Obstructive sleep apnea is much more than a sleep disorder. Researchers have just scratched the surface when it comes to determining the effects of disordered breathing during sleep. From insulin problems to cardiovascular dangers to stroke risk, obstructive sleep apnea has a far-reaching impact on patients’ health.

Our collection of articles on sleep apnea is designed to delve deeper into the presentation, diagnosis and treatment of this important health problem and contains information you won’t find anywhere else. We hope you enjoy it.
RESEARCH SHOWS that patients with untreated obstructive sleep apnea (OSA) are predisposed to cardiovascular death. Coexisting diabetes and insulin resistance have not been thoroughly explored in OSA. Research suggests that they should be.

OSA is a partial or complete narrowing of the pharynx due to sleep-induced relaxation of upper airway muscle tone. Apneic and hypopneic episodes during sleep are evidenced by airflow cessation, oxygen desaturation and interrupted sleep.

Increased patient morbidity and mortality have been linked to coexisting diabetes, insulin resistance and OSA. Additionally, insulin resistance leads to diminished insulin-mediated glucose clearance and is associated with metabolic syndrome. Continuous positive airway pressure (CPAP) can promptly improve insulin sensitivity in patients with OSA.

These facts prompt a clinically significant question: Does CPAP influence insulin sensitivity in adult patients who have OSA with or without a diagnosis of type 2 diabetes?

Background
Metabolic syndrome is associated with insulin resistance. Insulin resistance in OSA may be due to stimulation of sympathetic pathways by the arousals, hypoxia and disruptive sleep that impair glucose tolerance.

The association between increased morbidity and mortality in patients with coexisting diabetes and insulin resistance as a result of OSA has been demonstrated but not thoroughly researched. OSA corresponds to stress that advances insulin resistance in obese and nonobese patients, as indicated by insulin resistance index and increased fasting serum insulin.
level based on the homeostasis model assessment method. Therefore, it is essential to evaluate all patients with OSA for insulin resistance.

Review of Literature
We performed a comprehensive literature search of CINAHL, MEDLINE, EBSCOhost, ProQuest and the Cochrane Library for reviews investigating the effects of CPAP on insulin resistance. We specifically sought studies published between January 1994 and February 2009. We selected randomized, controlled studies, with evaluation before and after application of CPAP for sleep apnea. From a total of 3,664 studies published, 47 fit the eligibility criteria for inclusion in our review.

Findings
Twenty-six of the 47 studies were randomized, controlled trials that were considered appropriate. Trials that included special patient populations with neuromuscular diseases or cerebral malformations or subjects with central sleep apnea were not reviewed.

We examined only studies whose participants had undergone a diagnostic test or intervention to support the diagnosis of OSA. Our electronic searches were complemented with a systematic search of the reference lists of all eligible studies and relevant review articles.

The findings of these studies suggest that CPAP has an independent positive effect on insulin resistance. Minimum CPAP oxygen saturations were a significant determinant of fasting insulin levels and preinsulin and postinsulin resistance levels. Most researchers believed that this phenomenon may be related to microawakenings or hypoxia-related nocturnal increases in sympathetic system and hypothalamic–pituitary–adrenal axis activities. Researchers believed that hyperinsulinemia was related to hypoxia and hypercapnea during OSA caused by an overly stimulated sympathetic nervous system.

The mechanism believed responsible for increased insulin resistance is a stress response activated by chemoreceptors in the carotids, the aortic arch and the medulla of the brainstem in the presence of hypoxia. The corollary of these events is the release of catecholamines, epinephrine, norepinephrine and cortisol. Based on this assumption, OSA plays a role in obesity by impeding glucose management with insulin.

Another general hypothesis is that increased adrenergic activity with excess catecholamine production contributes to insulin resistance and arises as a consequence of increased sympathetic nerve activity in OSA. OSA either directly increases risk factors for metabolic syndrome, or OSA and metabolic syndrome share a common risk factor, such as obesity and sedentary lifestyle.

We found further support in a study showing that increases in the apnea–hypopnea index were associated with worsening insulin resistance independent of obesity, and increased rates of OSA were associated with increased rates of impaired glucose tolerance.

A study of nonhypertensive, nondiabetic, nonobese Korean men showed changing patterns of glucose and insulin levels at 1 hour and 2 hours after the ingestion of 75 g of glucose. These levels were appreciably different between habitual snorers and nonhabitual snorers, but no significant difference in blood glucose and insulin levels occurred.

OSA is related to hyperinsulinemia, which may be correlated to primary insulin resistance mediated by increased catecholamine secretion. As a result, CPAP treatment of OSA may reduce insulin resistance.
and non-diabetic patients.4

In healthy patients, transient hypoxia (30 minutes) can impair glucose tolerance and is associated with insulin resistance. These findings support that OSA is independently associated with insulin resistance and improvement is achieved with CPAP.3,9

Studies also show that patients with OSA who are at risk for metabolic syndrome would benefit from CPAP treatment because it improves insulin resistance. Longer CPAP use (3 weeks or more) produces greater reduction in fasting insulin levels and a reduction in OSA symptoms.18

The use of stable CPAP at a pressure level to re-establish patency of the upper airway has been the foundation of treatment for OSA since the 1980s.19 CPAP has demonstrated prompt improvement in insulin sensitivity. It follows, then, that CPAP may be effective in inhibiting OSA-related insulin resistance in adults with or without diabetes.

Recognizing Risk

All NPs and PAs should recognize risk factors for OSA. Patients with fatty deposits around the neck, neck circumference greater than 17 inches, self-reported daytime tiredness, or loud or heroic snoring should be screened for OSA with sleep studies.

Patients with OSA are at increased risk for insulin resistance, and patients with known OSA and diabetes are at risk for complications as a result of nonadherence to CPAP therapy. Question patients with OSA about their adherence.4

The use of CPAP can produce improvement in insulin resistance, and in patients with diabetes it may result in diminished mortality and morbidity related to end-stage organ disease.5

References

“CHRIS,” a 6-year-old boy of Asian-American descent, presents with a 12-month history of behavioral problems in school. Today’s visit with this established patient was prompted by the teacher’s suggestion that Chris be evaluated for attention deficit–hyperactivity disorder (ADHD) due to lack of attentiveness and hyperactivity in the classroom. Both parents accompanied the child, with a DSM-IV Conners’ rating scale in hand.

Past Medical History
The chief complaint began at the age of 5, while Chris was attending preschool. His teacher reported recurrent episodes of disruptive behavior in the classroom. Comments on the progress reports stated that Chris was inattentive, had difficulty following directions, did not comply with classroom rules, and demonstrated lack of self-control. The parents said Chris does not exhibit these behaviors at home.

Chris resides at home with both parents and his 9-month-old infant brother. He has no surgical history, and his only medication is a daily vitamin. Chris is allergic to amoxicillin; this was diagnosed at age 12 months after a 7-day treatment for acute otitis media with effusion (AOME).

The family history was noncontributory with no reports of mental illness, asthma, allergies, sleep disorders, hypertension or cardiac disease.

Past medical history included treatment for strep throat twice in the past year (treated with Biaxin) and one episode of bilateral AOME and sinusitis (treated with Omnicef and Flonase).

Chris was born at full term, 38 weeks. He failed his initial newborn hearing screen but passed the second time, and he had physiologic jaundice at birth, with bilirubin levels peaking at 15 mg/dL. Past infections include varicella at 13 months and roseola at 14 months. Immunizations are up-to-date.

Review of Systems
The parents report that Chris does not exhibit daytime sleepiness, sleepwalking or talking in his sleep. Chris is well-behaved and plays appropriately with his brother. The parents say Chris sleeps independently in his own room and gets 10 hours of sleep each night. He is in bed by 8:00 p.m. and awakens at 6:30 a.m. However, they say he is difficult to arouse in the morning and appears to have had a restless night. He complains of being thirsty upon awakening. He has a dry mouth and a crusted, white residue around his lips. He drools at night. He has reported no headache, dizziness or ringing in the ears.

Diet and nutritional questioning reveals that Chris has a poor appetite and eats only several small snacks daily. He doesn’t consume any caffeine or chocolate, and sweets are limited. Chris takes a long time to finish meals and seems bored while eating.

The patient was breastfed for 9 months, began eating solids at 6 months of age, and currently drinks 15 ounces of milk daily. He has no particular food allergies or dislikes.

PATRICIA L. BARROZO is a family nurse practitioner student at the University of North Florida in Jacksonville. She is scheduled to graduate in April 2011.
months, and has no known food allergies or sensitivities. The parents report no nausea, vomiting, diarrhea or constipation, heartburn or belching. The genitourinary system review finds no history of bedwetting or encopresis. The endocrine review is negative for diabetes or growth retardation.

The cardiovascular and respiratory review reveals loud snoring with snorting and gagging noises. Mom reports sleeping in the room overnight to observe the behavior. She noticed Chris breathing with his mouth open, snoring throughout the night, and restlessness and labored breathing.

**Physical Examination**

Chris is in the 50th percentile for height and weight. All immunizations are up-to-date. Vital signs are as follows: temperature 97.4 F, heart rate 88, blood pressure 100/68 mm Hg and respiratory rate 22. He communicates well, does not appear anxious and looks healthy and well-nourished.

The patient’s head is normocephalic. Bilateral tympanic membranes are intact, with no retraction or perforation. The NP notes a small amount of

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### Table 1

**Pediatric Sleep Snoring Assessment**

*Developed by Bruce R. Maddern, MD; appears with permission.*

#### Introduction and Background

In general when children sleep, they should sleep with their mouth closed, breathing quietly through their nose. If this is not the case, your child may have some component of sleep disturbance. Your observations of your child’s sleep pattern will help us determine whether the “snoring” is significant and warrants further evaluation or treatment. Some snoring may be a normal event in healthy children.

#### Parent Instructions

Please answer the questions about the behavior of your child during sleep and when awake. Please watch your child sleep when in good health and answer the questions for an average night’s sleep. Specifically, after your child has been asleep for 30 to 60 minutes, sit in his or her bedroom and observe the sleep pattern for 10 to 15 minutes on 5 or 6 separate evenings. Please check the appropriate response.

- **Occasionally** means “on less than half the nights” or “less than half the time.”
- **Usually** means “on more than half the nights” or “more than half the time.”
- **Choose when ill** if the problem is only noted with illness.

<table>
<thead>
<tr>
<th>When sleeping, on a typical night, does your child …</th>
<th>Never</th>
<th>Occasionally</th>
<th>Usually</th>
<th>Nightly</th>
<th>When ill</th>
</tr>
</thead>
<tbody>
<tr>
<td>Snore?</td>
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<tr>
<td>Is it loud</td>
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<tr>
<td>Have loud or heavy breathing?</td>
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<tr>
<td>Sleep with the mouth open?</td>
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<td>Repeatedly cough, gag or gasp?</td>
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<tr>
<td>Move about restlessly?</td>
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<td>Struggle to breathe?</td>
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<tr>
<td>Pause or stop breathing?</td>
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<td>If so, how many seconds? __________</td>
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<td>Have episodes of self-awakening?</td>
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<tr>
<td>Wet the bed? <em>(after being potty-trained and dry)</em></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Is your child …</th>
<th>Never</th>
<th>Occasionally</th>
<th>Usually</th>
<th>Nightly</th>
<th>When ill</th>
</tr>
</thead>
<tbody>
<tr>
<td>A mouth breather during the day?</td>
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<tr>
<td>Hard to wake after sleeping?</td>
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<tr>
<td>Sleepy or tired during the day?</td>
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<tr>
<td>Overly active, have attention or learning problems?</td>
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<tr>
<td>Check One:</td>
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<tr>
<td>Underweight</td>
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<td></td>
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<tr>
<td>Normal weight</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overweight</td>
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</tbody>
</table>
bilateral amber serous drainage from the ears. The right external auditory canal is partially occluded with cerumen. The nasal mucosa is pink and moist. A deviated septum is present, but no other craniofacial deformities are evident. Mild erythema of the throat is present. Tonsils are 3+ with notable adenoidal hypertrophy. The respiratory examination reveals mild nasal congestion and speech with a nasal tone. Lung sounds are clear with no cough, wheezing, stridor or tachypnea. No lymphadenopathy is present.

The cardiovascular examination reveals normal rate and rhythm. Musculoskeletal development is appropriate for age with full range of motion in all extremities.

**Assessment & Differentials**

Studies suggest that as many as 10% of school-aged children experience some problem related to sleep disturbance or disruption.1 It is imperative that clinicians recognize the link between sleep-disordered breathing and hyperactivity. Without proper identification of the underlying cause of inattentive or hyperactive behavior, a premature and inaccurate diagnosis can be made. This can lead to unnecessary pharmacologic intervention.

Common problems associated with lack of sleep include hyperactivity, inattention, ADHD, excessive daytime sleepiness, poor cognitive function and inability to focus in school. The Conners’ rating scale is a behavioral assessment commonly used to assess for ADHD.1 It consists of an 80-item questionnaire that is completed by the teacher and by the parent prior to seeing the pediatrician or psychiatrist. This rating scale is not considered a diagnostic tool, but a screening examination based on observation.1

ADHD is the most common behavioral disorder of childhood, with an estimated prevalence of 20% to 40% in school-aged children.1,2 It affects boys 10 times more often than girls.1,2 The neurobehavioral symptoms of ADHD are often first noticed by the classroom educator. Children with ADHD typically have difficulty sitting still in class and require constant reinforcement to stay on task.3,4 The child may talk out of turn and daydream during instruction. Impulsivity and frequent attention seeking are also common.3,5,6 To make a diagnosis of ADHD, six or more symptoms of hyperactivity and impulsivity must persist for at least 6 months, to a degree that is maladaptive and inconsistent with developmental age performance.7,8

Anxiety was a potential diagnosis for Chris, but the NP ruled it out due to a lack of data supporting probable cause. The child has good eye contact, does not appear to be restless or anxious during evaluation, and is attentive during interviewing. According to parental report, he plays well with his sibling and has many friends at school and in the neighborhood.

The NP ruled out central apnea as the probable cause because it is primarily seen in newborns and premature infants. She then considered obstructive sleep apnea (OSA) as the diagnosis. OSA has an estimated prevalence between 1% and 3% in preschool and school-aged children.3,9,10-12 Pediatric OSA was first described in the medical literature in 1975.4 Symptoms include snoring, breathing cessation, choking, gasping, struggling to breathe, restlessness and frequent awakening from sleep.9,10 The gold standard for

### Table 2

**Questions From the Tayside Children’s Sleep Questionnaire**

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. How long after going to bed does your child usually fall asleep?</td>
<td>Time taken after going to bed.</td>
</tr>
<tr>
<td>2. Does your child go to bed reluctantly?</td>
<td>Reasons for reluctance to go to bed.</td>
</tr>
<tr>
<td>3. Does your child have difficulty getting to sleep at night (and maybe require a parent to be present)?</td>
<td>Difficulty in falling asleep at night.</td>
</tr>
<tr>
<td>4. Does your child fall asleep in his or her own bed or somewhere else?</td>
<td>Sleep location.</td>
</tr>
<tr>
<td>5. Does your child wake up two or more times in the average night?</td>
<td>Frequency of waking.</td>
</tr>
<tr>
<td>6. After waking up during the night, does your child have difficulty falling asleep again by himself or herself?</td>
<td>Difficulty in falling asleep again.</td>
</tr>
<tr>
<td>7. Does your child sleeps in your (the parents’) bed at some time during the night?</td>
<td>Sleeping arrangements.</td>
</tr>
<tr>
<td>8. If the child wakes, does he or she need a comfort object to return to sleep?</td>
<td>Comfort objects needed.</td>
</tr>
<tr>
<td>9. Does the child usually want a drink during the night (including breast milk or formula)?</td>
<td>Drink needs during the night.</td>
</tr>
<tr>
<td>10. Do you think your child has sleeping difficulties?</td>
<td>Perception of child’s sleeping difficulties.</td>
</tr>
</tbody>
</table>

OSA begins with early signs of snoring and later leads to increased upper airway resistance syndrome and partial occlusion. The American Thoracic Society's consensus committee on OSA recommends that PSG be obtained before a tonsillectomy and adenoidectomy (T&A) to differentiate snoring from true OSA.

However, the diagnostic criteria are based on adult performance measures for test interpretation. Some pulmonologists and pediatric ENTs suggest that PSG is not necessary for positive diagnosis in children, and they proceed with T&A if a child presents with repeated episodes of tonsillitis and middle ear dysfunction or has adenotonsillar hypertrophy. Screening measures such as the pediatric snoring assessment by Bruce Maddern, MD (Table 1) or the Tayside Children's Sleep Questionnaire (Table 2) may be used.

Treatment Measures
T&A is considered the first-line treatment for OSA in children, and it is 85% to 90% effective. CPAP and oxygen therapy are not recommended for children because they may worsen hypoventilation and cause oxygen toxicity. Weight loss is clinically indicated for children who are obese.

Patients with OSA should avoid allergens such as indoor pollutants, cigarette smoke and outdoor pollen. Short-term pharmacologic interventions to reduce upper airway inflammation and irritation include any combination of saline mist, topical nasal steroids and mast cell stabilizers, as well as oral systemic antihistamines and leukotriene inhibitors. However, these treatments are only indicated for temporary relief. The Food and Drug Administration has issued a public advisory about these types of medications and their risk for potential side effects in children ages 2 to 11.

Treatment Plan
The NP referred Chris to a pediatric pulmonologist. At the initial visit, an in-office radiologic exam of the head and neck showed severe adenotonsilar hypertrophy with approximately 75% airway occlusion (see figure). The pulmonologist referred Chris to a pediatric ENT and recommended surgical intervention. The ENT determined that a T&A was necessary.

Chris returned to his pediatric provider's office for preoperative screening. A PSG was completed the next week, and it documented moderate to severe sleep apnea with periods of hypopnea, hypoventilation and low oxygen saturation dropping as low as 88% on pulse oximetry. An audiologist performed speech and hearing screenings and found no deficits on audiometry and tympanometry. Laboratory analysis included a complete blood count, and all values were within normal parameters.

At a primary care, postoperative follow-up 1 month after surgery, Chris’ parents reported he was sleeping through the night with no complications. They saw immediate resolution of sleep apnea symptoms. Importantly, Chris has made significant improvements in classroom behavior.

References
OBSTRUCTIVE SLEEP APNEA (OSA) is a chronic condition in which the pharyngeal structures cause occlusion of the upper airway, leading to cessation of breathing while asleep. These occlusions elicit awakenings or arousals to regain patency of the airway.¹ OSA affects 12 million to 18 million U.S. residents, and 80% to 90% of cases are undiagnosed.¹⁻³

**Risk Factors**
OSA risk factors and symptoms (Table 1) tend to overlap with or remain hidden by other conditions, contributing to underrecognition.¹⁻³ Yet hypertension, obesity, gender, age, crowding of the oropharynx and family history are easily recognizable. Any patient with hypertension should be screened for OSA, as should all obese patients.⁴

Excess neck circumference (more than 17 inches in men and 16 inches in women) also is a risk factor for OSA.⁵ Men are twice as likely as women to be affected, and a higher prevalence has been noted in older adults.¹

Evidence from racial, familial and twin studies indicates that OSA has a strong genetic basis, with 35% to 40% of variance attributed to genetic factors. Therefore, family history is essential to a thorough evaluation.⁶

**Presentation and Screening**
The symptoms of OSA are vague and mimic other common health conditions. Patients can reliably describe daytime fatigue, hypersomnia, morning headaches and decreased concentration. But during sleep, patients are not aware of snoring, gasping or excessive leg movements unless a sleeping partner observes them. Always inquire about a partner’s observations.

Obesity, an easily identifiable risk factor, should always prompt questioning. A quick examination of the oropharynx provides further objective information about a patient’s risk for OSA. Hypertension, obesity, diabetes, metabolic syndrome and family history must be included in the evaluation for possible OSA. Asking whether anyone in the patient’s family has been diagnosed with OSA may not yield the necessary information. Instead, ask, “Does anyone in your family snore loudly?”
Complications
As our understanding of OSA has broadened, links to other disease states have been identified. OSA can be linked to uncontrolled hypertension, coronary artery disease, cerebrovascular accident, type 2 diabetes and pulmonary hypertension.7 OSA has been identified as a primary cause of hypertension.8 In addition, cardiovascular disease is linked with undiagnosed and untreated OSA.9 This increased cardiovascular risk is likely related to repeated apneic events leading to hypoxia or hypoxemia, changes in intrathoracic pressure, and central nervous system arousals.

A decreased variance in heart rate and blood pressure during sleep (approximately 10% to 15%) is normal, but this is not the case in patients with OSA.10 These patients experience an increase in systemic blood pressure due to decreased oxygen saturation and increased oxygen demand related to apneas.11-14 Over time, these changes create the potential for increased cardiovascular complications such as uncontrolled hypertension, arrhythmias and heart failure.15

In patients with OSA, stroke may occur as a result of subclinical cerebrovascular disease, also known as silent brain infarction. This can be detected with magnetic resonance imaging.16 The recognition of OSA as a risk factor for ischemic stroke is derived primarily from data indicating that it is present in hypertension and cardiovascular disease; both are major risk factors for stroke. The risks for stroke related to OSA include extreme blood pressure variations and reduction in cerebral blood flow.17 Another mechanism can be inflammatory responses that occur in cardiovascular disease and diabetes. These escalate the activation of platelets, boosting the prevalence of silent brain infarction or stroke.16 Studies have also noted a correlation between OSA and insulin resistance.18 Patients with OSA are at increased risk for automobile and occupational accidents related to daytime sleepiness. They may have neurocognitive impairment due to repeated sleep disturbance and oxygen desaturation, putting them at high risk for accidents.19

Screening Tools
Several OSA screening tools are available. The Epworth Sleepiness Scale (http://epworthsleepnessscale.com/) assesses level of daytime sleepiness and is an excellent predictor of OSA. This questionnaire, which provides subjective data, consists of only eight questions. It is self-administered and is easily understood by a majority of the population, making it a convenient option for most offices. The Berlin Questionnaire (http://www.edward.org/workfiles/sleep%20center%20Berlin%20Sleep%20Eval.pdf) is more predictive of the presence of OSA because it focuses on multiple risk factors as opposed to daytime sleepiness alone. It is a self-administered screening tool that provides some objective data as well. The STOP-BANG questionnaire (http://www.norcalmutual.com/publications/claimsrx/feb_10.pdf), another convenient screening tool, provides almost exclusively objective data. This self-administered test provides a scale of low risk to high risk for OSA, as well as possible severity.

Sleep Evaluation
Another way to perform OSA screening is to include questions about sleep in the patient history at each visit (Table 2). Simply ask patients about their history of snoring, daytime sleepiness and witnessed apneas. This can increase the diagnosis of OSA significantly.20

The primary care office is an excellent setting for OSA evaluation, but each provider involved in a patient’s care has the opportunity to do so as well. For example, a patient with new onset atrial fibrillation will be sent for routine cardiac evaluation. Screening for OSA should be added at this point, since OSA is a contributing and complicating factor in arrhythmias. Patient

Table 1
Clinical Presentation Of OSA

<table>
<thead>
<tr>
<th>Risk Factors</th>
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<tbody>
<tr>
<td>Older than 45 years</td>
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<tr>
<td>Male</td>
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<tr>
<td>Obese</td>
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<tr>
<td>Hypertension</td>
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<tr>
<td>Family history of OSA or loud snoring</td>
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<tr>
<td>Micrognathia</td>
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<tr>
<td>Crowded oropharynx</td>
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</table>

<table>
<thead>
<tr>
<th>Symptoms</th>
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<tbody>
<tr>
<td>Loud snoring</td>
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<tr>
<td>Witnessed apneas</td>
<td></td>
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<tr>
<td>Excessive daytime sleepiness</td>
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<tr>
<td>Reduced quality of life</td>
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<tr>
<td>Daytime fatigue</td>
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<tr>
<td>Morning headaches</td>
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<tr>
<td>Unrefreshing sleep</td>
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<tr>
<td>Gasping sensation at night</td>
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<tr>
<td>Nocturia</td>
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<tr>
<td>Kicking or excessive movement during sleep</td>
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complaints of concentration difficulty, morning headaches or memory changes may be subtle indications of OSA. After routine labs are completed and are normal, a neurology consult may be in order. OSA could be added to the differential diagnosis list by adding a few simple questions. Screening questions can be easily added to an existing protocol on the electronic health record or as an office form in a paper chart.

Once significant risk factors or signs and symptoms have been recognized through screening and physical examination, a referral for diagnostic testing is needed. The standard diagnostic tool for OSA is polysomnography.2

## Treatment
Treatment for OSA consists of keeping the airway open during sleep. In minimal to mild cases, weight loss and positional therapy may be effective. In positional therapy, patients avoid the supine position. For moderate to severe cases, the gold standard of care is continuous positive airway pressure.30 Devices inserted into the mouth for sleep can be successful in treating snoring and mild cases of OSA.30 Several surgical procedures are available for severe cases. OSA in children can typically be corrected with a tonsillectomy.21-23

## OSA Screening Using the Patient History

<table>
<thead>
<tr>
<th>Patient Completes:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Do you snore or have you been told that you snore?</td>
</tr>
<tr>
<td>2. Do you have daytime sleepiness?</td>
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<tr>
<td>3. Do you have fatigue?</td>
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<tr>
<td>4. Have you ever had a gasping sensation at night?</td>
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<tr>
<td>5. Have you ever been told that you stop breathing while you are asleep?</td>
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<tr>
<td>6. Have you ever fallen asleep while driving or felt like you might?</td>
</tr>
<tr>
<td>7. Does anyone in your family have OSA or snore loudly?</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Provider Completes:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Patient snores: <strong>Y or N</strong></td>
</tr>
<tr>
<td>2. Obese: <strong>Y or N</strong></td>
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<tr>
<td>3. Crowded oropharynx: <strong>Y or N</strong></td>
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<tr>
<td>4. Positive family history of OSA or snoring: <strong>Y or N</strong></td>
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<tr>
<td>5. Hypertension: <strong>Y or N</strong></td>
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A “yes” answer to any of these questions indicates the need for further sleep evaluation.

## References

ThE USE OF noninvasive positive airway pressure (PAP) to manage sleep-disordered breathing is increasing among children. PAP therapy can be appropriate for obstructive sleep apnea as well as chronic respiratory insufficiency in children with obesity or neuromuscular weakness.

“Megan” is an 18-year-old girl with Down syndrome who has been treated for OSA at the pediatric sleep disorders center at Children’s Hospital of Wisconsin in Milwaukee. Her story highlights several key issues in PAP therapy for children. First and foremost, it is important to identify the appropriate system and mask interface. Megan and her family required extensive support and education during the initiation of PAP therapy. She had difficulty acclimating to the mask interface and required frequent monitoring during the desensitization period.

Children with Down syndrome are at higher risk for OSA than unaffected children due to craniofacial anomalies. The incidence of OSA in the general pediatric population is estimated at 3%. The most common cause of OSA in children is adenotonsillar hypertrophy, and adenotonsillectomy is the primary treatment. In certain circumstances, PAP therapy may be indicated as follows:

- when adenotonsillectomy is not indicated or contraindicated
- when adenotonsillectomy fails to resolve symptoms, usually children with additional risk factors (e.g., obesity or craniofacial anomalies)
- prior to adenotonsillectomy in children with severe sleep apnea
- as a means of noninvasive ventilatory support in children with hypventilation secondary to neuromuscular weakness or central sleep apnea secondary to a brain abnormality.

PAP Systems

PAP therapy typically is initiated in school-age children or adolescents. The Food and Drug Administration has approved use of PAP for children 7 and older who weigh more than 40 pounds. Anecdotal reports state that children as young as 2 years may be managed with PAP therapy. A letter of medical necessity, in combination with pediatric specialty consultation, is recommended.
with sleep study results, usually is adequate to obtain insurance reimbursement for PAP.

Several PAP modes are available for children. Continuous positive airway pressure is the mainstay of therapy for OSA management. Guidelines from the American Academy of Sleep Medicine (AASM) recommend that for children younger than 12 years, the minimum pressure should be 4 cm H₂O, with a maximum of 15 cm H₂O. For children older than 12 years, the recommended maximum pressure is 20 cm H₂O.

Bilevel positive airway pressure therapy is appropriate if the child cannot tolerate higher CPAP pressures or if continued respiratory events occur at the maximum recommended pressure. Bilevel therapy can be used as a means of noninvasive ventilatory support for children with hypoventilation secondary to obesity or neuromuscular weakness.

According to AASM guidelines, the minimum starting expiratory positive airway pressure (EPAP) is 4 cm H₂O, with a minimum inspiratory positive airway pressure (IPAP) of 8 cm H₂O. The recommended maximum IPAP is 20 cm H₂O for children under 12 years old and a maximum of 30 cm H₂O for children over 12 years old. An excess of 30 cm H₂O of upper airway pressure increases the risk for barotrauma and other morbidities.

A backup rate is added to bilevel therapy when central apneas are observed. Of note, children with hypoventilation secondary to neuromuscular weakness may be prone to intermittent central apneas and usually benefit from a backup rate.

A ramp feature may be helpful for children who are unable to fall asleep with the PAP in place. The ramp is set for 10 to 20 minutes, and the child falls asleep as the PAP ramps up to the final setting.

**PAP Interfaces**

It is important to select an appropriate interface that fits a child well and stays secure. Only a few interfaces are FDA-approved for children. Sometimes the smallest size of an adult interface can be used, although it may be necessary to modify the headgear to fit a child’s smaller head.

Only one full face mask is designed specifically for children. Some children cannot take off the full face mask by themselves and have significant risk of aspiration if they vomit. Consider an alternate interface and chin strap if the child is prone to mouth breathing.

In some cases, it may be necessary to try a variety of mask interfaces. For example, when a child is unable to tolerate a nasal mask due to skin breakdown, claustrophobia or other issues, nasal pillows or a nasal cannula device may be selected.

Retrusion of the maxilla or upper teeth is a concern that has developed as more children and younger children are being started on PAP therapy. The hypothesis is that pressure from the mask, even a properly fitted mask, can causes displacement of the teeth, in some cases causing a significant underbite. The mask interface may need to be adjusted or nasal pillows considered. Dentists should be made aware if a child is being treated with PAP therapy.

**Getting Started**

Adherence to PAP therapy can be improved with patient and family education and support. Often this starts once a child has a diagnostic sleep
study to confirm the presence and severity of sleep-disordered breathing; PAP therapy is initiated during a separate study. In the interim, the child is fitted with a mask interface and tries out the mask at home gradually.

Initially, the child wears the mask during periods of wakefulness. A common trial time is while watching television. Eventually, the child is able to fall asleep with the mask on, and the parents remove it once the child is asleep.

Success during this desensitization period often is based on the parents’ ability to remain calm and persistent. They can encourage their child to pretend to be a jet pilot or scuba diver, or have their child put the mask on a teddy bear or doll. In Megan’s case, positive rewards were helpful, including a favorite breakfast of French toast if she wore the mask at night.

**Maintaining Compliance**

Using PAP therapy on a consistent basis takes commitment from the child and family. A sleep center staff member with expertise in family-centered care can provide tips to improve PAP adherence and address common complaints.

Eye irritation can be corrected by reseating the mask on the face and readjusting the headgear; the mask should be as loose as possible while still creating a seal.

Skin irritation occurs if the mask does not fit properly or if it becomes dirty or worn out. The mask should be washed daily and then air-dried. Most insurance companies allow for a mask replacement every 6 months. Vitamin E cream can be useful to heal any associated skin irritation.

Children using PAP therapy may complain of a dry mouth upon awakening in the morning. Nasal congestion and nosebleeds also may occur because of breathing in dry air. This usually is relieved by adding humidity (either heated or cool) into the PAP system. The amount and temperature of the humidity depends on the time of year and ambient temperature. A nasal saline spray at bedtime and in the morning may be helpful.

For children with allergic rhinitis, it is especially important that the PAP machine is placed on the bedside table rather than the floor, to avoid dust being pulled into the system.

Children using PAP therapy can be managed successfully for the long term. They should be followed in an outpatient setting at least twice yearly. Retitration sleep studies should be scheduled periodically, especially if the child has experienced a significant change in weight.

Our pediatric sleep disorders center managed Megan’s care for several years, and we paid close attention to her overall growth and development with appropriate interventions as necessary. She soon will be transitioning to a sleep specialist who follows adult patients. •

For children with allergic rhinitis, the PAP machine should be placed on the bedside table rather than the floor, to avoid the entry of dust into the system. To manage the effects of dry air, provide humidity.