# Evaluation and testing for obstructive sleep apnea

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Obstructive sleep apnea is a common condition with significant social, neurophysiologic, and cardiovascular morbidity as well as increased risk of mortality. Identification of patients who require sleep testing requires a high index of suspicion. The goal of testing is to identify disease, to assess its severity, and, in many cases, to initiate treatment. The common gold standard of diagnosis is attended in-lab nocturnal polysomnography. Many alternatives have been suggested, but none has been adequately evaluated or widely accepted in this country. Criteria for selecting patients for testing depend on the spectrum of the disease that is being treated and assessment of risk factors. It is clear that as the spectrum of disease treated widens to include a larger population of patients, methods of testing will need to change. Curr Opin Otolaryngol Head Neck Surg 2000, 8:199–205 

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**Abbreviations**

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| **CPAP** | continuous positive airway pressure |
| **OSA** | obstructive sleep apnea |
| **RDI** | respiratory disturbance index |
| **UARS** | upper airway respiratory syndrome |

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## Prevalence and consequences of sleep-disordered breathing

Obstructive sleep apnea (OSA) syndrome and snoring are common disorders. Performing sleep testing on all individuals who snore is both unnecessary and unrealistic. Criteria determining who needs sleep testing and the appropriate type of testing required continue to evolve. The Wisconsin Sleep Cohort study estimated that 4% of middle-aged men and 2% of middle-aged women have obstructive sleep apnea based on a definition of both a respiratory disturbance index (RDI) of greater than 5 events per hour and the presence of excessive daytime sleepiness [1]. The actual prevalence varies, and estimates vary according to the definition used for OSA and according to the population studied. In the Wisconsin Cohort, which studied middle-aged employees of the state of Wisconsin, prevalence changed when the OSA definition was based solely on polysomnographic parameters [1]. Using a RDI of greater than 5 events per hour, sleep apnea was present in 24% of men and in 9% of women. When 15 events per hour was used as a definition, prevalence was 9% of men and 4% of women. Other studies have found similar results. Population-based cross-sectional studies of Australian middle-aged men demonstrated that 10% of men had an RDI of greater than 10 events per hour [2]. Comparative data has been lacking. Now, cross-sectional data in Italian women using similar definitions to other studies also identifies a 10% prevalence [3•]. In women, although snoring was correlated to apnea, it did not necessarily indicate the presence of sleep apnea. Fifty percent of women who snored severely on objective testing demonstrated no evidence of sleep apnea as measured by RDI. Men and women manifest OSA differently.

Obstructive sleep apnea significantly impacts quality of life, health care utilization, medical morbidity, and mortality. Peker *et al.* [4] identified an independent association between coronary artery disease and OSA. In patients with and without coronary artery disease matched for age, sex, and body mass index, OSA was more prevalent in the coronary artery disease group (OSA defined as RDI greater than 10 events per hour). The odds ratio for OSA (3.0, 95% CI of 1.2–7.5) was similar to odds ratios of other risk factors such as current smoking (OR = 8.1, 95% CI 2.2–29), diabetes (OR = 4.2, 95% CI 1.1–16.1), and hypertension (OR = 1.5, 95% CI 0.7–3.2). Zamarron *et al.* [5•] presented adjusted relative increased risk of myocardial infarc-

tion in snorers compared with that in nonsnorers (3.08, 95% CI 1.01–9.46). Similarly, snoring was associated with an increased risk of mortality. The combination of snoring and excessive daytime sleepiness was associated with significantly increased mortality in men (OR = 2.7, 95% CI 1.6–4.5). When adjusted for body mass index, hypertension, cardiac disease, and diabetes, relative mortality risk was still increased by snoring and excessive daytime sleepiness (RR = 2.2, 95% CI 1.3–3.8). Mortality risks may not be increased in the elderly. Foley *et al.*, [6] in a study of Japanese American men older than 70 years of age, were unable to associate any symptoms of OSA with increased risk of mortality. These data may have important implications. The Sleep Heart Health Study, which is attempting to evaluate medical morbidity and OSA, is disproportionately weighted toward the elderly.

Obstructive sleep apnea syndrome has been associated with cerebral vascular disease and stroke. Friedlander *et al.* [7•] studied carotid atheromas on panoramic radiographs in patients with OSA and age-matched controls. Carotid atheromas were significantly more prevalent in patients with OSA (22% vs 3.7%, $P = 0.0079$), particularly in patients with Type II diabetes mellitus.

Race is a risk factor with obstructive sleep apnea syndrome. A population-based study of children identified black race, obesity, and sinus symptoms as significant risk factors for the development of sleep apnea [8••]. In adults, race correlates with severity of disease. Asian American patients, on presentation, were found to have significantly worse obstructive sleep apnea than matched white patients [9]. Asian patients were more likely to have a RDI greater than 50 events per hour, oxygen desaturation less than 70%, and more abnormal esophageal pressure measures during sleep. Increased severity was independent of age, sex, and body mass index. More severe OSA was present despite equivalent snoring symptoms and Epworth Sleepiness Scores (ESS).

Obstructive sleep apnea has been associated with increased health care utilization before the diagnosis and with decreased utilization after the diagnosis and treatment. Ronald *et al.* [10••] demonstrated that OSA patients use approximately twice as many health care services in the 10 years before their diagnosis and experienced more overnight hospitalizations. Bahammam *et al.* [11•] demonstrated that patients adhering to treatment have a decrease in the number of hospital stays and physician claims compared with patients untreated or not adhering to treatment. Direct health care costs among OSA patients are higher than those in age-matched and sex-matched control subjects [12]. This increase in health care cost was significantly related to the apnea hypopnea index and apnea severity. It was estimated that in the United States, untreated sleep apnea may cost 3.4 billion dollars in additional medical costs. It remains unknown whether medical savings would occur after treatment.

Sleep apnea has been associated with an increased risk of motor vehicle accidents. Patients with sleep-disordered breathing have significantly more motor vehicle accidents than those without. George and Smiley [13] retrospectively reviewed a 4-year database from the Ministry of Transportation of Ontario for OSA patients as well as age-matched and sex-matched controls. Patients with OSA had a significantly higher rate of accidents per year ($0.09 \pm 0.14$ vs $0.07 \pm 0.14$; $P < 0.05$). Obstructive sleep apnea patients also had twice as many citations as controls. Differences were entirely accounted for by an increased accident rate in OSA patients with severe disease (apnea hypopnea index > 40 events per hour). Mild OSA did not contribute to risk in this study. Identifying these patients at risk continues to be a problem. Hakkanen *et al.* [14] identified blink duration as a significant indicator of driver sleepiness in professional bus drivers. Blink duration correlated to sleepiness as measured in the maintenance of wakefulness test. Blink duration also decreased to normal after successful treatment of OSA. Whether this test would help to identify patients at risk or to alter driving risk is unknown.

## Respiratory events and arousals

Defining pathologic respiratory events on polysomnography may be difficult. Although there are uniform and readily identifiable criteria for obstructive apneas, defining hypopneas continues to be controversial. Bennett *et al.* [15] compared electroencephalogram (EEG) arousals, autonomic arousals, and body movement arousals with subjective and objective measures of sleepiness. Body movement and index was the marker of arousal that was the best predictor of nasal continuous positive airway pressure (CPAP) responsiveness, improvement in subjective sleepiness, and improvement in objective sleepiness. Although EEG arousals were associated with improvement in Epworth Sleepiness Score and objective sleepiness, they did not predict clinical outcome or nasal CPAP compliance. It is interesting and unexplained why only non-EEG measures were predictive. Arousals associated with major body movements or significant oxygen desaturations vary with respiratory events and may be speculated to be physiologically different than those without [16]. Fietze *et al.* [17] compared hypopneas and nonapneic respiratory events with EEG arousal to hypopneas and respiratory events with body movement. Respiratory events of both types were decreased by nasal continuous positive airway pressure. Multiple sleep latency improved in association with this reduction.

Tsai *et al.* [18•] evaluated hypopneas, comparing those with a greater than 4% decrease in oxygen desaturation to hypopneas with EEG microarousals (3 seconds). Excellent correlation existed between both definitions of hypopnea ($r > 0.98$). Because arousal-based scoring for hypopneas only resulted in small changes in apnea hypopnea index, both definitions may be clinically equivalent. If both are equivalent, this study provides support that respiratory studies that do not measure sleep may be valid measures of hypopnea. Those authors postulated that even small differences in criteria may have important influences when devices are used in population-based studies.

Arousals and micro-arousals are scored differently by different observers. Drinnan *et al.* [19] compared arousal scoring between observers and found that agreement was moderate to poor between observers (Kappa = 0.47). Agreement was especially poor during light sleep stages (Kappa = 0.28). For 50% of events, agreement was poor. During light sleep stages (stages 1 and 2), 70% of events demonstrated poor agreement among observers. This has significant implications when criteria for hypopneas includes the presence or absence of arousals to define events. There is potential for significant variability among scorers. Such variability must be considered when evaluating the results of sleep studies. Referring physicians need to know and understand how different sleep labs score studies.

Arousals and respiratory event indices correlate poorly with sleepiness. This reduces the value of sleep testing. The reason for this poor correlation is unknown. Stradling *et al.* [20] used neural network processing of EEG during respiratory events to evaluate effects on sleep. There were considerable interindividual and interevent differences in the degree of sleep disruption. Standard arousal definitions did not observe similar differences. Failure of traditional methods of scoring and staging sleep may miss critical differences that determine sleepiness.

Loube and Andrada [21] evaluated respiratory events in patients with the upper airway resistance syndrome (UARS) compared with patients with OSA. Patients differed in oxygen desaturation and apnea hypopnea indices; however, UARS patients were found to not differ in arousals or esophageal pressure manometry compared with OSA patients. Because increased esophageal pressure is a key pathophysiologic measure in OSA, the similarity in UARS and OSA supports the concept that the two disorders are equivalent. Greater efforts may be needed to identify and treat patients with UARS.s

Upper airway resistance syndrome was reviewed in detail by Exar and Collop, [22•] with several methods of diagnoses discussed, including using esophageal pressures and nasal pressure cannulas. Twenty percent alterations in esophageal pressure from baseline were thought to be reflective of respiratory events. Alternatively, using the square root of the pressure signal from nasal cannulas has been shown to be proportional and closely reflective of measures made by pneumotachographs. Finally, measures from nasal pressure cannulas accurately reflect the shape of the airflow signal. Flattening of the airflow signal during an inspiratory breath is reflective of airflow limitation and increased upper airway resistance. Novel methods of measuring pulse transit time may be accurate measures of respiratory events [23].

## Diagnostic testing

Is attended polysomnography with measures of sleep, respiration, and leg movements necessary? Some studies question the need for measures of sleep, others question the need for attended technicians, and some question both. The consensus of sleep physicians is that complete attended nocturnal polysomnography remains the gold standard of sleep testing. Yet, unattended polysomnography is increasingly used. Concerns about the tests range from accuracy, technical failure rate, ability to titrate nasal CPAP, validity in identifying other nonapneic sleep disorders, and use by physicians not trained in diagnosing and treating sleep disorders. Advocates of such testing question the need for full polysomnography to diagnose OSA, which is the test's primary indication.

There are four main reasons to perform polysomnography. First, in patients at risk for sleep-disordered breathing, the study should confirm the presence or absence of OSA or other sleep disorders. Several factors help determine the measured sensitivity of sleep testing, including the accuracy of the test and the prevalence of the disease in the population being studied. In populations with a high prevalence of disease, less sensitive measures may perform equally to more sensitive methods. In lower risk populations, potential differences may be observed when tests are compared. The second goal of sleep testing is to identify or exclude coexisting sleep or respiratory disease. This may include central sleep apnea, periodic leg movements, or hypoventilation. Third, sleep studies measure disease severity. Often the severity of OSA will determine treatment recommendations. For medical devices such as nasal CPAP, decisions may be straightforward (i.e., to treat or not to treat). Patients with profoundly severe OSA may warrant urgent intervention. Severity of disease is very important when deciding on the aggressiveness of surgery. Finally, the fourth goal of sleep studies is to initiate treatment (usually with nasal CPAP). During this portion of

the test, the sleep technician traditionally adjusts CPAP pressure to maximize the reduction in RDI. Nasal CPAP titrations may be separate complete night studies or CPAP may be titrated in the second half of a diagnostic study (i.e., "split night" study). Attended sleep studies also provide the opportunity for technicians to educate and reassure patients about nasal CPAP.

Various types of sleep tests have been used to diagnose OSA. Fry *et al.* [24] used full polysomnography in a nonattended home setting. Respiratory outcomes were equivalent in nonattended home and attended sleep lab studies. Nonattended studies also are being performed in the Sleep Heart Health Study. White *et al.* [25] performed nonattended home studies with measures of both sleep and respiration compared with those from attended polysomnography. No differences were observed.

## Oximetry

Multiple studies have attempted to identify OSA patients using oximetry [26•–32]. Oximetry accurately identifies events associated with oxygen desaturation. Oximetry is limited because many respiratory events are associated with no or only minor desaturations. Oximetry when using a 4% desaturation criteria to define events is highly specific; however, sensitivity at this level is poor. Changing the criteria to improve sensitivity lowers specificity. A common indication for testing is in the low-risk OSA patient who only has symptoms of snoring. If the goal of the test is to exclude clinically significant OSA, a highly sensitive test (with few false-negative results) is desired. Oximetry has unacceptable sensitivity in most populations tested. In these studies, a greater than 10% false- negative rate has been encountered [27]. For patients who wish to pursue treatment only for snoring, this may be unacceptably high. Oximetry has been used to identify high-risk patients who subsequently proceeded to use nasal CPAP. Defining an abnormal study as one with an oxygen desaturation less than 90%, oximetry tests used by Gyulay *et al.* [31] were 100% sensitive in identifying CPAP use. Specificity, however, was only 51%. In low-risk patients (pretest probability 30%), oximetry's prediction of CPAP use had a positive predictive value of 83%. In high-risk OSA patients (pretest probability greater than 50%), the positive predictive value was more than 90%. Used for patients who will only go to nasal CPAP therapy, oximetry was useful in the high-risk but not in the low-risk patients. Epstein and Dorlac [26•] analyzed the cost effectiveness of using screening oximetry for treating sleep apnea. Two patterns of oximetry were analyzed, including a "deep" pattern with greater than 4% change in oxygen saturation and a "fluctuating" pattern with repetitive short duration fluctuation. The fluctuating pattern was more sensitive, and the deep pattern was more specific. Cost analysis showed that $4,290 would be saved per 100 patients evaluated; however, 61% of patients with normal oximetry had treatable conditions detected by polysomnography but missed by oximetry. The authors advocated savings be achieved with increased utilization of other diagnostic algorithms such as split night polysomnography, with both diagnostic and therapeutic portions performed.

An alternative screening test to oximetry is "snore tapes" [33•]. These commonly have been used as an abbreviated test to identify patients at risk for obstructive sleep apnea syndrome. These tapes have been evaluated in children. In a study by Lamm et al, [33•] snore tapes had a sensitivity of 46% and a specificity of 83%. The authors' findings were that a home audiotape can be suggestive of OSA but not sufficiently specific to reliably distinguish primary snoring from OSA.

## Cardiorespiratory tests for obstructive sleep apnea

Many devices have been assessed to identify OSA [34]. Most validation studies have been performed in laboratory environments. Many of the devices are intended for testing in nonattended environments such as the home, but few have been evaluated there. In the sleep lab, patients and technicians may be more familiar and comfortable with testing devices. This may affect results, making it difficult to extrapolate in lab findings to in-home use. Case series also may be biased by a high pretest probability of disease skewing sensitivity and specificity outcomes. Some evidence has indicated that sleep disordered breathing may be different in the home compared with that observed in the sleep lab. Before acceptance of home testing, respiratory studies must be validated in a nonattended setting on comparable populations. Further, the effects of home diagnostics on treatment outcome (*ie,* nasal CPAP) will need to be assessed. Establishing the diagnosis only to have the treatment fail is of little benefit.

A major change in diagnostics may be the development of reliable and accurate autotitrating nasal CPAP machines. These devices use a variety of algorithms to modify CPAP pressure to the individual patient. Algorithms and mechanisms may vary widely. Variables used to adjust CPAP pressure may include minute ventilation, snoring, and measures of airflow limitation. Not all machines are alike, with some machines having good results and others poor. Teschler *et al.*, [35] using Autoset, found that autosetting pressures were 0.84 cm less than manually titrated pressures. Residual respiratory disturbance was equal with both fixed and autoset. During an 8-month follow-up period, respiratory outcomes between autosetting and fixed setting nasal

CPAP remained stable. Juhasz *et al.* [36] raised concerns about autotitrating CPAP machines using the VITAPAP device. This device resulted in an increase in central apneas and episodic hypoxia patients as well as increasing ectopy in patients who demonstrated ectopic events before treatment. It was recommended that auto CPAP machines be avoided in patients with underlying cardiovascular disease. Ayappa *et al.* [37•] evaluated change in airflow with acute pressure decreases with auto CPAP. They found that airflow limitation preceded and occurred without the presence of snoring in more than 64% of events recorded. This indicates that auto CPAP algorithms should depend predominantly on airflow limitation and not snoring sounds if they are to avoid and prevent apneic events rather than "creating" and reacting to events.

## Identification of patients at risk

The definition of OSA requires both sleep disordered breathing and symptoms. Testing established the diagnosis on objectively measured breathing abnormalities. Symptoms alone, however, have very low predictive value in identifying and diagnosing OSA patients [38]. In a group of 600 patients evaluated by sleep physicians, subjective clinical impression was only 60% sensitive and 63% specific in identifying patients with an apnea–hypopnea index of greater than 10 events per hour. Subjective clinical impression is not sufficient to reliably identify patients with or without obstructive sleep apnea syndrome. Maislin *et al.* [39] developed a multivariable apnea risk index (MAP index). Symptoms of loud snoring, breathing cessation, snorting, and gasping were useful in predicting OSA. Predictive value was improved by including body mass index and age. Tami *et al.* [40•] evaluated 94 patients from the community for snoring. In this group, there was a very high prevalence of OSA (72%). Only snoring, struggling to breathe, and observed ventilatory pauses were associated with OSA. Symptoms of headache, sleepiness, and nasal symptoms did not discriminate apnea. Pradhan *et al.* [41] prospectively compared algorithms using polysomnography, screening questionnaires, and oximetry in patients seen for evaluation of snoring. Using a Medicare 1995 fee schedule and a 35–40% prevalence rate in the population, estimated cost savings compared with universal polysomnography were only $80 per patient using questionnaires and actually increased by $60 with oximetry.

## Physical exam/clinical features

The presence of OSA has been associated with multiple structural abnormalities involving both skeletal and soft tissues. A strong association has been made with obesity and body mass index. An even stronger correlation exists in patients with increased neck circumference, presumably due to factors including increased cervical adiposity. Mortimore et al. [42] demonstrated that patients with OSA have significantly greater neck fat deposition when compared with body mass index-matched nonsnoring and non-OSA control subjects. Body mass index was evaluated by Rollheim *et al.* [43]. Both apnea severity and level of obstruction measured manometrically during sleep were related to body mass index. Apnea severity increased as the site of pharyngeal obstruction shifted to a lower level of the pharynx. In almost all patients with a body mass index of greater than 30, predominant levels of obstruction were in the lower pharynx.

An ongoing study of untreated subjects with OSA demonstrated that more than 30% had worsening or new onset of sleep disordered breathing during a 10-year period. No relation was found in this group between age, weight gain, or smoking [44•].

Epworth Sleepiness Scores in the Sleep Heart Health study of greater than 11, which were thought to be pathologic, were present in only 21% of patients with RDIs of less than 5 events per hour but were present in 35% of patients with more than 30 events per hour. Higher Epworth Sleepiness Scores also were associated with increased snoring scores even in the absence of OSA (i.e., RDI < 5) [45•].

The effects of body mass index on outcomes were evaluated by Sampol *et al.* [46]. In 216 overweight patients, 24 were cured by weight loss alone and reevaluated, on average, 7.5 years later. Only 13 patients maintained their weight, and in 6 of these patients, OSA had recurred to severe levels. Obstructive sleep apnea also had recurred in 8 of the 11 patients who had regained weight. Ultimately, only 10 of 216 patients were successfully treated during the long term with weight-loss therapy.

Friedman *et al.* [47••] evaluated physical findings that may predict apnea and found that Mallampati grade, tonsil size, and body mass index were reliable predictors of OSA. Not only was this model predictive of OSA, but it also correlated with OSA severity. Similarly, Woodson and Naganuma [48] demonstrated that abnormal results of passive supine endoscopy correlated and were predictive of RDI and airway size as measured on cephalometric radiographs. Risk factors for sleep disordered breathing in children have been evaluated in a cross-sectional study [8••]. Significant predictors of OSA were black race, obesity, sinus problems, and observed wheezing. Rosen [49] evaluated traditional criteria to predict OSA in children. Tonsillar hypertrophy increased the likelihood of OSA. Neither gender nor obesity increased the likelihood of diagnosis. Symptoms of daytime sleepiness or tiredness were no more

common in patients with apnea than in those children without apnea. In adults, OSA symptom score based on symptoms of nightly snoring, observed struggling, observed breath holding, tiredness, or daytime sleepiness increased predictive value [41]. However, the positive predictive value was only 76%, with a negative predictive value of 67%. Fifty-five percent of patients with OSA had an indeterminate sleep symptom score. Benbadis *et al.* [50•] evaluated sleepiness in a population presenting for driver's registration. Twenty-six percent of the population demonstrated an Epworth Sleepiness Score of 10 or more. Measures of sleepiness continue to be problematic. Benbadis *et al.* [51•] demonstrated that Epworth Sleepiness Scores did not correlate to mean sleep latency as measured on the multiple sleep latency test.

## Conclusion

Patient evaluation and testing for OSA is evolving. Evidence has accumulated that OSA is a common disorder with important medical consequences that warrant treatment. Depending on the criteria used to define the disease, risk factors that identify patients will vary. Diagnosing patients with severe apnea is straightforward in most cases. It is patients with mild disease and UARS that pose difficulties. The diagnostic gold standard continues to be polysomnography. Although a variety of diagnostic devices have been "validated" in controlled laboratory situations, few have been validated in nonattended settings where their use is intended. To be useful, devices must do more than identify high-risk patients for OSA. Data indicates that oximetry offers no benefits in cost savings to diagnose OSA in that it lacks sensitivity in identifying low-risk patients. With the advent of new autotitrating nasal CPAP devices, the need for in-lab CPAP titrations will be lessened, and the use of alternative diagnostic devices may be broadened. Such testing is still required because using symptoms to screen for sleep apnea is inaccurate.

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