Sleep-Related Breathing Disorders in Children

Family physicians need to be aware of the clinical features of various sleep-related breathing disorders, as they will be called on to evaluate symptoms that may either comprise a normal variation in human physiology or indicate serious disease.

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Relative hypoventilation is a normal event during sleep. The combination of a loss of upper airway tone, and a reduction in respiratory drive, results in a small, but significant, rise in arterial pCO_2 and a fall in arterial pO_2.[1] Consequently, any disorder or disease that adversely affects respiration will be exacerbated during sleep.

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There are a number of respiratory disorders that are specific to sleep. It has been more than 25 years since Guilleminault first described obstructive sleep apnea (OSA) in children.[2] In the intervening period, we have gained significant knowledge with respect to sleep-related respiratory disorders. At the same time, however, there remain significant gaps in our knowledge, particularly in terms of the natural history and epidemiology of the various disorders.[1] The lay literature contains several articles on the various disorders, with some creating significant parental anxiety (*i.e.*, with respect to sudden infant death syndrome [SIDS]).

The family physician, therefore, needs to be aware of the various disorders and their presentations to determine which symptoms can be treated

Summary

Sleep-Related Breathing Disorders in Children

- Obstructive sleep apnea (OSA) is the most common sleep-related respiratory disorder in both adults and children, with the basic pathophysiology being similar across all ages.
- The current "gold standard" for diagnosing OSA is polysomnography, where both respiratory status and sleep state are monitored on a continuous basis overnight.
- Central sleep apnea in adults is commonly defined as cessation of respiration for at least 10 seconds. It is common during the neonatal period, particularly in premature infants.
- There is a definite association between the incidence of sudden infant death syndrome (SIDS) and the child's sleeping position. A public education policy emphasizing the importance of infants sleeping in a supine position has produced a significant reduction in the overall incidence of SIDS.
- Diagnosis of an apparent life-threatening event is based upon clinical history. The primary concern is to identify underlying disorders that may have triggered the event.
- Congenital central alveolar hypoventilation can occur as a result of an isolated developmental defect of the brainstem (sometimes in association with other disorders) or as part of a more global central nervous system insult.
- There are several disorders that may be confused with sleep-related respiratory disorders. Any disorders causing daytime hypersomnolence may be inferred as due to sleep fragmentation secondary to a respiratory disorder, particularly if the child snores.

simply with parental reassurance, and which need more aggressive investigation.

Obstructive Sleep Apnea

OSA is the most common sleep-related respiratory disorder in both adults and children, with the basic pathophysiology being similar across all ages. Sleep-related hypotonia of the laryngeal muscles results in a loss of upper airway tone; hence, the upper airway collapses during inspiration. In most individuals the negative intratracheal pressure alone is sufficient to trigger arousal, resulting in sleep fragmentation without desaturation. If recurrent, or with blunted arousal, the obstructed breathing results in desaturation, with the associated hypercapnia (rather than hypoxemia) triggering arousal and a return of normal ventilation. Except in the most severe cases (*i.e.*, where sleep fragmentation has resulted in blunting of the respiratory drive), the hypercapnia is transient, with arterial CO_2 returning to normal immediately upon arousal. There are, however, significant differences between adults and children with respect to the presentation, clinical sequelae and treatment of OSA (Table 1).

In children, the incidence of OSA is the same in both sexes. It usually arises from anatomical causes (*i.e.*, adenoidal hypertrophy or facial anomalies) and tends to occur predominantly during rapid eye movement (REM) sleep. Adults tend to develop frank obstruction, with repeated arousal and fragmentation of all sleep states — the resulting loss of slow-wave sleep (stages 3 and 4 non-REM) leading to increased sleepiness and reduced alertness. In contrast, children tend to develop obstructive hypoventilation, with primarily REM fragmentation and an absence of overt arousals.[2] There is evidence that REM sleep is necessary for

Table 1

Obstructive Sleep Apnea Syndrome — Children *Versus* Adults

Clinical characteristics	Children	Adults
Peak age	Preschoolers	Elderly
Sex ratio	M = F	M > F 5-8:1 (pre-menopause)
Etiology	Adenotonsillar hypertrophy	Obesity
Weight	Range; failure to thrive, normal, occasionally obese	Obese
Daytime symptoms	Hyperactivity, developmental delay	Hypersomnolence, cognitive impairment, decreased vigilance
Treatment	Surgical: 1° adenoidectomy, occasionally CPAP	Medical: Weight loss/CPAP

CPAP = continuous positive airway pressure

the processing of new information, explaining why children (especially infants) have significantly greater REM sleep requirements than adults. In children, OSA is frequently associated with behavioral changes and learning disorders rather than hypersomnolence.[2,3] Obstructive apnea has been described in infants, primarily those with facial anomalies, such as Pierre Robin sequence. It has, however, been implicated in SIDS as well.

Clinical Diagnosis

OSA rarely occurs in the absence of snoring. Approximately 5% to 6% of normal children, however, snore. The diagnostic dilemma is to separate the approximately 25% of children with snoring that have significant OSA from the 75% who have uncomplicated snoring, but no apparent pathophysiologic sequelae. There *are* symptoms and signs that are indicative of the presence of significant OSA (Table 2).[4] In the absence of laboratory diagnosis, the presence of obstructed breathing during sleep (*i.e.*, loud snoring, bedclothes disordered, excessive night-time sweating), along with observed obstructive apneas, evidence of significant nasal obstruction and daytime intellectual sequelae are the primary clinical indicators of OSA. Unfortunately, clinical criteria alone are unreliable for making the diagnosis.[2]

Laboratory Diagnosis

Overnight oximetry with continuous recording can be used to make the diagnosis. Patients with significant OSA will demonstrate typical "saw tooth" desaturations, characterized by alternating cycles of desaturations and normal saturations, with each episode lasting between 45 and 90 seconds.[5] This is due to repeated cycles of obstruction, desaturation, arousal and a return to normal respiration, followed by repeat obstruction upon returning to sleep. This typically occurs in 20- to 30-minute periods, reflecting exacerbation during REM sleep (Figure 1). Although diagnostic of OSA, overnight oximetry has low sensitivity, with normal saturation occurring in children with clinically significant OSA.[5] The

Table 2

Symptoms And Signs Of Obstructive Sleep Apnea

Symptoms	Signs
• Persisting snoring, usually nightly	• Increased respiratory effort during sleep, with obvious struggling and indrawing
• Restless sleep, or "thrashing"	• Excessive night-time sweating
• Hypersomnolence (occasionally)	• Hypertrophied adenoids/tonsils
• Poor school performance/attention	• Chronic mouth breathing
• Enuresis (variable)	• Growth impairment

current "gold standard" for diagnosing OSA is polysomnography, where both respiratory status and sleep state are monitored on a continuous basis overnight. There are recognized standards for the performance of polysomnography, as well as reasonable normal data.[6]

Adults with OSA tend to develop either complete obstruction (apnea), or partial obstruction with associated desaturation (hypopnea). Compared to adults, children with OSA tend to develop ongoing obstructive hypoventilation, rather than episodic apnea/hypopnea. Consequently, there is not a clear correlation between severity, as determined by a number of apneas/hypopneas and clinical sequelae.[2] This can make determination of appropriate management difficult.

Treatment

In children, there is usually an anatomical etiology for OSA, with treatment usually involving surgery. Although there is not a close correlation between adenotonsillar size and presence or severity of OSA, close to 90% of children with OSA will have their obstruction successfully ameliorated, if not completely cured, by adenoidectomy.[2] The difficulties arise in determining which patients require surgical intervention. The original papers described the most severely affected children — that is, those with severe hypersomnolence, growth failure, and cor pulmonale arising from chronic sleep fragmentation and ensuing hypoventilation — in whom surgical intervention was clearly indicated.[2] OSA, however, constitutes a spectrum of disorder, ranging from the most severe cases with obstructive hypoventilation and its sequelae, through to children with primary snoring without any significant sequalae.

There are little longitudinal data that allow us to predict the natural history of untreated OSA, particularly in the milder end of the spectrum of disease.[1] There are no objective data for risk/benefit evaluation of the place of adenoidectomy in treatment; therefore, therapy has to be individualized. In the ideal situation, formal polysomnography would be available for objective evaluation, but this is frequently not the case. Clinical grounds (with all their limitations) often are the only criteria available.

Children with correctable craniofacial abnormalities usually improve significantly following surgical repair. This, however, may need to be delayed until an appropriate age for surgical cor-

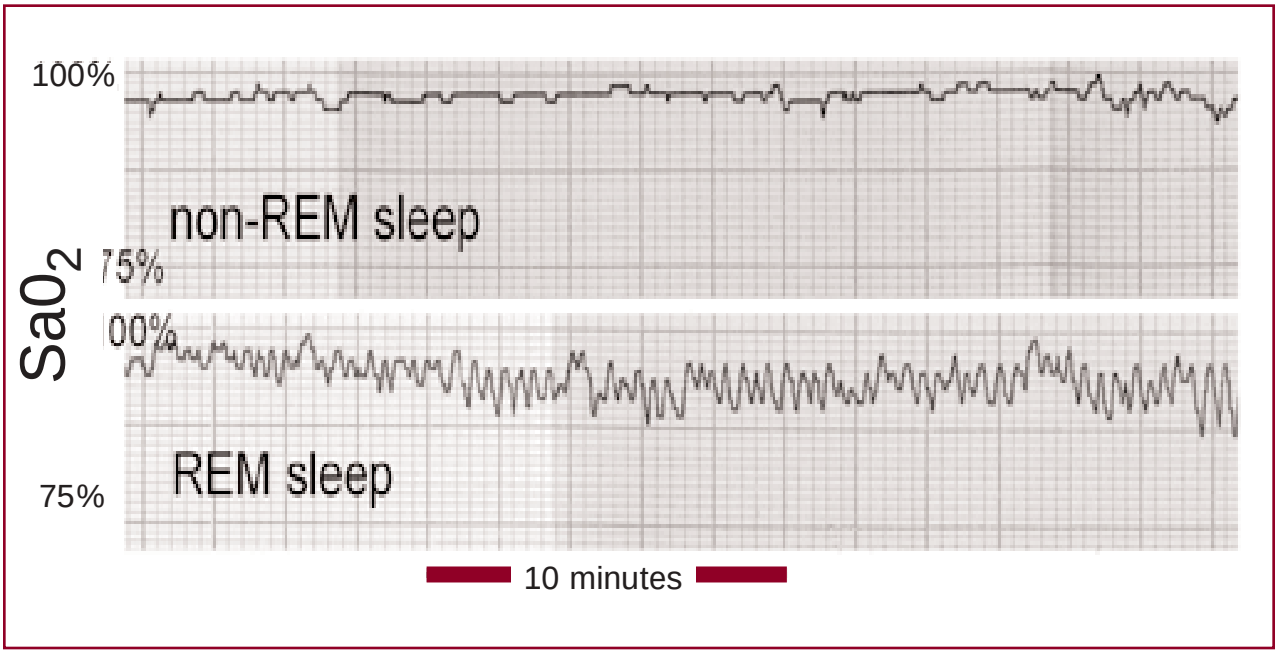


Figure 1. Continuous recording strip of overnight oximetry in a child with OSA.

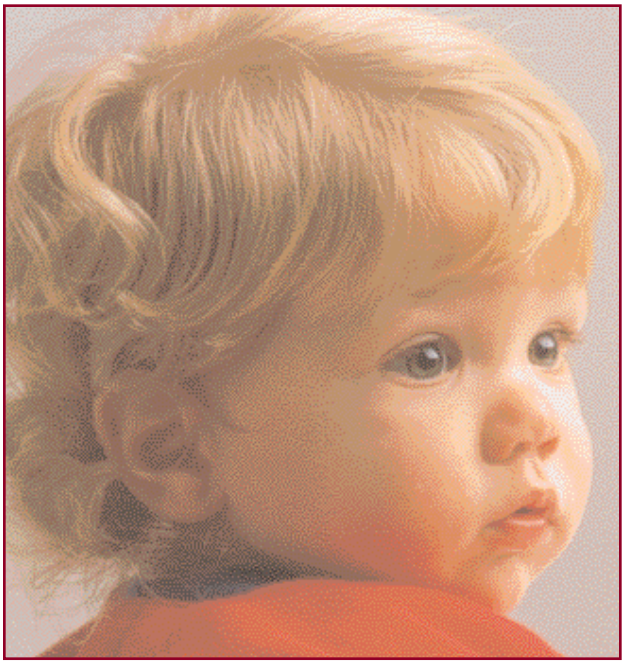
rection. Nasal CPAP is used much less in children than adults, with its use being limited primarily to children for whom surgery is either inappropriate or unsuccessful, or to provide temporary amelioration until an appropriate time for surgical correction. Although dental appliances are used successfully in adults, there are concerns regarding their impact on facial and dental growth in children. Dental appliances should probably only be used as part of a formal trial.

Central Sleep Apnea

Central Sleep Apnea (CSA) in adults is commonly defined as cessation of respiration for at least 10 seconds. Since children have a significantly higher respiratory rate, there is ongoing debate as to whether this is an appropriate definition for children. Some authors suggest a certain number of missed breaths instead.[6] Central apnea is not abnormal *per se*, being seen in completely normal children. A typical physiologic central apnea usually occurs during sleep-stage transition, commonly follows an inspiratory breath, is usually under 20 seconds long (although apneas of up to 25 seconds have been described in normal children)[7] and is not usually associated with any significant desaturation. Brainstem damage may present with central apnea, but it presents more commonly with hypoventilation.

Clinical Diagnosis

Central apnea is common during the neonatal period, particularly in premature infants. Repeated episodes of apnea during infancy usually signal immaturity of the respiratory drive, but may be in response to any noxious stimulus (*i.e.*, hypoglycemia, hypothermia, sepsis or seizures). Outside of infancy, CSA usually is physiologic. As a result, the child will be asymptomatic. A common scenario is that a parent becomes aware of his/her child's breathing pattern (the child snores or the mother reads about sleep apnea), and then



Apnea in infancy usually responds to stimulation and, if persistent, may require treatment with a respiratory stimulant, such as caffeine.

notices pauses in his/her child's breathing. Because of the widespread lay literature regarding SIDS, parents may observe a physiologic central apnea and become concerned that their child has a case of "missed" SIDS or an apparent life-threatening event (ALTE).

The diagnosis can usually be made solely on clinical grounds. The child is completely neurologically normal, the apneas single and infrequent, without any color change noted during an episode. The mother may misinterpret an episode as obstruction, in that frequently there is a gasp at the end of the apnea. Compared to OSA, however, the child shows no evidence of any respiratory effort during the apnea.

Laboratory Diagnosis

During infancy, the primary concern is whether the apneas are uncomplicated (signalled by an immature respiratory drive) or whether they reflect underlying disease. The laboratory investigations are aimed at excluding precipitating causes of apnea in infancy, such as sepsis, seizures, hypoglycemia or arrhythmia.

Treatment

Apnea in infancy usually responds to stimulation and, if persistent, may require treatment with a respiratory stimulant, such as caffeine. The apnea usually resolves with maturation, and the caffeine is discontinued. Physiologic central apneas require no treatment.

Sudden Infant Death Syndrome

SIDS is defined as "the sudden death of a child under one year of age, which remains unexplained after a complete post-mortem examination, including an investigation of the death scene and review of the case history."[8] It is, therefore, wrong to identify SIDS as the diagnosis whenever the actual cause of death can be explained.

There is evidence that, for a significant number of cases, a common underlying pathophysiology remains. It is rare during the first month of life, with a peak incidence at three months (now less than one in 2,000 live births). It then becomes decreasingly common by nine months, not occurring once infancy has ended.[9] Since SIDS usually occurs without obvious warning in an otherwise apparently healthy infant, and cannot be predicted, it is particularly worrisome for parents. As a result, there is an extensive body of lay literature on SIDS, and parents of young children are very well aware of its presence.

They frequently bring in their children for evaluation, concerned usually with observed episodes of perceived apnea. The family physician is faced with the difficult task of reassuring most parents that their child is normal, while attempting to identify the rare child that is truly at risk.

Treatment

The primary goal is prevention. There is an apparent association between preceding sleep-related apnea and subsequent SIDS. Consequently, apnea monitors have been prescribed in the hope that early detection of the preceding apnea would allow for early resuscitation and prevention of SIDS. Although used for many years, objective proof of their effectiveness is still not available.[10] There is a definite association between the incidence of SIDS and the child's sleeping position. In fact, a public education policy emphasizing the importance of infants sleeping in a supine position has produced a significant reduction in the overall incidence of SIDS.[9]

Apparent Life-Threatening Event

ALTE is defined as a "sudden, unexpected change in the child's behavior that is frightening to the caregiver, not leading to death/persistent collapse."[8] This is an unsatisfactory criterion, since it is too dependent upon the experience and observational skills of the observer. The parental concern is usually whether ALTE is an episode of missed SIDS. Although there is a link between ALTE and SIDS, it is not a particularly tight link; only between 7% and 9% of subsequent SIDS cases have a prior history of ALTE.

Numerous disorders may trigger an episode of ALTE (Table 3), and need to be excluded in the

Table 3

Causes Of Apparent Life-Threatening Events

Infectious
- Sepsis
- Meningitis
- Bronchiolitis (respiratory syncytial virus)
- Pertussis

Cardiac
- Failure
- Arrhythmia (short QT syndrome)

Neurologic
- Seizure
- Tumor
- Congenital/acquired hypoventilation syndrome

Metabolic
- Hypoglycemia
- Inherited disorders of metabolism
- Hypothermia
- Drug reaction

Abuse
- Trauma (intracranial hemorrhage/post-traumatic edema)
- Suffocation
- Spurious ("Munchausen's by proxy"/drugs)

Gastro-esophageal reflux

Respiratory
- Asthma
- Obstructive sleep apnea
- Vascular ring

Normal child (parental misinterpretation)

Unexplained (idiopathic)

Table 4

Causes Of Central Hypoventilation

Primary

- Congenital central hypoventilation syndrome

Secondary

- Obesity hypoventilation syndrome
- Brainstem lesions (*i.e.,* Arnold–Chiari malformation, hydrocephalus, achondroplasia [stenosis of the foramen magnum], meningoencephalitis, poliomyelitis)
- Respiratory muscle weakness (*i.e.,* spinal muscle atrophy, myopathy/muscular dystrophy, phrenic nerve paresis)
- Autonomic neuropathies (including familial dysautonomia)
- Neurodegenerative syndromes
- Drugs (respiratory depressants)

child's evaluation. Full polysomnography may be useful in excluding a diagnosis of hypoventilation or OSA, but it has little reliability in predicting the risk of subsequent SIDS.

Diagnosis

Diagnosis of ALTE is based upon clinical history. The primary concern is to identify underlying disorders that may have triggered the event. Clinical history, physical examination and laboratory studies are aimed at excluding underlying disorders.

Treatment

Primary treatment is aimed at any underlying etiologies, such as sepsis, seizure or cardiac arrhythmia. The primary concern with an idiopathic ALTE is the association with subsequent SIDS. The relationship between an ALTE and subsequent SIDS remains unclear; some studies reporting a no greater risk after a single event, while others report an incidence as high as 30% in children with repeated ALTEs requiring resuscitation.[1]

Although apnea monitoring has been used for many years in cases where no precipitating etiology can be identified, it remains unproven whether it actually has any significant impact on the subsequent risk of SIDS.[10] Subsequent deaths have occurred despite adequate monitoring, while false alarms are a common problem, even with the newer monitors. Apnea monitoring should only be used in selected cases, with appropriate family education and ongoing follow-up.

Hypoventilation Syndromes

Damage or disturbance of function of the peripheral chemoreceptors (carotid bodies), central chemoreceptors and respiratory nuclei (situated on the floor of the fourth ventricle), or failure of the respiratory muscles, will result in hypoventilation (Table 4). As noted, wakefulness is a respiratory agonist, so any of these syndromes is liable to be most severe during sleep.

Congenital Disorders

Congenital central alveolar hypoventilation (CCHS) can occur as a result of an isolated developmental defect of the brainstem, sometimes in association with other disorders (*i.e.,* neuroblastoma, Hirschsprung's disease and autonomic dysfunction) or as part of a more global central nervous system (CNS) insult (*i.e.,* hypoxic encaphalopathy or intracranial bleed).[1] In the case of CCHS as a result of a brainstem defect, the child may be born seemingly normal, except for the fact that there is minimal or no respiratory effort, with he/she requiring immediate ventilatory assistance. Subsequent attempts at withdrawing ventilation result in either profound hypo-

ventilation or protracted apnea. In the case of CCHS as part of a more global CNS insult, the primary CNS insult constitutes the major presentation, but subsequent attempts at withdrawing inflation have a similar result.

Clinical Diagnosis

Evidence of global CNS insult usually is clinically evident. In the absence of any apparent underlying neurologic insult, associated disorders, such as autonomic dysfunction or Hirschsprung's disease, need to be looked for.

Laboratory Diagnosis

Blunting, or a complete absence of ventilatory response to carbon dioxide, is diagnostic of CCHS. Formal testing of respiratory drive, however, is rarely necessary since all attempts at withdrawing ventilation are associated with either complete apnea or progressive hypercapnia in the presence of minimal respiratory effort. In children with apparent isolated brainstem dysfunction, laboratory testing is used to evaluate overall brainstem function (evoked potentials) and exclude anatomical lesions (magnetic resonance imaging [MRI]). Other associated disorders (*i.e.*, neuroblastoma) need to be excluded.

Treatment

Other than the rare situation involving a correctable primary lesion, the only available treatment is long-term ventilation. This usually requires a tracheostomy, at least for the first two to three years of life. In children with isolated brainstem dysfunction, there is usually steady improvement with age. The child is able to breathe spontaneously during the day, but requires some form of assisted ventilation during sleep. At that point, the tracheostomy may be successfully removed and the child transferred to nocturnal noninvasive ventilation.

Although there are limited long-term data, prognosis for children with idiopathic CCHS seems good, with the long-term survival being over 70%.[1] In children with more global CNS disease, the prognosis is dependent upon the underlying process. Although a number of respiratory stimulants, such as theophylline, acetazolamide and progesterone, have been tried, none have proven particularly effective. Their effectiveness was more than outweighed by their toxicity.

Acquired Disorders

- ***Primary.*** Any disorder that causes disturbance of the brainstem, particularly to the floor of the fourth ventricle at the level of the respiratory nuclei (such as Arnold-Chiari malformation, myelomeningocoele or brainstem tumor), is liable to result in disturbed ventilation during sleep. Since nerve roots of the 10th, 11th and 12th cranial nerves arise adjacent to this area, there may be associated disturbance in swallowing.
- ***Secondary.*** Patients with progressive neuromotor disease (*i.e.*, spinal muscle atrophy, muscular dystrophy) will eventually develop progressive hypoventilation as a result of a loss of respiratory muscle function. Similarly, patients with chronic, progressive pulmonary disease will eventually develop respiratory exhaustion, and again develop progressive hypoventilation, with secondary blunting of the respiratory drive.

Due to the chronic, progressive nature of these disorders, the majority of these patients may be remarkably asymptomatic, with the hypo-

ventilation developing insidiously. The usual symptoms are morning lethargy, headaches, anorexia and morning weakness. Affected children may actually be afraid to go to sleep, since they may still have an arousal response to hypercapnia and hypoxemia; being aware of the discomfort they experience following arousal.

Although apnea monitoring has been used for many years in cases where no precipitating etiology can be identified, it remains unproven whether it actually has any significant impact on the subsequent risk of SIDS.

Commonly, it only is when the hypoventilation has been corrected that the severity of the morning symptoms are appreciated. The symptoms are frequently masked by the primary disorder, the morning lethargy and weakness being attributed to muscle wasting. Malnutrition due to decreased appetite also may be attributed to the primary disorder. Unfortunately, in a number of cases, the diagnosis is only made when an opportunistic viral infection tips the child into frank cardiorespiratory failure, with the child being found to have unsuspected chronic respiratory acidosis.

Clinical Diagnosis

As noted, the symptoms and signs of acquired CSA are frequently masked by the primary disorder. Consequently, any child at risk of sleep-related hypoventilation needs to be evaluated for chronic or progressive hypercapnia. In addition, overnight carbon dioxide monitoring or morning blood gas testing needs to be performed if there is any doubt.

Laboratory Diagnosis

A blunted respiratory drive is the primary diagnostic criteria. In children old enough to comply, formal measurement of ventilatory response to carbon dioxide is the definitive test. A demonstration of nocturnal or morning hypercapnia with a clinically evident reduction in respiratory effort is usually all that is required.

Treatment

In the ideal situation, the primary disorder is amenable to therapy. Unfortunately, this is not usually the case and the primary disorder is either not amenable to therapy or slowly progressive and, ultimately, fatal. If the disorder is nonprogressive, non-invasive ventilation usually is effective in producing dramatic symptomatic improvement, associated with an improved appetite and a significant improvement in quality of life.[11] In progressive disorders, non-invasive ventilation usually is employed as a component of palliative treatment. The expected progress of the disease and the ultimate outcome (especially plans to progress to invasive ventilation) need to be discussed before embarking upon any form of long-term ventilation.

Non-respiratory Disorders

There are several disorders that may be confused with sleep-related respiratory disorders. Any disorders causing daytime hypersomnolence may be inferred as due to sleep fragmentation secondary to respiratory disorder, particularly if the child snores.

The most common problem is sleep-phase delay. This is particularly common during the teenage years, when the child has very poor sleep hygiene. This causes a phase shift or "jet lag," and subsequently he/she will complain of weekday morning hypersomnolence when having to get up early for school.

Narcolepsy is a rare condition in children, which may cause episodic daytime hypersomnolence. It is

characterized by episodes of inappropriate or partial onset of REM sleep, usually occurring in association with other symptoms (*i.e.*, cataplexy, hypnagogic hallucinations, sleep paralysis). Unfortunately, although clinical history may be highly suggestive, formal polysomnography with multiple sleep latency testing usually is required for a reliable diagnosis.

Children with central hypoventilation may have both sleep fragmentation and morning hypersomnolence, but they may also be afraid of sleep and suffer from insomnia. Insomnia is common in children. In younger children it is frequently due to limit-setting disorder (bedtime routines), and in adolescents it is due to sleep-phase delay. Sleep-related hypoventilation, however, does need to be considered in any at-risk child complaining of insomnia.

Conclusion

Sleep-related respiratory disorders constitute a diverse spectrum of disorders, ranging in significance from normal physiology to life-threatening disorders. Despite the ever-growing body of literature, large gaps in our knowledge remain, particularly as to the epidemiology, natural history and long-term clinical impact of these disorders. There is growing appreciation in the lay literature of the extent and significance of these problems. Family physicians need to be aware of the clinical features of the various disorders, as they will be called on to evaluate symptoms that may either comprise a normal variation in human physiology or indicate serious disease.

To the author's knowledge, there are only four sleep laboratories across Canada dedicated solely to the study of sleep-related respiratory disorders in children. The family physicians' job is complicated by the fact that, although sleep-related respiratory disorders may be one of the most common pediatric problems, there are limited clinical resources available to study this problem. Some disorders, such as OSA, are relatively common. The large spectrum of disorders, the variability in presentation and the lack of clear-cut management guidelines, however, make it difficult for a community physician to be totally knowledgeable about all aspects of these disorders. When in doubt, referral to a tertiary center may be necessary to clarify whether further investigation is required. CME

References

1. Marcus CL: Sleep-disordered breathing in children. Am J Respir Crit Care Med 2001; 164:16-30.
2. Guilleminault C, Eldrige F, Simmons FB, et al: Sleep apnea in eight children. Pediatrics 1976; 58:23-30.
3. Gozal D: Sleep-disordered breathing and school performance in children. Pediatrics 1998; 102:616-20.
4. Carroll J, Loughlin GM: Obstructive sleep apnea syndrome in infants and children: Clinical features and pathophysiology. In: Ferber R, Kryger M (eds.): *Principles and Practice of Sleep Medicine in the Child.* WB Saunders Company, Phildelphia, 1995, pp. 163-91.
5. Brouillette RT, Morielli A, Leimanis A, et al: Nocturnal pulse oximetry as an abbreviated testing modality for pediatric obstructive sleep apnea. Pediatrics 2000; 105:405-12.
6. American Thoracic Society: Standards and indications for cardiopulmonary sleep studies in children. Am J Respir Crit Care Med 1996; 153:866-78.
7. Hunt CE, Hufford DR, Bourguignon C, et al: Home documented monitoring of cardiorespiratory pattern and oxygen saturation in healthy infants. Pediatr Res 1996; 39:216-22.
8. National Institutes of Health Consensus Development Conference on Infantile Apnea and Home Monitoring, Sept. 29 to Oct. 1, 1986. Pediatrics 1987; 79(2):292-9.
9. Hauck FR, Hunt CE: Sudden infant death syndrome in 2000. Curr Probl Pediatr 2000; 30:237-61.
10. Grogaard JB: Apnea monitors. Acta Paediatr Suppl 1993; 82:111-3.
11. Simonds AK, Muntoni F, Heather S, et al: Impact of nasal ventilation on survival in hypercapnic Duchenne muscular dystrophy. Thorax 1998; 53:949-52.

Suggested Reading

1. Ferber R, Kryger M: *Principles and Practice of Sleep Medicine in the Child.* WB Saunders Company, Philadelphia, 1995.
2. Hauck FR, Hunt CE: Sudden infant death syndrome in 2000. Curr Probl Pediatr 2000; 30:237-61.
3. Marcus CL: Sleep-disordered breathing in children. Am J Respir Crit Care Med 2001; 164:16-30.