inspiratory rib-cage movements decreases with age during REM (119). In adolescents, paradoxical inspiratory movements are not seen normally, even during REM (156).

Central apnea is an absence of both airflow at the nose and mouth and movements of the chest wall and abdomen. Central apnea without physiologic consequence is found in normal children of all ages. In general, these episodes are <20 s, although some normal children have been found to have central apneas longer than that (157).

Reference data on oxygenation come from several studies of normal infants and children (154, 157–160). These data demonstrate that after the first several months of life, Sa_{O_2} usually remains > 94% during sleep, and desaturation events of > 4% are uncommon. If they occur, they are typically brief: < 10 s (154, 157–160).

Nonrespiratory Variables

If there are clearcut abnormalities on the respiratory portion of the PSG, the study may be interpreted without performing complete sleep staging. However, the committee believed that the ability to stage sleep is important to documenting the sleep-state dependence of breathing. Breathing abnormalities, including obstructive apnea, may be exacerbated or only seen during REM sleep (161–163). Sleep staging is also important to determining that the quality and quantity of sleep during the study is within normal limits.

In children older than 6 mo, it is standard practice to stage sleep on PSG in 30-s epochs, according to the guidelines of Rechtschaffen and Kales (139). Some committee members recommended that the length of epochs for sleep staging vary according to the age of the child, but there is a need for more data before this approach can be recommended. Sleep in infants less than 6 mo old should be scored as active, quiet, or indeterminate (141, 142).

Although detailed sleep staging beyond simply identifying the occurrence of REM periods is not always necessary, arousals should be scored because these may be important consequences of abnormal breathing events during sleep. Quantifying mini- or micro-arousals in children to obtain a measure of sleep disturbance has been recommended (164), but needs more study. New guidelines for scoring arousals on PSG have recently been published by the American Sleep Disorders Association (16 and a modification of this system for children has been proposed. Arousal should be classified as respiratory if it occurs at the end of a respiratory event (apnea or hypopnea), technician-induced, or spontaneous. The frequency of movement arousals terminating obstructive apnea in children is unclear, but there is

a suggestion that children can sometimes terminate obstructive apnea without a cortical arousal (145, 163, 166).

Another nonrespiratory variable involves measurement of cardiac rhythm. The presence of cardiac arrhythmias, including sinus bradycardia, and whether they are associated with respiratory disturbances should be noted and the number of such events tabulated.

Consensus Recommendations for Scoring

Until more experience is gained with children's abnormal breathing during sleep, the entire record should be scored by a qualified individual who know the unique characteristics of breathing during sleep in children of different ages. Scoring based on sampling techniques must be validated for children (167).

- Given the volume of data accumulated during PSG, the use of computer-based data acquisition and scoring is attractive. Computerized data acquisition appears to be reliable, and a paperless polysomnograph can meet the needs of pediatric laboratories. However, computerized scoring of respiratory events and sleep staging in children requires validation (168, 169).
- Respiratory variables that should be scored include the number, type, and duration of apneas; episodes of partial airway obstruction measured as bypopnea or hypoventilation, whether obstructive or central; and the frequency and duration of paradoxical inspiratory rib-cage movements and associated desaturation, apnea, hypopnea, or increased end-tidal CO_2 values.
- Normative data in children suggest that obstructive apnea is rare in normal children (98, 154, 170, 171). Thus obstructive apnea of any length should be scored. If an obstructive apnea index — the number of events per hour of total sleep time — or a combined apnea/hypopnea index is used, appropriate pediatric normal values must be employed. Nonsigh, nonmovement-associated central apnea > 20 s should be counted, regardless of associated desaturation or bradycardia. Shorter episodes should also be scored if, associated with desaturation > 4% or age-specific bradycardia.
- If end-tidal CO_2 data is available, the following measurements should be scored: the end-tidal CO_2 at sleep onset, the peak end-tidal CO_2, the duration of end-tidal CO_2 > 50 mm Hg expressed as a percent of total sleep time, and the changes in CO_2 associated with respiratory events. The end-tidal CO_2 values should be correlated with chest wall and abdominal recordings to identify the pathophysiology of the elevated CO_2. The amount of time during which a technically adequate end-tidal CO_2 tracing was not obtainable should be recorded in order to avoid underestimating the total sleep time spent with values > 50 mm Hg.
- Maximum and minimum Sao_2, the number of desaturations ≥ 4%, and the percent of total sleep time spent with Sao_2 < 95%, 90%, 85%, etc., should be scored. Changes in Sao_2 values should be correlated with respiratory events.
- Snoring should be noted as present or absent. There are currently no standardized scoring systems for snoring in pediatric patients. Rating or grading the snoring as mild, moderate, or severe may be useful for an individual but, until the ratings are standardized, should be left to the discretion of each laboratory.
- Respiratory data should be subdivided according to sleeping position (prone, side, or supine) when there is an important difference between positions.
- The presence and type of arrhythmias and whether they are correlated with respiratory events should be noted. Epoch-by-epoch scoring of heart rate is not necessary for routine pediatric PSG.
- There was a consensus that 30-s epochs were adequate for scoring a PSG evaluating respiration, but a minority view held that the epoch length should vary according to the indication for the study. More data are needed before establishing an epoch length recommendation.
- The following sleep variables should be collected: total sleep time, sleep efficiency, distribution of sleep stages as percent of total sleep time, sleep latency, number of arousals, body movements, body position, and sleep behavior (parasomnias).
- The criteria of Rechtschaffen and Kales (139) can be used to stage sleep for children 6 mo and older. For most clinical indications, identification of active and quiet sleep is adequate for infants under 6 mo (141, 142).
- Complete sleep staging may not be necessary for most cardiopulmonary studies, unless there are specific questions regarding the impact of the sleep stages on the breathing disturbance. At a minimum, sleep should be divided into REM and non-REM. If formal sleep staging is performed, the following additional parameters should be scored: non-REM stages 1, 2, 3, and 4 and REM time expressed as percentages of total sleep time.
- A statement summarizing body movements during sleep was believed sufficient for pediatric polysomnography. Body position should be recorded to determine if the child slept in his or her "usual" sleeping position and if respiratory abnormalities are related to body position. Position should be recorded either by video or observation by the technician.
- Arousals as defined in the Sleep Disorders Atlas Task Force of the American Sleep Disorders Association (165) should be noted and correlated with respiratory abnormalities. Spontaneous nonrespiratory arousal should also be noted as a possible marker of sleep disturbance (145). Arousals caused by the activity of the technician need not be counted.
- Videotaping children during sleep may be used to help distinguish sleep from wakefulness, to help determine the cause of arousals, and to document body position and snoring (45, 46, 145). The tape can be useful for interpreting unusual behavioral events and assessing respiratory effort during sleep.

Reporting the Results

Quantitative data summarizing results of the study should be reported in a standardized format similar to that used for adults (5, 72), as follows:

1. Patient identification — age, sex, race, height, weight, indication for study, other medical conditions, and foods or medications that may affect study results.
2. Techniques used and variables measured.
3. The accompanying caregiver's opinion whether the child's sleep and breathing patterns in the laboratory were representative of the child's sleep at home.
4. The presence and quality of snoring during the study.
5. Sleep staging parameters, if staging is used: total sleep time, sleep latency, sleep efficiency, body movements, awakenings, time awake after sleep onset, number of arousals and their association with respiratory events, and any noteworthy sleep behavior. The presence and number of REM periods and the duration of all sleep stages, expressed as a percent of total sleep time, should be recorded. If complete sleep staging is not performed, the number of REM periods and the percentages of the total sleep time spent in REM and non-REM sleep should be recorded.
6. Respiratory rate during non-REM sleep. Apnea and hypopnea or hypoventilation should be reported by type, total number, average duration, longest event of each type, lowest Sao_2, and heart rate associated with the event.
7. Oxygen saturation as maximum Sao_2; minimum Sao_2; per-

cent total sleep time spent with $Sa_{O_2} < 95\%$, 90%, 85%, etc.; and the association between episodes of desaturation and apnea, hypopnea, or hypoventilation noted during the study. The relationship of desaturation episodes to sleep stage and body position should be noted.

8. End-tidal CO_2 data reported as time spent above 50 and 60 mm Hg expressed as a percent of total sleep time that Co_2 data was available. Correlation of elevated Co_2 with respiratory events should be included in the report.
9. Cardiac arrhythmias (including sinus bradycardia) and their relationship to respiratory abnormalities.
10. If therapy (oxygen, CPAP, or BiPAP®) was administered during the study, the Sa_{O_2} on room air and at each level of oxygen supplementation, pressure, or ventilator rate should be recorded. Effects of the therapeutic intervention of $PETCO_2$ or sleep quality should be reported if pertinent.
11. Technician's comments.
12. Interpretation. The individual responsible for the final interpretation of PSG performed on children to evaluate respiratory function during sleep should have expertise in sleep disorders in children, understand developmental cardiorespiratory physiology, and be certified in the medical evaluation of pediatric patients.

Before evaluating the study, the interpreter must ascertain if the child's sleep and/or breathing during the night of the PSG was representative for that child at home. The medical history should also be reviewed to determine if the results answer the question that prompted the study.

To determine if the results of a sleep study are abnormal, the interpreter should know the baseline awake values for respiratory rate, Sa_{O_2}, and end-tidal CO_2 to determine if sleep itself is associated with changes in these values. Cardiac arrhythmia, adequacy of sleep, and the degree of sleep disruption should also be factored into the decision-making process. Sleep stages should be interpreted in light of published age-appropriate normative values (139–142). Unfortunately, normal values for variables such as the number of arousals or body movements, movement time, or other indicators of disturbed sleep are not available for children. More data are needed.

Some broad guidelines can be used to determine abnormality for the respiratory events:

Central apnea $\geq$ 20 s has been shown to occur in normal children and adolescents, especially following a sigh or movement (156, 157, 173, 174). The clinical significance of these episodes must be interpreted in light of the indications for the study. If they are not associated with any physiologic abnormalities (bradycardia, hypoxemia), they may be considered within the broad range or normal values. An event of any duration associated with a $\geq$ 4% drop in SaO2 should be considered abnormal if the frequency of these events exceeds three per hour (175) or if it is associated with a $\geq$ 25% change in heart rate.

After the first month of life, normal infants and children do not exhibit more than one obstructive apnea per hour of sleep time (99, 170, 171). Studies of infants 1–6 mo old (174) and children 1-17 yr (153, 173) found obstructive apnea indices of 0.04 $\pm$ 0.13 apneas/h and 0.1 $\pm$ 0.5 apneas/h, respectively. These observations suggest that, in children, obstructive apnea of any duration exceeding 1 apnea/h should be noted and considered abnormal. Nonetheless, the clinical significance of isolated or infrequent obstructive events without desaturation or arousal is yet to be determined.

Oxygenation status must be interpreted in light of changes in saturation from both the awake values and the stable baseline reading preceeding any respiratory event. Some normal children have brief desaturations of $\geq$ 4% occurring at a rate of $\leq$ 3 events/h (175). Desaturation episodes to < 90% are rare, and their frequency decreases with age (119). Any change in saturation must be interpreted in light of the preceeding baseline value. Sustained saturation values < 90% are abnormal.

Partial airway obstruction associated with paradoxical inspiratory rib-cage movements, labored breathing, disturbed sleep, and heavy sweating, yet without desaturation, has been linked to excessive daytime sleepiness and behavioral disturbances (122, 128). This breathing pattern has been associated with significant elevations in esophageal pressure swings during tidal breathing (176). This pattern of breathing may also be identified by reviewing both the audiovideo recording and respiratory channels of the PSG. Its presence should be considered abnormal (176).

Hypopneic events ($\geq$ 50% reduction in the RIP sum signal, and/or in the thermistor signal associated with arousal, and/or desaturation of > 4% or sustained values < 90%) should also be considered abnormal (128, 177).

Hypoventilation events indicated by elevation of end-tidal CO_2 to > 53 mm Hg or > 50 mm Hg for > 8% of total sleep time, or a change in end-tidal CO_2 of > 13 mm Hg from baseline, indicates alveolar hypoventilation (154).

The etiology of the desaturation, apnea, hypopnea, or hypoventilation events can be determined by correlating the changes in airflow or end-tidal CO_2 with the respiratory patterns obtained from the chest wall and abdominal recordings. For example, reduced airflow or desaturation associated with paradoxical inspiratory rib-cage movement and/or snoring suggests partial airway obstruction.

Arousals terminating respiratory events indicate a clinically significant event (145, 177). Although the absence of a cortical (EEG) arousal following a severe episode of desaturation or an extended period of airway obstruction is not uncommon in children (163, 166), lack or an arousal (EEG, or movement) to an episode of sustained airway obstruction, hypoventilation, or hypoxemia suggests a compromise in the child's respiratory defenses and should be considered abnormal (163, 177).

Deviations from normal sleep patterns (139–142) and frequent spontaneous arousals must be interpreted cautiously in light of the impact on sleep quality of the first night in the sleep lab (107, 108). However, disturbed, restless sleep can mark a significant disruption of respiration during sleep (6,7).

RESEARCH RECOMMENDATIONS

The committee found that the following areas needed additional investigation to refine current recommendations.

- Age-specific normal values for respiratory events, Sa_{O_2}, end-tidal CO_2, sleep efficiency, arousals, and sleep disruption need to be generated. Additionally, more data are needed to define thresholds of clinical significance for PSG parameters in children.
- Which PSG abnormalities (number of respiratory events, cumulative hypercapnia, severity of desaturation, and degree of sleep disruption) in infants and children with OSAS correlate with morbidity? How do severity and duration of events such as desaturation, airway obstruction, and CO_2 interact to produce morbidity?
- What are the neuropsychological and cognitive consequences of OSAS in children? Are these a consequence of disturbed sleep, chronic/recurrent hypoxemia during sleep, or both?
- What is the natural history of OSAS in children? What is the link between OSAS in children and in adults?
- What are the risks that a child who has been treated for OSAS will develop recurrent symptoms of OSAS as an older child or adult?

- What is the sensitivity and reproducibility of a single night's Sao_2 recording or PSG? Do they change with age, puberty, or minor illness?
- What constitutes a clinically significant episode or period of desaturation in an infant, child, and adolescent?
- What is the most accurate and meaningful way of documenting desaturation during sleep?
- What is the role of formal sleep staging in PSG performed to assess cardiorespiratory function?
- What is the role of end-tidal CO_2 recording? What are age-appropriate reference values?
- How well do qualitative airflow measures such as thermistors and changes in ventilation, or semiquantitative measurements such as RIP, track changes in gas exchange?
- What are indications and limitations of unattended PSG in the home or hospital?
- What is the prevalence of OSAS in infants, children, and adolescents?
- What severity of illness or what degree of PSG warrants therapy?
- What is the role of nasal CPAP or BiPAP® in treating OSAS in children?
- What medical therapies are useful in treating obstructive sleep apnea in children?
- What is the best method of documenting partial airway obstruction? What is the role of esophageal pressure measurements in documenting partial airway obstruction?

This statement was prepared by an ad hoc Committee of the Scientific Assembly on Pediatrics. Members of the Committee were:

GERALD M. LOUGHLIN, *Cochair*
ROBERT T. BROUILLETTE, *Cochair*
LEE J. BROOKS
JOHN L. CARROLL
BRADLEY E. CHIPPS
SANDRA J. ENGLAND
RICHARD FERBER
NALTON F. FERRARO
CLAUDE GAULTIER
DEBORAH C. GIVAN
GABRIEL G. HADDAD
BRUCE R. MADDERN
GEORGE B. MALLORY
IAN T. NATHANSON
CAROL L. ROSEN
BRADLEY T. THACH
SALLY L. DAVIDSON WARD
DEBRA E. WEESE-MAYER
MARY ELLEN WOHL

References

1. Gaultier, C. 1985. Breathing and sleep during growth: physiology and pathology. *Bull. Eur. Physiopathol. Respir.* 21:55–112.
2. Phillipson, E. A. 1978. Respiratory adaptations in sleep. *Annu. Rev. Physiol.* 40:669–675.
3. Gaultier, C., and J. P. Praud., A. Clément, A. M. D'Allest, M. Khiata, G. Tournier and F. Girard. 1985. Respiration during sleep in children with COPD. *Chest* 87:168–173.
4. Gaultier, C. 1992. Clinical and therapeutic aspects of obstructive sleep apnea syndrome in infants and children. *Sleep* 15:536–538.
5. American Thoracic Society. 1989. Consensus conference on indications and standards for cardiopulmonary sleep studies. *Am. Rev. Respir. Dis.* 139:559–568.
6. Guilleminault, C., R. Korobkin, and R. Winkle. 1981. A review of 50 children with obstructive sleep apnea syndrome. *Lung* 159:275–287.
7. Brouillette, R., S. Fernbach, and C. Hunt. 1982. Obstructive sleep apnea in infants and children. *J. Pediatr.* 100:31–40.
8. Gorbo, G. M., F. Fuciarelli, A. Foresi, and F. Debenedetto. 1989. Snoring in children: association with respiratory symptoms and passive smoking. *Br. Med. J.* 299:1491–1494.
9. Ali, N. J., D. Pitson, and J. R. Stradling. 1993. The prevalence of snoring, sleep disturbance, and sleep-related breathing disorders and their relation to daytime sleepiness in 4–5-year-old children. *Arch. Dis. Child* 68:360–363.
10. Teculescu, D. B., I. Caillier, P. Perrin, E. Rebstock, and A. Rauch. 1992. Snoring in French preschool children. *Pediatr. Pulmonol.* 13:239–244.
11. Weissbluth, M., A. Davis, J. Poncher, and J. Reiff. 1983. Signs of airway obstruction during sleep and behavioral, developmental and academic problems. *Dev. Behav. Pediatr.* 4:119–121.
12. Everett, A., W. Kock, and F. Saulsbury. 1987. Failure to thrive due to obstructive sleep apnea. *Clin. Pediatr.* 26:90–92.
13. Menashe, V. D., F. Farrehi, and M. Miller. 1965. Hypoventilation and cor pulmonale due to chronic upper airway obstruction. *J. Pediatr.* 67:198–203.
14. Ross, R. D., S. R. Daniels, J. M. H. Loggie, R. A. Meyer, and E. T. Ballard. 1987. Sleep apnea–associated hypertension and reversible left ventricular hypertrophy. *J. Pediatr.* 111:253–255.
15. Frank, Y., R. E. Kravath, C. P. Pollack, and E. D. Weitzman. 1983. Obstructive sleep apnea and its therapy: clinical and polysomnographic manifestations. *Pediatrics* 71:737–742.
16. Weider, D. J., and P. J. Hauri. 1985. Nocturnal enuresis in children with upper airway obstruction. *Int. J. Pediatr. Otorhinolaryngol* 9:173–182.
17. Kunzman, L. A., T. G. Keens, and S. L. Davidson Ward. 1990. Incidence of systemic hypertension in children with obstructive sleep apnea syndrome. *Am. Rev. Respir. Dis.* 141:A808.
18. Seid, A. B., P. J. Martin, S. M. Pransky, and D. B. Kearns. 1990. Surgical therapy of obstructive sleep apnea in children with severe mental deficiency. *Laryngoscope* 100:507–510.
19. Ellis, E. R., P. T. P. Bye, J. W. Bruderer, and C. E. Sullivan. 1987. Treatment of respiratory failure during sleep in patients with neuromuscular disease. *Am. Rev. Respir. Dis.* 135:148–152.
20. Mallory, G. B., Jr., D. H. Fiser, and R. Jackson. 1989. Sleep-associated breathing disorders in morbidly obese children and adolescents. *J. Pediatr.* 115:892–897.
21. Schafer, M. E. 1982. Upper airway obstruction and sleep disorders in children with craniofacial anomalies. *Clin. Plast. Surg.* 9:555–567.
22. Handler, S. D. 1985. Upper airway obstruction in craniofacial anomalies: diagnosis and management. *Birth Defects* 21:15–31.
23. Loughlin, G. M., J. W. Wynne, and B. E. Victoria. 1981. Sleep apnea as a possible cause of pulmonary hypertension in Down syndrome. *J. Pediatr.* 98:435–437.
24. Marcus, C. L., D. M. Crockett, and S. L. Davidson-Ward. 1990. Evaluation of epiglottoplasty as treatment for severe laryngomalacia. *J. Pediatr.* 117:706–710.
25. Shprintzen, R. J. 1988. Pharyngeal flap surgery and the pediatric upper airway. *Int. Anesthesiol. Clin.* 26:79–88.
26. Sammuels, M. P., V. A. Stebbens, S. C. Davies, E. Picton-Jones, and D. P. Southall, 1992. Sleep-related upper airway obstruction and hypoxemia in sickle cell disease. *Arch Dis. Child.* 67:925–929.
27. Dure, L. S., A. K. Percy, W. R. Cheek and J. P. Laurent. 1989. Chiari Type 1 malformations in children. *J. Pediatr.* 115:573–576.
28. Shapiro, J., M. Strome, and A. C. Crocker. 1985. Airway obstruction and sleep apnea in Hurler and Hunter syndromes. *Ann. Otol. Rhinol. Laryngol.* 94:458–461.
29. McNicholas, W. T., S. Tarlo, P. Cole, N. Zamel, R. Rutherford, D. Griffin, and E. A. Phillipson. 1982. Obstructive apneas during sleep in patients with seasonal allergic rhinitis. *Am. Rev. Respir. Dis.* 126:625–628.
30. Rosen, C. L., L. D'Andrea, and G. G. Haddad. 1992. Adult criteria for obstructive sleep apnea do not identify children with serious obstruction. *Am. Rev. Respir. Dis.* 146:1231–1234.
31. Carroll, J. L., and G. M. Loughlin. 1992. Diagnostic criteria for obstructive sleep apnea syndrome in children. *Pediatr. Pulmonol.* 14: 71–74.
32. Diagnostic Classification Steering Committee and M. J. C. Thorpy. 1990. International Classification of Sleep Disorders: Diagnostic and Coding Manual. American Association of Sleep Disorders Association, Rochester, Minnesota.
33. Carroll, J. L., S. A. McColley, C. L. Marcus, S. Curtis, P. Pyzik, and G. M. Loughlin, 1995. Inability of clinical history to distinguish primary snoring from obstructive sleep apnea syndrome in children. *Chest* 108:610–619.
34. Carroll, J. L., S. A. McColley, C. L. Marcus, S. Curtis, P. Pyzik,

and G. M. Loughlin. 1992. Can childhood obstructive sleep apnea be diagnosed by a clinical symptom score (subtract)? *Am. Rev. Respir.. Dis.* 145:A179

Brouillette, R., D. Hanson, R. David, L. Klemka, A Szatkowski, S. Fernback, and C. Hunt. 1984. A diagnostic approach tc suspected obstructive sleep apnea in children. *J. Pediatr.* 105:10-14

36. McColley, S A., J. L. Carroll, M. M. April, R. N Naclerio, and G. M. Loughlin. 1992. Respiratory compromise after adenotsillectomy in children with obstructive sleep apnea *Arch. Otolaryngol. Head Neck Surg.* 118:940-943.
37. Rosen, G. M., R P. Muckie, M. W. Mahowald, G S. Goding, and C. Ullevig. 1994. Postoperative respiratory compromise in children with obstructive sleep apnea syndrome; can it be anticipated? *Pediatrics* 93:784-788.
38. Silvestri, J. M., D. E. Weese-Mayer, M. Bass, A. Morrow-Kenny, and S. Hauptman. 1993. Polysomnography in obese children with a history of sleep-associated breathing disorders. *Pediatr. Pulmonol.* 16:124-129.
39. Maddern, B. R., H. T. Reed, K. Ohene-Frempong, and R. C. Beckerman. 1989. Obstructive sleep apnea syndrome in sickle cell disease. *Ann. Otol. Rhinol. Laryngol.* 98:174-178.
40. Craft, J. A., E. Alessandrini, L. B. Kenney, B. Klein, G. Bray, N. L. C. Lwban, R. Meek, and V. M. Nadkarni. 1994. Comparison of oxygenation measurements in pediatric patients during sickle cell crises. *J. Pediatr.* 124:93-95.
41. Suen, J. S., J. E. Arnold, and L. J. Brooks. 1995. Adenotonsillectomy for the treatment of obstructive apnea in children. *Arch. Otolarynol Head Neck Surg* 121:525-530.
42. Carskadon, M. A., W. C. Dement, M. M. Mittler, T. Roth, P. R. Westbrook, and S. Keeran. 1986. Guidelines for the multiple sleep latency test (MSLT): a standard measure of sleepiness. *Sleep* 9:519-524.
43. Clarskadon, M. A., K. Harvey, P. Duke, T. F. Anders, I. F. Litt, and W. C. Dement, 1980. Pubertal changes in daytime sleepiness. *Sleep* 2:453-460.
44. Feinberg, I. 1974. Changes in the sleep cycle pattern with age. *J. Psychiatr. Res.* 10:283-306.
. Jaeob, S. V., A. Morielli, F. M. Ducharme, M. D. Schloss, and R. T. Brouillette. 1995. Home testing for pediatric obstructive sleep apnea secondary to adenotonsillar hypertrophy. *Pediatr Pulmonol* 20:241-252.
46. Morielli, A., F. M. Ducharme, and R. T. Brouillette. 1993. Sleep and wakefulnes can be distinguished in children by videotape and cardiorespiratory recordings (abstract). *Am. Rev. Respir. Dis.* 147:A762.
47. O'Bradovich, J. M., and R. B. Mellins, 1985. Bronchopulmonary dysplasia: unresolved neonatal aeute lung innjury. *Am. Rev. Respir. Dis.* 132:694-709.
48. Northway, W. J., Jr., R. B. Moss, K. B. Carlisle, B. R. Parker, R. L. Popp, P. T. Pitlick, I. Eichler, R. L. Lamm, and B. W. Brown, Jr. 1990. Late pulmonary sequalae of bronchopulmonary dysplasia. *N. Engl. J. Med.* 323:1793-1799.
49. Garg, M., S. I. Kurzner, D. B. Bautista and T. B. Keens. 1988. Clinically unsuspected hypoxia during sleep and feeding in infants with bronchopulmonary dysplasia. *Pediatrics* 81:635-642.
50. Loughlin, G. M., R. P. Allen, et al. 1987. Sleep-related hypoxemia in children with bronchopulmonary dysplasia (BPD) and adequate oxygen saturation awake, abstracted. *Sleep* (Res.) 16:486.
51. Singer, L., R. J. Martin, S. W. Hawkins, L. J. Benson-Szekely, T. S. Yamashita and W. A. Carlos. 1991. Oxygen desaturation complicates feeding in infants with bronchopulmonary dysplasia after discharge. *Pediatrics* 90:380-384.
52. Trang, T. T. H., J. P. Praud, M. Pagani, M. Goldman, and C. Gaultier. 1993. Sleep disordered respiration in infants and children with severe bronchopulmonary dysplasia (abstract). *Am. Rev. Respir. Dis.* 147:A343.
53. Goldman, M., M. Pagnini, T. T. H. Trang, J. P. Praud, M. Sartene, and C. Gaultier. 1993. Asynchronous chest wall movements during NREM and REM in children with bronchopulmonary dysplasia. *Am. Rev. Respir. Dis.* 147:1175-1184.
54. Rome, E. S., M. J. Miller, Goldthwait, I. O. Osorio, A. A. Farnaroff and R. J. Martin. 1987. Effect of sleep state on chest wall movements and gas exchange in infants with resolving bronchopulmonary dysplasia. *Pediatr. Pulmonol.* 3:259-263.
55. Rome, E. S., E. K. Stork, W. A. Carlo and R. J. Martin. 1984. Limitations of trancutaneous Po_2, Pco_2 monitoring in infants with bronchopulmonary dysplasia. *Pediatric* 74:217-220.
56. Lafontaine, V., F. M. Ducharme, and R. T. Brouillette. 1994. Can we rely on pulse oximetry desaturation events (abstract)? *Am. Rev. Respir. Dis.* 149:69A.
57. Muller, N. L., P. W. Francis, D. Gurwitz, J. Levinson, and A. C. Bryan. 1980. Mechanism of hemoglobin desaturation during rapid-eye-movement sleep in normal subjects and in patients with cystic fibrosis. *Am. Rev. Respir. Dis.* 121:463-469.
58. Stokes, D. C., J. T. McBride, M. A. Wall, G. Erba and D. J. Streder. 1980. Sleep hypoxemia in young adults with cystic fibrosis. *Am. J. Dis. Child* 134:741-743.
59. Tepper, R. S., J. B. Skatrud, and J. A. Dempisey, 1983. Ventilation and oxygenation changes during sleep in cystic fibrosis. *Chest* 84:388-393.
60. Versteegh, F. G. A., J. M. Bogaard, J. W. Raatgever, H. Stam, H. J. Neijens and K. F. Kerrebijn. 1990. Relationship between airway obstruction, desaturation during exercise and nocturnal hypoxemia in cystic fibrosis patients. *Eur. Respir. J.* 3:68-73.
61. Montgomery, M., W. Wiebicke, H. Bibi, R. D. Pagtakan and H. Pasterkamp. 1989. Home measurement of oxygen saturation during sleep in patients with cystic fibrosis. *Pediatr. Pulmonol.* 7:29-34.
62. Bowton, D. L., P. F. Scuderi, L. Harris, and E. F. Haponick. 1991. Pulse oximetry monitoring outside the intensive care unit: progress or problem? *Ann. Intern Med.* 115:450-454.
63. Clark, T. J. H., and M. R. Hetzel. 1977. Diurnal variation of asthma *Br. J. Dis. Chest* 71:87-92.
64. Busse, W. W. 1988. Pathogenesis and pathophysiology of nocturnal asthma. *Am. J. Med.* 85(Supple. 1B):24-29.
65. Martin, R. J., L. C. Cicutto, and R. D. Ballard. 1990. Factors related tc the nocturnal worsening of asthma. *Am. Rev. Respir. Dis.* 141:33-38.
66. Chipps, B. E., H. Mak, K. C. Schuberth, J. H. Talamo and H. A. Menkes. 1980. Nocturnal oxygen saturation in normal and asthmatic children. *Pediatric* 65:1157-1159.
67. Smith, T. F., and D. W. Hudgel. 1980. Arterial oxygen desaturation during sleep in children with asthma and its relation to airway obstruction and ventilatory drive. *Pediatrics* 66:746-751.
68. Smith, P. E. M., P. M. A. Calverley, and R. H. T. Edwards. 1988. Hypoxemia during sleep in Duchenne museular dystrophy. *Am. Rev. Respir. Dis.* 121:281-289.
69. Begin, R., M. A. Bureau, L. Lupien, and B. Lemieux. 1980. Control and modulation of respiration in Steinert's myotinic dystrophy. *Am Rev. Respir. Dis.* 121:281-289.
70. Gilgoff, I. S., E. Kahlstrom, E. MacLaughlin, and T. G. Keens. 1989. Long-term ventilatory support in spinal muscular atrophy. *J. Pediatr.* 115:904-909.
71. Skatrud, J., C. Iber, W. McHugh, H. Rasmussen, and D. Nichols. 1980. Determinants of hypoventilation during wakefulness and sleep in diaphragmatic paralysis. *Am. Rev. Respir. Dis.* 121:587-593.
72. Bye, P. T. P., E. R. Ellis, F. G. Issa, P. M. Donnelly, and C. E. Sullivan. 1990. Respiratory failure and sleep in neuromuscular disease. *Thorax* 45:241-247.
73. Eichacker, P. Q., A. Spiro, M. Sherman, E. Lazar, J. Reichel, and F. Dodick. 1988. Respiratory muscle dysfunction in herediatry monitor sensory neuropathy, type I. *Arch. Intern. Med.* 148:1739-1740.
74. Riley, D. J., T V. Santiago, D. P. Daniele, B. Schall, and N. H. Edelman. 1977. Blunted respiratory drive in cogenital myopathy. *Am. J. Med.* 63:459-446.
75. O'Leary, J., R. King, M. Liblanc, R. Moss, M. Liebhaber, and N. Lewiston, 1979. Cuirass ventilation in childhood neuromuscular disease. *J. Pediatr.* 94:419-421.
76. Maavan, C. H., C. Springer, Y. Armon, E. Bar-Yishay, Y. Shapira, and S. Godfrey. 1986. Nemaline myopathy as a cause of sleep hypoventilation. *Pediatrics.* 77:390-395.
77. Hyckmatt, J. Z., L. Loh, and V. Dubowitz. 1989. Nocturnal hypoventilation in children with nonprogressive neuromuscular disease. *Pediatrics* 83:250-255.
78. Rideau, Y., L. W. Jankowski, and J. Grellet. 1981. Respiratory function in the muscular dystrophies. *Muscle Nerve* 4:155-164.
79. Baydur, A., I. Gilgoff, and W. Prentice. 1985. Guidelines for assisted ventilation in Duchenne's muscular dystrophy. *Am. Rev. Respir. Dis.* 131 (suppl):A268.
80. Martinez, B. A., and B. D. Lake. 1987. Childhood nemaline myopathy: a review of elinical presentation in relation to prognosis. *Dev. Med. Child Neurol.* 29:815-820.
81. Alberts, M. J., and A. D. Roses. 1989. Myotonic muscular dystrophy. *Neurol. Clin.* 7:1-8.
82. Manni, R., A. Ottolini, I. Cerveri, C. Bruschi, M. C. Zoia, G. Lanzi, and A. Tartara. 1989. Breathing patterns and $HbSaO_2$ changes during nocturnal sleep in patients with Duchennes muscular dystrophy. *J. Neurol.* 236:361-394.
83. Serisier, D. E., F. L. Mastaglia, and G. J. Gibson. 1982. Respiratory

muscle function and ventilatory control in patients with neurone disease and in patients with myotonic dystrophy. *Q. J. Med* 202:205-226.

84. Ellis, E. R., P. T. P. Bye, J. W. Bruderer, and C. E. Sullivan. 1987. Treatment of respiratory failure during sleep in patients with neuromuscular disease. *Am. Rev. Respir. Dis.* 135:148-152.
85. Newsom-Davis, J., M. GOldman, L. Loh, and M. Casson. 1976. Diaphragm function and alveolar hypoventilation. *Q. J. Med.* 45:87-100, 148-152.
86. Goldstein, R. S., N. Molotiu, R. Skrastins, S. Long, J. DeRoste, M. Contreras, J. Poplin, R. Rutherford, and E. A. Phillipson. 1987. Reversal of sleep-induced hypoventilation and chronic respiratory failure by nocturnal negative pressure ventilation in patients with restrictive ventilatory impairment. *Am. Rev. Respir. Dis.* 135:1029-1055.
87. Aldrich, M. S. 1990. Neurologic aspects of sleep apnea and related respiratory disturbances. *Otolarynol. Clin. North Am.* 23:761-769.
88. Mallory, G. B., and P. C. Stillwell. 1991. The ventilator dependent child: issues in diagnosis and management. *Arch Phys. Med. Rehabil.* 72:43-55.
89. Weese-Mayer, D. E., J. M. Silvestri, L. J. Menzies, A. S. Morrow-Kenny, C. E. Hunt, and S. A. Hauptman. 1992. Congenital central hypoventilation syndrome: diagnosis, management, and long-term outcome in thirty-two children. *J. Pediatr.* 120:381-387.
90. Weese-Mayer, D. E., C. E. Hunt, and R. T. Brouillette. 1992. Alveolar hypoventilation syndromes. *In* R. C. Becerman, R. T. Brouillette, and C. E. Hunt, editors. Respiratory Control Disorders in Infants and Children. Williams and Wilkins, Baltimore.
91. Silvestri, J. M., D. E. Weese-Mayer, and M. N. Nelson. 1992. Neuropsychologic abnormalities in children with congenital central hypoventilation syndrome. *J. Pediatr.* 123:388-393.
92. Weese-Mayer, D. E., C. E. Hunt, R. T. Brouillete, and J. M. Silvestri. 1992. Diaphragm pacing in infants and children. *J. Pediatr.* 120:1-8.
93. Martin, R. M., M. J. Miller, and W. A. Carlo. 1986. Pathogenesis of apena in ptreterm infants. *J. Pediatr.* 109:733-741.
94. Henderson-Smart, D. J. 1981. The effect of gestational age on the incidence and duration of recurrent apnea in newborn babies. *Aust. Paediatr. J.* 17:273-276.
95. Milner, A. D., A. W. Boon, R. A. Saunders, and J. E. Hopkin. 1980. Upper airway obstruction and apnea in preterm babies. *Arch. Dis. Child.* 55:22-25.
96. National Institutes of Health, Consensus Development Conference on Infantile Apnea and Home Monitoring. 1986. U. S. Department of Health and Human Services, Washington, DC. NIH Publication No. 87-2905.
97. Series, F., Y. Cormier, and J. La Forge. 1991. Validity of diurnal sleep recordings in the diagnosis of sleep apnea syndrome. *Am. Rev. Respir. Dis.* 143:947-949.
98. Silvestri, R., C. Guilleminault, R. Coleman, T. Roth, and W. C. Dement. 1982. Nocturnal sleep versus daytime nap findings in patients with breathing abnormalities during sleep. *Sleep Res.* 11:174.
99. Marcus, C. L., T. G. Keens, and S. L. Ward. 1992. Comparison of nap and overnight polysomnography in children. *Pediatr. Pulmonol.* 13:16-21.
100. Canet, E., C. Gaultier, A. M. D'Allest, and M. Deban. 1989. Effects of sleep deprivation on respiratory events during sleep in healthy infants. *J. Appl. Physiol.* 128:984-986.
101. White, D. P., N. J. Douglas, C. K. Pickett, C. W. Zwillich and J. R. 1983. Sleep deprivation and the control of ventilation. *Am. Rev. Respir. Dis.* 128:984-986.
102. Leiter, J. C., S. L. Knuth, and D. Bartlett. 1985. The effect of sleep deprivation on activity of the genioglossus muscle. *Am. Rev. Respir. Dis.* 132: 1242-1245.
103. Hershenson, M., R. T. Brouillette, E. Olsen, and C. E. Hunt. 1984. The effect of chloral hydrate on genioglossus and diaphragmatic activity. *Pediatr. Res.* 18:516-519.
104. Biban, P., E. Baraldi, and A. Pettenazz. 1993. The adverse effect of chloral hydrate in choldren with OSA. *Pediatrics* 92:461-463.
105. Kales, A. J. D. Kales, R. M. Sly, *et al.* 1970. Sleep patterns of asthmatic children; all-night electrencephalographic studies. *J. Allergy.* 46:300-308.
106. Kales, J. D., A. Kales, A. Jacobsen, J. Po and J. Green. 1968. Baseline sleep and recall studies in children. *Psychophysiology* 4:391.
107. Agnew, H. W., W. B. Webb, and R. L. Williams. 1966. The first night effect: an EEG study of sleep. *Psychophysiology* 2:263-266.
108. Wittig, R. M., A. Romaker, F. J. Zorick, T. A. Roehrs, W. A. Conway and J. Roth. 1984. Night-to-night consistency of apneas during sleep. *Am. Rev. Respir. Dis.* 129:244-246.
109. Kryger, M. H. 1989. Monitoring respiratory and cardiac funtion. In M. H. Kryger, T. Roth, and W. C. Dement, editors. Principles and Practice of Sleep Medicine. Saunders, Philadelphia. 702-716.
110. Sivan, Y., S. D. Ward, T. Deakers, T. G. Keens, and C. J. Newth. 1991. Rib-cage to abdominal asynchrony in children undergoing polygraphic sleep studies. *Pediatr. Pulmonol.* 11:141-146.
111. Konno, K., and J. Mead. 1967. Measurement of the separate volume changes of the rib cage and abdomen during breathing. *J. Appl. Physiol.* 22:407-422.
112. Shapiro, A. and H. D. Cohen. 1965. The use of mercury capillary length gauges for the measurement of the volume of thoracic and diaphragmatic components of human respiration: a theoretical analysis and a practical method. *Trans. N. Y. Acad. Sci. Ser II.* 27:634-649.
113. Duffty, P., L. Spriet, M. H. Bryan, and A. C. Bryan. 1981. Respiratory inductive plethysmogrpahy (Respitrace™), and evaluation of its use in the infant. *Am. Rev. Respir. Dis.* 123:542-546.
114. Adams, J. A., I. A. Zabaleta, D. Stroh, A. Johnson and M. A. Sackner. 1993. Tidal volume measurements in newborns using respiratory inductive plethysmography. *Am. Rev. Respir. Dis.* 148:585-588.
115. Tabachnik, E., N. Muller, B. Toye, and H. Levison. 1981. Measurement of ventilation in children using the respiratory inductive plethysmograph. J. Pediatr. 99:895-899.
116. Warren, R. H., and S. H. Alderson. 1985. Calibration of computer-assisted (Respicomp) respiratory inductive plethysmography in newborns. *Am. Rev. Respir. Dis.* 136:416-419.
117. Mead, J., N. Peterson, and G. Grimby. 1967. Pulmonary ventilation measured from body surface measurements. *Science* 156:1386-1384.
118. Sharp, J. T., W. S. Druz, J. R. Foster, and M. S. Wicks. 1980. Use of the respiratory magnetometer in diagnosis and classification of sleep apnea. *Chest* 77.350-353.
119. Gaultier, C. L., J. P. Praud, E. Canet, F. Delaperchem and A. M. D'Allest. 1987. Paradoxical inward rib-cage motion during rapid-eye-movement sleep in infants and young children. *J. Dev. Physiol.* 9:391-397.
120. Guilleminault, C. 1987. Obstructive sleep apnea syndrome and its treatment in children; areas of agreement and controversy. *Pediatr Pulmonal* 3:429-436.
121. Strollo, P. J., and M. H. Sanders. 1993. Significance and treatment of nonapneic snoring. *Sleep* 16:403-408.
122. Guilleminault, C., R. Stoohs, A. Clerk, M. Cetel, and P. Maistros. 1993. A cause of excessive daytime sleepiness: the upper airway resistance syndrome. *Chest* 104:781-787.
123. Konno, A., T. Hoshino, and K. Togawa. 1980. Influence of upper airway obstyruction by enlarged tonsils and adenoids upon recurrent infection of the lower airway in childhood. *Laryngoscope* 90:1709-1716.
124. Dolfin, D., D. Duffy, D. Wilkes, S. England, and H. Bryan. 1983. Effects of facemask and pneumotachograph on breathing in sleeping infants. *Am. Rev. Respir. Dis.* 128:977-979.
125. Swedlow, D. B. 1986. Capnometry and capnography; the anesthesia disaster warning system. *Seminars in Anesthesia* 5:194-205.
126. Krumpe, P. E., and J. M. Cummiskey. 1980. Use of laryngeal sound recordings to monitor apnea. *Am. Rev. Respir. Dis.* 122:797-801.
127. Cummiskey, J. M., T. C. Williams, P. E. Krumpe, and C. Guilleminault. 1982. The detection and quantification of sleep apnea by tracheal sound recordings. *Am. Rev. Respir. Dis.* 126:221-224.
128. Gould, G. A., K. F. Whyte, G. B. Rhind, M. A. Airlie, J. R. Catterall, C. M. Shapiro and H. J. Douglas. 1988. The sleep hypopnea syndrome. *Am. Rev. Respir. Dis.* 137:895-898.
129. Cantineau, J. P., P. Escourrou, Startine, C. Gaultier, M. Goldman. 1992. Accuracy of respiratory inductive plethysmography during wakefulness and sleep in patients with obstructive sleep apnea. *Chest* 102:1145-1151.
130. Brouillette, R. T., A. S. Morrow, D. E. Weese-Mayer, and C. E. Hunt. 1987. Comparison of respiratory inductive plethysmography and thoracic impedance for apnea monitoring. *J. Pediatr.* 111:377-383.
131. Clark, J. S., B. Votteri, R. L. Anagno, P. Cheung, J. H. Eichorn, R. J. Fallot, W. E. Lee, C. J. L. Newth, H. Rotman, D. Y. Sue. 1992. Noninvasive assessment of blood gasses. *Am. Rev. Respir. Dis.* 145:220-232.
132. Morielli, A., D. Desjardins, and R. T. Bouillette. 1993. To assess hypoventilation during pediatric polysomnography both trancutaneous and end-tidal CO_2 should be measured. Am. Rev. Respir. Dis. 148:1599-1604.
133. Hansen, T. N., and W. H. Tooley. 1979. Skin surface carbon dioxide tension in sick infants. *Pediatrics* 64:942-945.
134. Yoshiya, I., Y. Shamida, and K. Tanaka. 1980. Spectrophotometric

monitoring of arterial oxygen saturation in the fingertip. *Med. Biol. Eng. Comput.* 18:27-32

135. Dear, P. R. F. 1987. Monitoring oxygen in the newborn, saturation or partial pressure? *Arch. Dis. Child.* 62:879-881.

136. Barrington, K. J., N. N. Diner, and C. A. Ryan. 1988. Evaluation of pulse oximetry as a continuous monitoring technique in the neonatal intensive care unit. *Crit. Care Med.* 16:1147-1153.

137. Huch, R., A. Huch, M. Albani, M. Gabriel, F. J. Schulte, H. Wolf, G. Rupprath, P. Emminch, V. Stechele, G. Duc and H. Bucher. 1976. Transcutaneous Po_2 monitoring in routine management of infants and children with cardiorespiratory problems. *Pediatrics* 57:681-690.

138. Herrell, N., R. J. Martin, M. Pultusker, M. Lough, and A. Faranoff. 1980. Optimal temperature for the measurement of transcutaneous carbon dioxide tension in the neonate. *J. Pediatr.* 97:114-117.

139. Rechtschaffen, A., and A. Kales. 1968. A Manual of Standardized Terminology, Techniques and Scoring Systems for Sleep Stages of Human Subjects. National Institutes of Health, Washington, DC.

140. Carskadon, M. A., and A. Rechtschaffen. 1989. Monitoring and staging human sleep. *In* M. H. Kryger, T. Roth, and W. C. Dement, editors. Principles and PRactice of Sleep Medicine, W. B. Saunders, Philadelphia. 665-683.

141. Anders, T., R. Emde, and A. Parmelee, editor. 1971. A Manual of Standardized Terminology, Techniques and Criteria for Scoring of States of Sleep and Wakefulness in Newborn Infants. UCLA Brain Information Service/Brain Research Institute, Los Angeles.

142. Navelet, Y., O. Benoit, and G. Gourard. 1958. Nocturnal sleep organization during the first months of life. *Electrencephalogr. Clin. Neurophysiol.* 10:371-375.

143. Walters, A. S., D. Picchietti, W. Hening, and A. Lazzarini. 1990. Variable expressivity in familial restless legs syndrome. *Arch. Neurol.* 47:1219-1220.

144. Jolley, S. G., J. J. Herbst, D. G. Johnson, M. E. Matlak and L. S. Book. 1981. Esophageal pH monitoring during sleep identifies children with respiratory symptoms from gastroesophageal reflux. *Gastroenterology* 80:1501-1506.

145. Mograss, M. A., F. M. Ducharme, and R. T. Brouillette. 1994. Moment/arousals: description, classification, and relationship to sleep apnea in children. *Am. J. Respir. Crit. Care Med.* 150:1690-1696.

146. Gleeson, K., C. Zwillich, K. Braier and D. P. White. 1986. Breathing route during sleep. *Am. Rev. Respir. Dis.* 134:115-120.

147. Warren, D. 1990. Effect of airway obstruction on facial growth, *Otolaryngol. Clin. North Am.* 23:699-712.

148. Jasper, H. H., Committee Chairman. 1958. The ten-twenty electrode system of the International Federation. *Electroencephalogr. Clin. Neurophysio.* 10:371-375.

149. Marsh, R. R., W. P. Postic, and P. S. Pasquariello. 1989. Reliability of sleep sonography in detecting upper airway obstruction in children. *Int. J. Pediatr. Otorhinolaryngol* 18:1-8.

150. Potsic, W. P., P. S. Pasquariello, C. C. Baranak, R. R. Marsh, and L. M. Miller. 1986. Relief of upper airway obstruction by adenotonsillectomy. *Otolaryngol. Head Neck Surg.* 94:476-480.

151. Konno, A., K. Togawa, and T. Hoshino. 1980. The effect of nasal obstruction in infancy and early childhood upon ventilation. *Laryngoscope* 90:699-707.

152. Agency for Health Care Policy and Research. Polysomnography and Sleep Disorders Centers, 1992. Health Technology Assessment Reports, 1991, No. 4. U. S. Department of Health and Human Services, Washington, DC. U. S. DHHS Publication No. 92-0027.

153. Moser, N. J., B. A. Phillips, D. T. R. Berry, and L. Harbison. 1994. What is hypopnea, anyway? *Chest* 105:426-428.

154. Marcus, C. L., K. J. Omlin, D. J. Basinski, S. L. Bailey, A. B. Rachal, W. S. Von Pechmann, T. G. Keens, and S. L. Davidson-Ward. 1992. Normal polysomnographic values for children and adolescents. *Am. Rev. Respir. Dis.* 146:1235-1239.

155. Curzi-Dascalova, L. 1978. Thoraco-abdominal respiratory correlations in infants: constancy and variability in different sleep states. *Early Hum. Dev.* 2:25-38.

156. Tabachnick, E., N. L. Muller, A. C. Bryan, and Levinson. 1981. Changes in ventilation and chest wall mechanics during sleep in normal adolescents. *J. Appl. Physiol.* 51:557-564.

157. Poets, C. F., V. A. Stebbins, M. P. Sammuels, and D. P. Southall. 1993. Oxygen saturation and breathing patterns in children. *Pediatrics* 92:686-690.

158. Stebbens, V. A., C. F. Poets, J. R. Alexander, W. A. Arrowsmith and D. P. Southall. 1991. Oxygen saturation and breathing patterns in infancy, 1: full-term infants in the second month of life. *Arch. Dis. Child* 66:569-573.

159. Poets, C. F., V. A. Stebbens, J. R. Alexander, W. A. Arrowsmith, S. A. W. Salfield, D. P. Southall. 1991. Oxygen saturation and breathing patterns in infancy, 2: preterm infants at discharge from special care. *Arch Dis. Child.* 66:574-578.

160. Chipps, G. A., H. Mak, K. C. Schuberth, J. H. Talomo, H. A. Menkes and M. S. Scherr. 1980. Nocturnal oxygen saturation in normal and asthmatic children. *Pediatrics* 65:1157-1160.

161. Muller, N. L., P. W. Francis, D. Gurwitz, H. Levinson and A. C. Bryan. 1980. Mechanism of hemoglobin desaturation during rapid-eye-movement sleep in normal subjects and in patients with cystic fibrosis. *Am. Rev. Respir. Dis.* 121:463-469.

162. Hudgel, D. W. 1992. Mechanisms of obstructive sleep apnea. *Chest* 101:541-549.

163. McGrath-Morrow, S. A., J. L. Carroll, S. A. McColley, P. Pyzik, and G. M. Loughlin. 1990. Termination of obstructive apnea in children is not associated with arousal (abstract). *Am. Rev. Respir. Dis.* 141:A195.

164. Guilleminault, C. 1987. Obstructive sleep apnea syndrome in children. *In* C. Guilleminault, editor. Sleep and Its Disorders in Children. Raven Press, New York. 213-224.

165. Sleep Disorders Atlas Task Force of the American Sleep Disorders Association. 1992. EEG arousals: scoring rules and examples. *Sleep* 2:174-183.

166. Praud, J. P., A. M. D'Allest, H. Nedelcoux, L. Curzi-Dascalova, C. Guilleminault, and C. Gaultier. Sleep-related abdominal muscle behavior during partial or complete obstructed breathing in prepubertal children. *Pediatr. Res.* 26:347-350.

167. Steyer, B. J., S. F. Quan, and W. J. Morgan. 1985. Polysomnography scoring for sleep apnea: use of a sampling method. *Am. Rev. Respir. Dis.* 131:592-595.

168. Nathanson, I. T., C. Ivey, T. Ricks, and S. Murphy. 1992. Comparison of manual, computer-assisted, and automated scoring of polysomnograms (PSGs). *Am. Rev. Respir. Dis.* 145:A173.

169. Norman, R. G., R. Zozula, J. A. Wasleben, and D. M. Rapoport. 1992. A likelihood-based computer approach to conventional scoring of sleep: validation in patients with sleep apnea. *Am. Rev. Respir. Dis.* 145:A174.

170. Gaultier, C. 1987. Respiratory adaptation during sleep from the neonatal period to adolescence. *In* C. Guilleminault, editor. Sleep and Its Disorders in Children. Raven Press Books, New York. 67-98.

171. Gaultier, C. 1995. Respiratory adaptation during sleep in infants and children. *Pediatr. Pulmonol.* 19:105-107.

172. Martin, R. J., A. J. Block, M. A. Cohn, W. A. Conway, D. W. Hudgel, A. C. Peter Powles, M. H. Sanders, and P. L. Smith. 1985. Indications and standards for cardiorespiratory sleep studies. *Sleep* 8:371-379.

173. Carskadon, M. A., K. Harvey, W. C. Dement, C. Guilleminault, F. B. Simons and T. F. Anders. 1978. Respiration during sleep in children. *West J. Med.* 128:477-481.

174. Guilhaume, A., and O. Benoit. 1976. Pauses respiratoires au cours du sommeil chez l'enfent normal: observations de trois cas pathologiques. *Rev. Electroencephalogr. Neurophysio. Clin.* 6:116-123.

175. Stradling, J. R., G. Thomas, A. R. H. Warley, P. Williams and A. Freeland. 1990. Effect of adenotonsillectomy on nocturnal hypoxaemia, sleep disturbance and symptoms in snoring children. *Lancet* 335:249-253.

176. Guilleminault, C., R. Winkle, R. Korobkin, and B. Simmons. 1982. Children and nocturnal snoring: evaluation of the effects of sleep-related respiratory resistive load and daytime functioning. *Eur. J. Pediatr.* 139:165-171.

177. Phillipson, E. A. 1978. Arousal: the forgotten response to repiratory stimuli. *Am. Rev. Respir. Dis.* 118:807-809.