



CIGNA MEDICAL COVERAGE POLICY

The following Coverage Policy applies to all plans administered by CIGNA Companies including plans administered by Great-West Healthcare, which is now a part of CIGNA.

**Subject Obstructive Sleep Apnea
Diagnosis and Treatment
Services**

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INSTRUCTIONS FOR USE

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Coverage Policy

Diagnostic Testing

CIGNA covers adult split-night in-laboratory polysomnography (PSG), in which the initial diagnostic portion of the PSG is followed by positive airway pressure (PAP) titration, as medically necessary when BOTH of the following criteria are met:

- signs/symptoms consistent with obstructive sleep apnea (OSA) (e.g., loud snoring, awakening with gasping or choking, excessive daytime sleepiness, observed cessation of breathing during sleep)
- apnea/hypopnea index (AHI) of 40 or higher during initial diagnostic portion of split-night study, or AHI > 20 with symptoms indicative of significant OSA (e.g., repetitive obstructions, significant oxygen desaturation)

CIGNA covers adult full night in-laboratory polysomnography (PSG) as medically necessary for ANY of the following indications:

- confirmation of obstructive sleep apnea (OSA) in individuals with signs/symptoms consistent with OSA (e.g., loud snoring, awakening with gasping or choking, excessive daytime sleepiness, observed cessation of breathing during sleep)
- titration of positive airway pressure (PAP) therapy when initial PSG confirms the diagnosis of OSA and PAP is warranted

- PAP titration following a split-night PSG, when the PAP titration portion of the original study was insufficient (e.g., less than three hours of titration or failure to effectively eliminate respiratory events)
- individual previously diagnosed with OSA who exhibits persistent snoring or other symptoms of sleep disordered breathing despite therapy
- confirmation of therapeutic benefit following final adjustment of mandibular repositioning appliance (MRA)

CIGNA covers pediatric in-laboratory polysomnography (PSG) as medically necessary for ANY the following indications:

- differentiation of benign or primary snoring from pathological snoring
- evaluation of disturbed sleep patterns, excessive daytime sleepiness, cor pulmonale, failure to thrive, or polycythemia unexplained by other factors or conditions
- when physician is uncertain whether clinical observation of obstructed breathing is sufficient to warrant surgery
- to determine whether child needs intensive postoperative monitoring following adenotonsillectomy or other pharyngeal surgery
- child previously diagnosed with OSA who exhibits persistent snoring or other symptoms of sleep disordered breathing despite therapy
- titration of continuous positive airway pressure (CPAP) levels and periodical reevaluation of the appropriateness of CPAP settings when indicated by growth-related changes

CIGNA covers a home/portable sleep study as medically necessary as an alternative to PSG for the diagnosis of OSA in an adult when ALL of the following criteria are met:

- Type II or Type III device is used, with the ability to record, at a minimum, ventilation (at least two channels of respiratory movement, or respiratory movement and airflow); electrocardiography or heart rate; and oxygen saturation
- Individual meets **ANY** of the following criteria:
 - High pretest probability of OSA (e.g., loud snoring, awakening with gasping or choking, excessive daytime sleepiness, observed cessation of breathing during sleep)
 - OSA is suspected and in-laboratory PSG is not possible
 - Diagnosis of OSA has been established, therapy has been initiated, and response to treatment is to be evaluated
- No significant co-morbid conditions that could impact the accuracy of the study (e.g., moderate to severe pulmonary disease, neuromuscular disease, congestive heart failure)
- No sleep disorders other than OSA are suspected (e.g., central sleep apnea, periodic limb movement disorder, insomnia, parasomnias, circadian rhythm disorders, narcolepsy)

CIGNA does not cover home/portable sleep studies for the diagnosis of OSA in children because it is considered experimental, investigational or unproven.

CIGNA does not cover in-laboratory or home/portable sleep studies for any of the following because these indications are considered experimental, investigational or unproven (this list may not be all-inclusive):

- chronic lung disease
- circadian rhythm disorders
- depression
- seizures in the absence of symptoms of sleep disorder
- transient or chronic insomnia
- insomnia associated with psychiatric disorders

CIGNA covers multiple sleep latency testing (MSLT) or maintenance of wakefulness testing (MWT) as medically necessary for the evaluation of individuals with suspected narcolepsy when other sleep disorders have been ruled out by prior PSG.

CIGNA does not cover MSLT or MWT for the diagnosis of OSA because it is considered experimental, investigational or unproven.

CIGNA does not cover the following devices/procedures for the diagnosis of OSA or other sleep disorders because they are considered experimental, investigational or unproven (This list may not be all-inclusive):

- Watch PAT™
- SleepStrip™
- Actigraphy

Nonsurgical Treatment

Coverage for continuous positive airway pressure (CPAP), PAP with expiratory pressure relief (e.g., C-Flex [Respironics, Inc., Murrysville, PA]), auto-titrating positive airway pressure (APAP), and bi-level positive airway pressure (BPAP) devices is subject to the terms, conditions and limitations of the applicable benefit plan's Durable Medical Equipment (DME) benefit and schedule of copayments. Please refer to the applicable benefit plan document to determine benefit availability and the terms, conditions and limitations of coverage. Under many benefit plans, coverage for DME is limited to the lowest-cost alternative.

If coverage for positive airway pressure (PAP) devices is available, the following conditions of coverage apply.

CIGNA covers CPAP with or without a humidifier as medically necessary for the treatment of OSA in adults when EITHER of the following criteria is met:

- apnea/hypopnea index (AHI) \geq 15 as documented by polysomnography (PSG)
- AHI \geq 5 and $<$ 15 as documented by PSG, when accompanied by symptoms of OSA (e.g., excessive daytime sleepiness, impaired cognition, mood disorders or insomnia) or when individual has hypertension, ischemic heart disease or history of stroke

CIGNA covers CPAP with or without a humidifier as medically necessary for the treatment of OSA in children when ALL of the following criteria are met:

- OSA diagnosis established by PSG
- child weighs 30 kilograms (66 pounds) or more
- adenotonsillectomy has been unsuccessful or is contraindicated, or when definitive surgery is indicated but must await complete dental and facial development

CIGNA covers PAP with expiratory pressure relief (e.g., C-Flex [Respironics, Inc., Murrysville, PA]), or auto-titrating PAP (APAP) with or without a humidifier as medically necessary for the treatment of OSA for individuals who meet the criteria for CPAP but demonstrate intolerance, either at the time of CPAP titration or during an appropriate trial.

CIGNA covers bi-level positive airway pressure (BPAP) with or without a humidifier as medically necessary for the treatment of OSA for individuals with coexisting central hypoventilation or for individuals who require, but prove intolerant to, high pressures of CPAP, PAP with expiratory pressure relief (e.g., C-Flex) or APAP.

CIGNA covers ANY ONE of the following interfaces for use with CPAP, PAP with expiratory pressure relief (e.g., C-Flex), APAP, or BPAP as medically necessary:

- nasal mask
- nasal pillows/prongs
- full face mask
- Oracle™ Oral Mask (Payne & Raykel Healthcare, Irvine, CA)

CIGNA does not cover an interface consisting of a boil and bite mouthpiece connected to nasal inserts (e.g., CPAP PRO® [Stevenson Industries, Inc., Simi Valley, CA]) because it is considered experimental, investigational or unproven.

Coverage for oral appliances may be subject to the terms, conditions and limitations of the applicable benefit plan's External Prosthetic Appliances and Devices (EPA) or Durable Medical Equipment (DME) benefit and schedule of copayments. Please refer to the applicable benefit plan document to determine benefit availability and terms, conditions and limitations of coverage

If coverage for oral appliances is available, the following conditions of coverage apply.

CIGNA covers a tongue-retaining device or a mandibular repositioning appliance, also referred to as mandibular advancement appliance or mandibular advancement splint, as medically necessary for individuals with mild or moderate OSA when EITHER of the following criteria is met:

- apnea/hypopnea index (AHI) \geq 15 and $<$ 30, as documented by polysomnography (PSG)
- AHI \geq 5 and $<$ 15 as documented by PSG, when accompanied by symptoms of OSA (e.g., excessive daytime sleepiness, impaired cognition, mood disorders or insomnia) or when individual has hypertension, ischemic heart disease or history of stroke

CIGNA covers a tongue-retaining device or a mandibular repositioning appliance as medically necessary for individuals with severe OSA (i.e., AHI \geq 30) who are unwilling or unable to comply with PAP treatment.

Over-the-counter (OTC) oral appliances that can be obtained without a prescription are excluded under many benefit plans and therefore are generally not covered. In addition, OTC oral appliances are not considered medically necessary.

Surgical Treatment

CIGNA covers tonsillectomy and/or adenoidectomy as medically necessary for the treatment of OSA, as diagnosed by polysomnography (PSG).

CIGNA covers uvulopalatopharyngoplasty (UPPP) as medically necessary for the treatment of OSA when ALL of the following criteria are met:

- presence of narrowing or collapse of the retropalatal region
- criteria for PAP met and individual has proved intolerant to or failed a trial of PAP
- consideration has been given to use of mandibular repositioning appliance (MRA) or tongue-retaining appliance

CIGNA does not cover uvulectomy as a stand-alone procedure for the treatment of OSA because it is considered experimental, investigational or unproven. (Note: this Coverage Policy is not intended to address uvulectomy performed for other indications (e.g., acute inflammation/angioedema of the uvula).

CIGNA covers inferior sagittal mandibular osteotomy (ISO) and genioglossal advancement with hyoid myotomy and suspension (GAHM), with or without UPPP, as medically necessary for the treatment of OSA when ALL of the following criteria are met:

- presence of retropalatal and retrolingual obstruction
- criteria for PAP met and individual has proved intolerant to or failed a trial of PAP
- consideration has been given to use of mandibular repositioning appliance (MRA) or tongue-retaining appliance

CIGNA covers mandibular and maxillary osteotomy and advancement as medically necessary for the treatment of OSA when BOTH of the following criteria are met:

- criteria for PAP met and individual has proved intolerant to or failed a trial of PAP
- individual has failed prior less invasive surgical procedures **OR** has craniofacial skeletal abnormalities that are associated with a narrowed posterior airway space and tongue-base obstruction

CIGNA covers tracheostomy as medically necessary for the treatment of OSA when other medical and surgical options do not exist, have failed or are refused, or when deemed necessary by clinical urgency.

Additional Procedures/Services

CIGNA does not cover any of the following procedures or services for the treatment of OSA because they are considered experimental, investigational or unproven:

- laser-assisted uvulopalatoplasty (LAUP)
- cauterly-assisted palatal stiffening operation (CAPSO)
- Pillar™ Palatal Implant System
- radiofrequency volumetric tissue reduction (RFVTR) of the soft palate, uvula, or tongue base (e.g., Coblation®, Somnoplasty®)
- tongue-base suspension using the Repose™ Bone Screw System
- Provent™ Professional Sleep Apnea Therapy Device
- electrosleep therapy
- injection Snoreplasty
- atrial overdrive pacing

CIGNA does not cover treatment of upper airway resistance syndrome (UARS) using any of the methods of treatment in this policy, including CPAP, BPAP and APAP, because they are considered experimental, investigational or unproven.

CIGNA does not cover the treatment of snoring by any method because it is not considered medically necessary.

General Background

Obstructive sleep apnea (OSA) is a treatable form of sleep disordered breathing characterized by repetitive obstruction of the upper airway resulting in oxygen desaturation and arousal from sleep. Apnea is defined as a drop in airflow of 90% or more, lasting 10 seconds, and is considered obstructive if there is effort to breathe during the episode. There is less consensus on the definition of hypopnea. The American Academy of Sleep Medicine (AASM) has proposed that hypopnea be defined as an abnormal respiratory event with at least a 30% reduction in thoracoabdominal movement or airflow as compared to baseline, lasting at least 10 seconds, with \geq 4% oxygen desaturation. This definition is used by the Centers for Medicare and Medicaid Services (CMS) and has been used in several key studies. Hypopnea may result in partial obstruction of the airway, and most researchers have recognized that the clinical impact of apneas and hypopneas is virtually indistinguishable (AASM Task Force, 1999, (Kushida, et al., 2005; Goroll, et al., 2006).

Sleep is generally defined by combining behavioral observation with electrophysiological recording and consists of rapid eye movement (REM) sleep and nonrapid eye movement (NREM) sleep. NREM sleep usually precedes REM sleep and is divided into four stages. Sleep is usually entered through stage I sleep, a transitional phase when eye movements become slow and skeletal muscles relax. Stage I may not be perceived as sleep, although there is less sensory awareness and mental activity becomes dream-like. Sleep-deprived individuals enter unavoidable periods of microsleep consisting of five- to ten-second bouts of stage I sleep. These episodes may have serious consequences, especially in situations that demand constant attention, such as driving a motor vehicle. Stage II and subsequent stages are perceived as sleep. Stages III and IV are referred to as slow-wave sleep or deep sleep.

OSA occurs when the patency of the nasopharyngeal airway becomes insufficient during sleep. Anatomic risk factors include nuchal obesity (cricothyroid neck circumference greater than 17 inches in men or 16 inches in

women), deviated septum, nasal polyps, enlarged uvula and soft palate, small chin with deep overbite, enlarged tonsils, and hypertrophy of the lateral pharyngeal musculature. In addition to anatomical predisposition, patients with OSA appear to be unable to maintain oropharyngeal muscle dilator activity during sleep sufficient to prevent airway collapse during the negative pressure of inspiration. Apneas and hypopneas are common during REM sleep, when muscles completely relax. When the pharyngeal muscles relax, the palate may fall backward, and relaxation of the genioglossus muscle at the base of the tongue allows the tongue to fall backward, occluding the airway. The apneic event is terminated by a brief arousal to wakefulness or a lighter stage of sleep, which is accompanied by activation of the upper airway dilator and abductor muscles and restoration of airway patency and other physiologic responses (Lattimore, et al., 2003; Mason, 2005; Goroll, et al., 2006).

Snoring is highly prevalent in adults and children, and it is also the most common symptom of OSA. Snoring that is not accompanied by an AHI ≥ 5 in adults and not associated with reports of excessive daytime sleepiness is referred to as primary snoring. Snoring that is associated with OSA, however, is generally loud and intermittent, and is accompanied by awakening with gasping or choking, sleep fragmentation, restlessness, impaired concentration, and daytime sleepiness. Daytime sleepiness is thought to be related to sleep disruption and may also be related to recurrent hypoxemia. These typical symptoms are not always present or apparent, however. It is not unusual for patients subsequently diagnosed with OSA to initially present with hypertension, arrhythmias, or heart failure. There is mounting evidence that the presence and severity of OSA is associated with increased risk of cardiovascular disease. OSA is thought to play a role in the pathogenesis of systemic hypertension and heart failure and may also be associated with acute coronary syndromes, pulmonary hypertension, arrhythmias, and stroke (Nieto, et al., 2000; Lattimore, et al., 2003; Yaggi, et al., 2005).

Diagnosis of OSA

According to the American Academy of Sleep Medicine (AASM) Practice Parameters for the Indications for Polysomnography Procedures (Kushida, et al., 2005), clinical impression alone or categorization based on symptoms alone lack the accuracy needed to diagnose sleep-related breathing disorders, and objective testing is needed. A diagnosis of OSA should be based on evaluation of the presence of symptoms of OSA and the number of episodes of apnea and hypopnea. The frequency of apneas and hypopneas per hour of sleep is expressed as the apnea-hypopnea index (AHI). The AHI is the most commonly used reference to quantify OSA. The term respiratory disturbance index (RDI) has at times been used interchangeably with AHI, although RDI may also include a measure of respiratory effort-related arousals.

The AASM Task Force report, *Sleep-Related Breathing Disorders in Adults: Recommendations for Syndrome Definition and Measurement Techniques in Clinical Research* (1999), states that the diagnosis of OSA is made when overnight monitoring demonstrates five or more obstructed breathing events per hour during sleep and the patient has:

- excessive daytime sleepiness not better explained by other factors, and/or
- two or more of the following symptoms not better explained by other factors:
 - choking or gasping during sleep
 - recurrent awakening from sleep
 - unrefreshing sleep
 - daytime fatigue
 - impaired concentration

According to the same AASM recommendations, OSA severity is determined by the severity of daytime sleepiness and of sleep-related obstructive breathing based on overnight monitoring. A severity level is specified for each component. The overall rating of severity for OSA is based on the most severe component. The AASM severity criteria are as follows:

Sleepiness:

- **Mild:** Unwanted sleepiness or involuntary sleep episodes occur during activities that require little attention, such as watching television, reading, or traveling as a passenger. Symptoms produce only minor impairment of social or occupational function.
- **Moderate:** Unwanted sleepiness or involuntary sleep episodes occur during activities that require some attention, such as concerts, meetings or presentations. Symptoms produce moderate impairment of social or occupational function.

- Severe: Unwanted sleepiness or involuntary sleep episodes occur during activities that require more active attention, such as eating, conversation, walking, or driving. Symptoms produce marked impairment in social or occupational function.

Sleep related obstructive breathing events:

- Mild: ≥ 5 and < 15 events per hour
- Moderate: ≥ 15 and ≤ 30 events per hour
- Severe: > 30 events per hour

AHI levels greater than five may be present in 24% of men and 9% of women. Similarly, AHI levels of greater than 15 may be found in 9% of men and 4% of women. Using the definition of OSA above, which requires sleepiness as a second defining criterion, reduces the estimate of incidence to 4% of men and 2% of women. Despite the increasing awareness of OSA, most cases remain undiagnosed (Mason, 2005).

Upper airway resistance syndrome (UARS) is a lesser known disorder associated with frequent snoring-related arousals and reports of insomnia and/or excessive daytime sleepiness. UARS is not associated with the typical findings of apnea and hypopnea demonstrated on PSG in patients with OSA, however. With UARS, nearly normal airflow is maintained by compensatory respiratory efforts, at the expense of sleep arousals. Further research is required to understand the etiology, prevalence, and appropriate treatment for UARS (Cummings, 2005; Goroll, 2008).

Polysomnography (PSG): Polysomnography is the collective process of monitoring and recording physiologic data during sleep. Full-night in-laboratory PSG is broadly accepted as the definitive diagnostic tool for evaluating OSA. Based on AASM recommendations, four levels are used to classify the complexity of technology used in the diagnosis of sleep-related breathing disorders. Polysomnography, a Type I study, requires that a technician be present and must include the following recordings at a minimum: electroencephalogram (EEG), electrooculogram (EOG), chin electromyography (EMG), airflow, arterial oxygen saturation, respiratory effort, and electrocardiogram or heart rate. Although not a required component of PSG, anterior tibialis EMG is also useful to assist in detecting movement arousals and may assess periodic limb movements which coexist with sleep-related breathing disorders in many patients. Full-night PSG is indicated for patients with signs and symptoms consistent with OSA, such as awakening with choking, intense snoring, observed cessation of breathing during sleep, and daytime sleepiness, especially with impairment of driving.

Split-night PSG, in which the initial diagnostic portion of the PSG is followed by positive airway pressure (PAP) titration, is an alternative to a full-night diagnostic PSG followed by a repeat full-night PSG for PAP titration. Split-night PSG is a reasonable option when an AHI of at least 40 is documented during a minimum of two hours of diagnostic PSG, or when the AHI is 20–40 with symptoms indicative of significant OSA (e.g., repetitive obstructions, significant oxygen desaturation). When AHI values are below 40, however, determination of PAP requirements based on split-night studies may be less accurate than measurements obtained during full-night PSG. PAP titration should be performed for more than three hours, since respiratory events can progressively worsen during the night. Elimination or near elimination of respiratory events during REM and non-REM sleep, including REM sleep with the patient in the supine position, is required for a successful split-night PSG (Kushida, et al., 2005).

A follow-up PSG for PAP titration is appropriate when the initial PSG confirms a diagnosis of OSA and PAP is warranted. A follow-up PSG is also indicated when a diagnosis of OSA is confirmed during a split-night study but the PAP titration portion of the study is insufficient (e.g., less than three hours of titration or failure to effectively eliminate respiratory events, as described above).

PSG is not indicated for the diagnosis of chronic lung disease, circadian rhythm disorders, depression, or in cases of typical parasomnias when the diagnosis is clear, for patients with seizures when no symptoms of a sleep disorder are present, or for the diagnosis and treatment of restless leg syndrome. PSG is also not indicated for the routine evaluation of transient insomnia, chronic insomnia, or insomnia associated with psychiatric disorders (Kushida, et al., 2005; Littner, et al., 2002).

Type II, III, and IV Studies

As stated above, the AASM defined four levels to classify the complexity of technology used in the diagnosis of sleep-related breathing disorders. A Type II study, or comprehensive portable polysomnography, is similar to a

Type I study (i.e., PSG), but ECG can be replaced by a heart rate monitor and a technician is not in constant attendance. In a Type III study, referred to as a cardiopulmonary study or modified portable sleep apnea testing, at least four parameters are measured. Minimum requirements include recording of ventilation (at least two channels of respiratory movement, or respiratory movement and airflow); ECG or heart rate; and oxygen saturation. Personnel are needed for preparation, but the ability to intervene is not required for all studies. A Type IV study, or continuous single or dual bioparameter recording, generally uses oximetry and may employ a second airflow assessment parameter.

This classification system is no longer entirely clear, however, because of the recent proliferation of devices that measure various parameters and do not necessarily fit into the above categories. Several devices used for portable sleep studies measure more than the one or two parameters measured by early Class IV devices, but do not satisfy the criteria for a Class III device.

Unattended/Home Sleep Studies (Type II, III, IV)

A Hayes Directory Report, Home Sleep Studies for Diagnosis of Obstructive Sleep Apnea in Adults (2008), evaluated numerous studies published between 1986 and 2008 that assessed 15 different devices (Type II, III, or IV). Most studies used laboratory-based PSG as a reference "gold standard" to evaluate the diagnostic accuracy of the device to predict a diagnosis of OSA. The diagnostic accuracy in most studies was higher for more severe cases than for mild OSA. In those with AHI cutoff points ≥ 10 to ≥ 20 , sensitivity ranged from 60–100% and specificity ranged from 76–100%. Two studies reported specificity rates of only 8.5% and 25%, however. The majority of portable recording devices provided similar AHI values to those obtained from standard PSG. Portable oximeters were generally less accurate than devices that measured a number of other parameters, especially for the detection of milder cases of OSA. Most of the devices did not provide information that could be used to diagnose other sleep disorders, such as restless leg syndrome.

The report noted that the majority of studies evaluated individuals specifically referred to a sleep laboratory for suspected OSA. The high prevalence of OSA in these study populations could make the tests appear more specific, with a higher positive predictive value, than might be seen in a broader patient population. Comparisons across studies were difficult due to the wide variation in devices used, study setting (home, sleep clinic, laboratory), and scoring method (manual or automated). In addition, some devices were evaluated on a single night and others on multiple nights. The report concluded that there is moderate to good evidence that unattended home portable recording devices, especially those capable of recording a number of relevant parameters, can provide measurements that correlate well with those provided by standard PSG. The overall diagnostic accuracy to predict the possibility of OSA was relatively good, demonstrating acceptable sensitivity and specificity. Some of the devices had not been validated in a home environment, however, and it is unclear whether similar results can be achieved in a less controlled setting. In addition, results may be inaccurate if the patient is awake during the home study, since a diagnosis of OSA is based on a threshold level of apnea/hypopnea episodes per hour of sleep.

Ghegan et al. (2006) conducted a meta-analysis to compare the accuracy of home sleep studies with laboratory PSG in the diagnosis of OSA. A total of 27 studies were included, and nine of these studies provided sufficient data to allow a comparison of the primary outcome measure, RDI. Seven additional studies provided data on one or more of the secondary outcome variables: low oxygen saturation, sleep time, rate of inadequate recordings, and cost. Fourteen different devices were used, ranging from simple two-channel devices to devices with up to seven channels. RDI values on the portable sleep studies were 10% lower on average compared to laboratory studies (odds ratio [OR] 0.90; 95% confidence interval [CI], 0.87–0.92. No significant difference in the mean low oxygen saturation was seen on portable vs. in-lab studies (OR 1.0; 5% CI, 0.94–1.10). The grand mean when combining the studies demonstrated 13% longer sleep time for laboratory tests compared to portable studies (OR, 0.87; 95% CI, 0.86–0.89). There was a significant degree of heterogeneity ($p=0.00$) regarding average sleep times. The authors stated that this result was heavily influenced by a single large study with narrow confidence intervals. Portable studies were significantly more likely to give a poor recording compared to laboratory examinations ($p=.0001$). The rate of poor recordings was not found to be related to the level of complexity (e.g., number of leads) on the portable study.

Iber et al. (2004) reported a multicenter, randomized clinical trial to compare PSG recordings obtained in the home to those obtained in a laboratory setting. Sleep Heart Health Study (SHHS) standardized PSG recording and scoring techniques were used for both settings. Sixty-four of 76 non-SHHS participants recruited from seven SHHS field sites had both a laboratory and home PSG of acceptable quality. Median sleep duration was

greater in the home than in the laboratory (375 minutes vs. 318 minutes, respectively), as was sleep efficiency (86% vs. 82%, respectively). Very small but significant increases in percentage of REM sleep and decreases in stage 1 sleep were noted in the laboratory. The median RDI with 3% desaturation was similar in both settings, with a median of 12.4 in the home and 9.5 in the laboratory. Quartile analysis of laboratory RDI showed moderate agreement with home RDI measurements. Using a cutoff of 20, analysis of mean laboratory and home RDI showed an RDI 3% above 20 was more common in the recordings performed in the laboratory than in the home, and an RDI below 20 was more common in the recordings performed in the home than in the laboratory. The authors concluded that using SHHS methodology, median RDI was similar in the unattended home and attended laboratory setting with differences of small magnitude in some sleep parameters. Differences in RDI between settings resulted in a rate of disease misclassification that is similar to repeated studies in the same setting.

The SNAP™ Test (SNAP Laboratories, Glenview, IL) originally consisted of a digital recording system with a sound collection cannula and oximetry sensor. The sound collection and airflow device was placed on the upper lip for collection of oral and nasal respiratory sound and airflow information, and an oximetry sensor was placed on a finger. The device was subsequently modified to include sensors for respiratory effort, limb movement and body position. Data is recorded during sleep and returned to the SNAP laboratory for analysis using proprietary software.

Su et al. (2004) compared SNAP testing to PSG in a consecutive series of 60 adults referred to a sleep disorder clinic. PSG and SNAP testing were performed simultaneously. In addition to measurement of oronasal airflow, oronasal sound, pulse oximetry, and heart rate, this version of the SNAP system, a Class III device, included the capability to measure respiratory effort using a chest belt. Proprietary algorithms were used to detect the presence, absence, or reduction of airflow, RDI, and an apnea profile. The SNAP system is limited to a recording time of 338 minutes. Compared to PSG as the gold standard, the sensitivity, specificity, positive predictive value, and negative predictive value for patients with an RDI ≥ 5 was 98%, 40%, 89.1%, and 80%, respectively. For patients with RDI ≥ 15 , the sensitivity, specificity, positive predictive value, and negative predictive value was 83.9%, 75.9%, 78.8%, and 81.5%, respectively. The authors noted that, as is the case with other home testing devices, SNAP testing has not been validated in a home setting, and that no conclusions can be made about the reliability of SNAP testing in the home setting until further comparative studies have been undertaken in patients where the tool is actually used in that setting and compared to PSG.

Bar et al. (2003) conducted a case series to evaluate the reliability, efficacy and reproducibility of the Watch PAT™ 100S (Respironics, Murrysville, PA) as compared to standard PSG. Watch PAT device is worn on the wrist and uses a finger-mounted probe. This Class IV device is based on the peripheral arterial tone (PAT) signal, and measures heart rate derived from the PAT signal, pulse oximetry, and actigraphy. The Watch PAT is designed to indirectly detect apneas and hypopneas by identifying surges of sympathetic activation associated with the termination of these events. This information is combined with heart rate and pulse oximetry and is downloaded to a computer for analysis by proprietary algorithms. The study included 69 consecutive patients referred to a sleep center with suspected OSA, and 33 additional healthy volunteers without complaints of snoring or daytime sleepiness. PSGs were conducted simultaneously with Watch PAT recording. PSGs were blindly evaluated with RDI scores calculated, and Watch PAT data were analyzed by a proprietary algorithm. The authors reported that the RDI levels were highly correlated across a wide range of RDI levels. However, a subset of 14 patients underwent two additional unattended home sleep studies with the Watch PAT device only, and three of these 28 studies were rejected due to technical failure.

The SleepStrip™, a Class IV device, is an OSA screening device that incorporates signal detection, acquisition and display in a disposable package. The self-adhesive device is placed on the upper lip at bedtime and adjusted until respiration is detected, as indicated by a flashing light. Two nasal thermistors and one oral thermistor produce flow signals that are processed within the SleepStrip's microprocessor (CPU). The five possible results are as follows: zero (no apneas); one (mild sleep apnea, comparable to sleep lab AHI between 15 and 24); two (moderate sleep apnea, comparable to sleep lab AHI between 25 and 39); three (severe sleep apnea, comparable to sleep lab AHI of greater than 40); and E (error in measurement).

Pang et al. (2006) conducted a prospective, nonrandomized cohort study to investigate the role of the SleepStrip in the diagnosis of OSA. Patients with suspected OSA who were scheduled for PSG wore the device at home the night after the PSG. The AHI determined by PSG was compared with the results of the SleepStrip. The sensitivity and specificity of the SleepStrip in diagnosing severe OSA when the AHI was > 40 were 33.3%

and 95%, respectively. The sensitivity and specificity of the SleepStrip when the AHI was > 25 were 43.8% and 81.3%, respectively. When the AHI was > 15, the sensitivity and specificity of the test were 54.6% and 70%, respectively. The authors concluded that the SleepStrip has a low correlation with the AHI as measured by PSG, and that further studies are needed before this device can be recommended as a screening tool for the diagnosis of OSA.

AASM: An AASM clinical guideline for the use of unattended portable monitoring (Collop et al., for the Portable Monitoring Task Force, 2007) provides the following updated recommendations based on a review of the literature and consensus:

Indications for Portable Monitoring (PM)

- Portable monitoring (PM) for the diagnosis of OSA should be performed only in conjunction with a comprehensive sleep evaluation. Clinical sleep evaluations using PM must be supervised by a practitioner with board certification in sleep medicine or an individual who fulfills the eligibility criteria for the sleep medicine certification examination.
- PM may be used as an alternative to PSG for the diagnosis of OSA in patients with a high pretest probability of moderate to severe OSA. PM should not be used in the following patient groups:
 - patients with significant co-morbid conditions that may degrade the accuracy of PM, including but not limited to, moderate to severe pulmonary disease, neuromuscular disease, or congestive heart failure
 - patients suspected of having other sleep disorders, including central sleep apnea, periodic limb movement disorder, insomnia, parasomnias, circadian rhythm disorders, or narcolepsy
 - general screening of asymptomatic populations
- PM may be indicated for the diagnosis of OSA in patients for whom in-laboratory PSG is not possible by virtue of immobility, safety or critical illness.

Technology for PM

The AASM guidelines state that the validity of a scheme based on the number of channels (i.e., Type I–IV, discussed above) is no longer clear. The Task Force chose instead to focus on the types of signals used rather than their number.

- At a minimum, the PM must record airflow, respiratory effort, and blood oxygenation. The type of biosensors used to monitor these parameters for in-laboratory PSG are recommended for use in PMs.
- The sensor to detect apnea is an oronasal thermal sensor and to detect hypopnea is a nasal pressure transducer. Ideally, PMs should use both sensor types
- Ideally, the sensor for identification of respiratory effort is either calibrated or uncalibrated inductance plethysmography
- The sensor for the detection of blood oxygen is pulse oximetry with the appropriate signal averaging time and accommodation for motion artifact

Methodology for Portable Monitoring

- Testing should be performed under the auspices of an AASM accredited comprehensive sleep medicine program with policies and procedures for sensor application, scoring, and interpretation of PM
- An experienced sleep technician, sleep technologist, or appropriately trained healthcare practitioner must perform the application of PM sensors or directly educate the patient in the correct application of sensors.
- PM devices must allow for the display of raw data for manual scoring or editing of automated scoring by a trained and qualified sleep technician/technologist. Evaluation of PM data must include review of the raw data by a board certified sleep specialist or an individual who fulfills the eligibility criteria for the sleep medicine certification examination.
- Scoring criteria should be consistent with the current published AASM standards for scoring of apneas and hypopneas.
- Due to the known rate of false negative PM tests, in-laboratory PSG should be performed in cases where PM is technically inadequate or fails to establish the diagnosis of OSA in patients with a high pretest probability.
- A follow-up visit with a physician or other appropriately trained and supervised health care provider should be performed on all patients undergoing PM to discuss the results of the test.

Agency for Healthcare Research and Quality (AHRQ): A technology assessment based on a systematic review of the literature on home diagnosis of obstructive sleep apnea-hypopnea syndrome (OSAHS) was conducted by AHRQ for the Centers for Medicare and Medicaid Services (CMS) (Trikalinos et al., 2007). Eligible studies evaluated the ability of sleep studies at baseline to predict response to CPAP treatment or CPAP use; the comparison of measurements with portable monitors and facility-based PSG, and the safety of sleep studies. Although the AHRQ review provides support for home sleep testing, the focus of the analysis is on the response to CPAP and CPAP usage, rather than on the accuracy of unattended/home testing. The technology assessment provided the following conclusions:

- Baseline AHI, and additional data obtained from PSG (oxygen saturation, apnea index, hypopnea index, and frequency of arousal) are only modestly associated with response to CPAP or CPAP use among those with high pre-test probability of severe OSAHS. This difference cannot be used, therefore, to predict CPAP use or response to CPAP in this population.
- AHI measurements from portable monitors and facility-based PSG are not interchangeable, especially in the higher end of the AHI spectrum. Substantial differences may be seen between type II monitors and facility-based PSG, and even larger differences cannot be excluded for type III monitors, and more so for type IV monitors.
- Based on limited data, type II monitors may identify AHI suggestive of OSAHS with high likelihood ratios (>10) and low negative likelihood ratios (<0.1) when the portable monitors were studied in the sleep laboratory or at home
- Type III monitors have the ability to predict AHI suggestive of OSAS with high positive likelihood ratios and low negative likelihood ratios for various AHI cutoffs in laboratory-based studies, especially when manual scoring is used. The ability of type III monitors to predict AHI suggestive of OSAHS appears to be better in studies conducted in specialized sleep units compared to studies in the home setting.
- Studies of type IV monitors that record at least three bioparameters showed high positive likelihood ratios and low negative likelihood ratios. Studies of type IV monitors that record one or two bioparameters also had high positive likelihood ratios and low negative likelihood ratios, at least for selected sensitivity and specificity pairs from ROC (receiver operating characteristic) curve analyses. As is the case with type III monitors, the ability of type IV monitors to predict AHI suggestive of OSAHS appears to be better in studies conducted in specialized sleep units compared to studies in the home setting. Conditions that effect sleep (e.g., cardiac insufficiency, COPD, obesity hypoventilation syndrome, or periodic limb movements in sleep or restless leg syndrome) may be misdiagnosed as OSAHS by monitors that do not record channels necessary for differential diagnosis.
- Manual scoring or manual editing of automated scoring appears to have better agreement with facility-based PSG compared to automated scoring in the studies that assessed this factor. In addition, automated scoring algorithms differ among the devices, and their ability to recognize respiratory events may vary.

Summary

In-laboratory PSG remains the standard test for the diagnosis of OSA. Evidence on the accuracy of unattended/home testing compared to in-laboratory PSG is limited, and most devices have not been tested in an unattended home setting. Several devices used for unattended/home testing employ proprietary software and algorithms to determine test results, and do not allow manual interpretation and scoring of raw data. Portable testing does not assess sleep architecture or staging, and therefore cannot calculate AHI based on actual sleep time. Unattended/home sleep studies using a Type II or Type III device that records, at a minimum, ventilation (at least two channels of respiratory movement, or respiratory movement and airflow), may be a reasonable alternative to in-laboratory PSG in carefully selected patients, however, when a high pretest probability of moderate to severe OSA is present; when in-laboratory PSG is not possible; or when used to evaluate the response to therapy for OSA.

Although unattended/home testing may be used to confirm a diagnosis of OSA, a follow-up in-laboratory PSG is indicated for individuals with a high pretest probability of OSA when results of home testing are negative or inadequate. In-laboratory PSG is also generally performed for titration of CPAP in patients diagnosed with OSA based on an unattended/home test. Unattended/home testing therefore does not necessarily obviate the need for follow-up in-laboratory PSG. Unattended/home testing is not recommended for individuals with significant co-morbid conditions that may impact the accuracy of portable monitoring (e.g., moderate to severe pulmonary

disease, neuromuscular disease, or congestive heart failure), or when other sleep disorders are suspected (e.g., central sleep apnea, periodic limb movement disorder, insomnia, parasomnias, circadian rhythm disorders, narcolepsy).

There is insufficient evidence in the published medical literature to demonstrate the accuracy of Class IV devices (e.g., pulse oximetry, actigraphy, Sleep Strip, Watch PAT) in the diagnosis of OSA.

Multiple Sleep Latency Test (MSLT): The MSLT is used to measure physiological sleep tendency under standardized conditions in the absence of external alerting factors. It is based on the premise that sleep latency reflects the degree of sleepiness. The patient is given four or five opportunities to sleep for up to 20 minutes at two-hour intervals during the day. The mean time to fall asleep is monitored, and it is determined whether the patient has marked sleepiness, usually defined as a mean sleep latency of less than five minutes.

The MSLT is indicated as part of the evaluation of patients with suspected narcolepsy, since the narcoleptic patient, in addition to demonstrating sleepiness, usually experiences two or more episodes of REM sleep during these naps. This is unlikely with other conditions associated with excess sleepiness. The pathophysiology of narcolepsy involves intrusion of aspects of REM sleep (e.g., muscle atonia and dreams) into periods of wakefulness. The test may also be used to evaluate patients with suspected idiopathic hypersomnia to help differentiate between this condition and narcolepsy, and to evaluate response to medications in patients with idiopathic hypersomnia or narcolepsy (Sadock, et al., 2005; Littner, et al., 2005).

The MSLT is not routinely indicated in the initial evaluation and diagnosis of obstructive sleep apnea syndrome, or in assessment of change following treatment with nasal CPAP, nor is it routinely indicated for evaluation of sleepiness in medical and neurological disorders other than narcolepsy, or for insomnia, or circadian rhythm disorders (Littner, et al., 2005).

Maintenance of Wakefulness Test (MWT): The MWT measures the ability to stay awake for a defined period of time in patients with disorders associated with excessive sleepiness.

The MWT may be indicated in the assessment of individuals in whom the inability to remain awake constitutes a safety issue, or in patients with narcolepsy or idiopathic hypersomnia to assess response to treatment with medications. Since there is little evidence linking MWT sleep latency results with risk of accidents in real world circumstances, the MWT should be considered an option to be integrated with findings from the clinical history and compliance with treatment (Littner, et al., 2005).

Actigraphy: An actigraph is a small portable device that records movement over an extended period of time and is usually worn on the wrist. Actigraphy measures movement of a limb, and although it may provide an estimate of total sleep time, it does not actually measure sleep or the subjective experience of sleep.

According to updated AASM Practice Parameters for the Use of Actigraphy in the Assessment of Sleep and Sleep Disorders (Morgenthaler, et al., 2007) actigraphy is increasingly used in sleep research and the clinical care of patients with sleep and circadian rhythm abnormalities. The practice parameters state that actigraphy provides an acceptably accurate estimate of sleep patterns in normal, healthy adult populations and in patients suspected of certain sleep disorders. The practice parameters address the use of actigraphy in patients with advanced sleep phase syndrome, delayed sleep phase syndrome, shift work disorder, jet-lag, and non-24 hour sleep/wake syndrome. Regarding OSA, the AASM practice parameters state that, when PSG is not available, actigraphy is indicated as a method to estimate total sleep time in patients with OSA, and that combined with a validated way of monitoring respiratory events, use of actigraphy may improve accuracy in assessing the severity of OSA compared to using time in bed. In recommendations for further research, the practice parameters state that additional research is needed that compares results from different actigraphy devices and the variety of algorithms used to evaluate data in order to further establish standards of actigraphy technology, and that there is a need for additional study addressing the reliability and validity of actigraphy compared to reference standards such as PSG.

There is insufficient evidence in the published medical literature to demonstrate the accuracy of actigraphy in the diagnosis or management of OSA.

Treatment of OSA

Patients diagnosed with OSA receive education regarding the pathophysiology of OSA and the impact of lifestyle modifications, including weight loss, reduced alcohol consumption, especially at bedtime, and lateral sleeping position (vs. supine). While such noninvasive measures are encouraged, particularly in the obese or those with very poor sleep hygiene, OSA does not usually resolve with these measures alone. Potential treatment options for OSA include treatment with positive airway pressure (PAP), the use of oral appliances, and surgical interventions. Treatment decisions are based on condition severity, the presence of comorbidities and complicating factors, and the patient's tolerance and response to treatment.

Positive Airway Pressure (PAP) Treatment

PAP is the most effective and widespread treatment of OSA. A flow generator delivers pressurized air into the nose and/or mouth, providing a pneumatic splint to the airway, preventing development of subatmospheric collapsing pressure. The flow generator is set to a specific pressure sufficient to maintain airway patency and overcome respiratory disturbances. The flow generator is connected to the patient via connecting tubing and an interface attached to the patient's face. PAP may be provided using continuous positive airway pressure (CPAP), auto-titrating PAP (APAP), or bi-level positive airway pressure (BPAP).

PAP Interfaces: PAP is most commonly applied using a nasal mask, or alternately, nasal pillows or prongs. An Oracle™ Oral Mask (Fisher & Paykel Healthcare, Irvine, CA) may be used as an alternative to nasal interfaces. The Oracle interface delivers pressure through the mouth rather than the nose. The type of interface used is likely to influence acceptance and adherence to PAP therapy; compliance is affected by the incidence of side effects, including claustrophobia, air leaks, pressure sores, nasal stuffiness, dry mouth and mask discomfort (Chai et al, 2009)

In a Cochrane review, Chai et al. (2009) compared the efficacy of various CPAP delivery interfaces available for the treatment of OSA (n=132). Two studies compared nasal masks with the Oracle Oral Mask and showed no significant difference in compliance at one month. There were no significant differences in any of the physiological parameters (e.g., apnea-hypopnea index, arousal index, minimum oxygen saturation), Epworth Sleepiness Scale (ESS) or symptoms of OSA. One study comparing a nasal mask to nasal pillows showed a significant difference in compliance in favor of nasal pillows (p=0.02), fewer overall adverse effects (p<0.001), and greater interface satisfaction (p=0.001). A study comparing nasal mask with face mask showed significantly greater compliance and lower ESS scores with use of a nasal mask. The nasal mask was the preferred interface in almost all patients. The authors concluded that due to the limited number of studies comparing various interface types, the optimum form of delivery interface remains unclear. Nasal pillows or the Oracle oral mask may be useful alternatives when a patient is unable to tolerate conventional nasal masks. A full-face mask, while not a first-line interface, may be used if nasal obstruction or dryness limits the use of a nasal interface.

CPAP PRO® (Stevenson Industries, Inc., Simi Valley, CA) has been proposed as an interface alternative without straps or headgear. CPAP PRO consists of a boil and bite dental appliance that is snapped in place on the upper teeth, with a small bracket extending beyond the lips to attach to a pair of nasal tubes. The paired nasal tubes combine to form a "Y"; the lower arm is attached to a CPAP machine, and the upper arms terminate in soft silicone nasal inserts. There are no published studies of CPAP PRO in the medical literature. It is not possible to determine how this device compares to standard and broadly used CPAP interfaces.

Continuous Positive Airway Pressure (CPAP): Alajmi et al. (2007) conducted a meta-analysis of data from 10 randomized controlled trials to assess the impact of CPAP on blood pressure and to identify any patient characteristics that might explain variations in the outcomes in the different studies. The effects of CPAP on blood pressure were modest and not statistically significant. Compared to control, CPAP reduced systolic blood pressure by 1.38 mm Hg (p=0.23) and reduced diastolic blood pressure by 1.52 mm Hg (p=0.06). Six of the trials evaluated patients with more severe OSA (e.g., mean AHI > 30/hour, 313 patients). In these trials, CPAP reduced systolic blood pressure by 3.03 mm Hg (p=0.10), and reduced diastolic blood pressure by 2.03 mm Hg (p=0.05). There was a trend for systolic blood pressure reduction to be associated with CPAP compliance. The authors stated that because follow-up in most studies was short and only one study included follow-up longer than nine weeks, it is unclear whether blood pressure reductions are maintained or increase over time.

A systematic review of the literature conducted by an AASM Task Force (Gay, et al., 2006) evaluated the efficacy of PAP treatment, specifically CPAP and BPAP, for sleep-related breathing disorders in adults. The authors concluded that compared to placebo, conservative management or positional therapy, CPAP eliminates respiratory disturbances and therefore reduces the AHI. There was evidence supporting improved stage three

and four sleep and decreased EEG arousals with CPAP vs. placebo, but whether CPAP yields significant consistent improvement in overall sleep architecture or fragmentation is less clear. The authors reported that resolution of sleepiness was accompanied by improved driving performance, and that the majority of studies revealed a positive benefit on psychometric or vigilance measures, as well as neurobehavioral and quality of life measures. The large variation in testing methods, population selection and interventions made it difficult to form firm conclusions on these aspects of treatment, however. Adverse events related to CPAP were reported in the AASM review to be generally minor and reversible. The most common patient complaints were related to pressure intolerance and interface issues (i.e., mask, nasal prongs). Early adherence monitoring is important, since many side effects can occur during the first few weeks of CPAP use, leading to discontinuation of treatment. Common side effects include mask leak, dry mouth, pressure intolerance, sense of suffocation or difficulty exhaling, skin abrasion, conjunctivitis, claustrophobia, rhinitis, and sneezing. The authors reported some positive impact was seen with the use of different interfaces and the use of humidification.

Shivalkar et al. (2006) conducted a case series to evaluate structural and functional cardiac alterations in OSA, their relationship to the severity of OSA and the effects of treatment with CPAP in patients with no known cardiac disease (n=43). Left and right ventricular morphology and function were studied using echocardiography before and after treatment with CPAP in symptomatic patients with severe OSA. Structural and functional cardiac changes of the left and right ventricle were closely associated with the severity of OSA. Of the original 43 patients, 25 were evaluated following six months of treatment with CPAP. Significant improvements were seen in symptoms and hemodynamics and in left and right ventricular morphology and function. The authors concluded that the structural and functional consequences of OSA on the heart are influenced by the severity of AHI, and these effects are reversible if the apneic episodes are abolished.

A Cochrane systematic review of the effectiveness of CPAP for the treatment of OSA (Giles, et al., 2005) included 36 trials and 1718 patients. Included trials compared CPAP with an inactive control or use of an oral appliance in adults with OSA and an AHI greater than five per hour. Compared to control, CPAP showed significant improvements in objective and subjective sleepiness and several quality of life, cognitive function and depression measures. Twenty-four hour systolic and diastolic blood pressures were lower with CPAP compared to control. There was stronger evidence of effectiveness in symptomatic patients with moderate and severe AHI. Compared to oral appliances, CPAP significantly reduced the AHI and improved sleep efficiency and minimum oxygen saturation. There was no obvious difference in symptoms, however, and patients who responded to both CPAP and oral appliance therapy expressed a strong preference for the oral appliance. The authors concluded that available evidence supports the use of CPAP as first-line treatment for OSA patients with high AHI and moderate to severe daytime sleepiness. The authors further concluded that patients who do not accept or who struggle to continue with CPAP should be provided with alternative options.

Patients who are unable to tolerate conventional nasal CPAP despite an adequate trial may respond to second-generation devices that provide more flexibility in titrating the airway pressure, such as PAP with expiratory pressure relief (e.g., C-Flex), APAP and BPAP.

C-Flex: C-Flex (Respironics Inc., Murrysville, PA) received FDA 510(k) approval on Oct 10, 1999. C-Flex is a feature available on CPAP, APAP, and BPAP devices manufactured by Respironics. The C-Flex feature lowers the initial expiratory pressure in proportion to the patient's expiratory flow rate. The pressure is then increased to therapeutic levels near the end of exhalation when airway collapse is most likely. It has been proposed that C-Flex could result in increased comfort and may improve treatment adherence.

Pepin et al. (2009) conducted a double-blind randomized controlled trial to determine whether pressure reduction during exhalation (C-Flex) would improve CPAP compliance, comfort and quality of life. Patients newly diagnosed with OSA (n=218) were randomized to three months of treatment with CPAP (n=108) or C-Flex (n=110). Objective compliance, generic quality of life (SF-36) scores, and visual analog scales for CPAP comfort and side effects were evaluated at baseline and after three months. After three months, patients in the CPAP arm were moved to the C-Flex arm for an additional three months (open study). There was no difference at three months between CPAP and C-Flex in compliance, rate of side effects, and comfort. Patients who moved from CPAP to C-Flex after the initial three month trial did not significantly change their PAP adherence, but there was a significant increase in PAP adherence in the subgroup of CPAP patients classified as low compliers (i.e., < 4 hours of use) (p=0.04).

Aloia et al. (2005) conducted a comparison study of CPAP therapy vs. therapy using the C-Flex device in participants with moderate to severe obstructive sleep apnea. Participants were recruited from and followed through an academic sleep disorders center. Eighty-nine participants were recruited into the study following in-laboratory PSG and prior to initiation of therapy. Participants received either therapy with CPAP (n=41) or with the C-Flex device (n=48), depending on the available treatment at the time of recruitment. Follow-up assessments were conducted at three months. The mean treatment adherence over the three-month follow-up period was higher in the C-Flex group compared to the CPAP group (weeks 2–4, 4.2 vs. 3.5, respectively; weeks 9–12, 4.8 vs. 3.1, respectively). Clinical outcomes and attitudes toward treatment (self-efficacy) were also measured. There was no difference between the two groups in subjective sleepiness and functional outcomes associated with sleep. Self-efficacy showed a trend toward being higher at follow-up in those patients who had been treated with the C-Flex device compared to CPAP treatment. The authors concluded that therapy with the C-Flex device may improve overall adherence over three months compared to standard therapy with CPAP, and although subjective sleepiness and functional outcomes did not improve, C-Flex users may be more confident about their ability to adhere to treatment. The authors concluded that randomized controlled trials are needed to confirm these findings.

Although there is limited evidence regarding the impact of PAP with expiratory pressure relief (e.g., C-Flex), the addition of expiratory pressure relief to CPAP treatment may result in increased comfort and improved compliance in patients who are intolerant of the constant expiratory pressures of traditional CPAP. There is no evidence to indicate that the addition of expiratory pressure relief to APAP or BPAP, however, results in improved compliance or clinical outcomes.

Auto-Titrating Positive Airway Pressure (APAP): The pressure required to maintain airway patency changes during a night of sleep depending on body position, sleep stage, nasal obstruction, and ingestion of alcohol or hypnotic agents. Pressure requirements also change over time based on changes in body weight and upper airway properties. As stated earlier, during CPAP titration, the minimum amount of positive pressure required to eliminate or nearly eliminate respiratory events in REM and NREM sleep, including REM sleep with the patient in the supine position, is determined. Traditional CPAP maintains this effective fixed pressure at all times and may well be higher than needed for most of the night. A number of auto-titrating devices have been developed that deliver variable pressure according to the needs of the patient. When an obstructive event is detected, an APAP device will increase pressure until the event is eliminated. If no further events are detected during a set time period, the device will decrease pressure to a pre-set minimum. APAP devices may use combinations of physiologic signals to detect airflow obstruction, including snoring, flow, or impedance. Because the minimum pressure required to keep the airway open is used, the mean pressure applied throughout the night is reduced. It has been proposed that this reduction in mean pressure may improve patient tolerance, resulting in improved adherence with the use of PAP (Ayas, et al., 2004; Nussbaumer, et al., 2006).

In a randomized, double-blind, controlled cross-over trial, Nussbaumer et al. (2006) compared the efficacy of APAP and CPAP treatment. A series of 36 OSA patients were randomly assigned to one month of home therapy with APAP followed by one month of CPAP, or vice versa. After one month of treatment, the mean ESS score, sleep resistance time and AHI were significantly improved with both treatments. Twenty-six patients preferred APAP over CPAP in the initial phase of therapy. The authors concluded that the effectiveness of APAP in improving major outcomes was equivalent to CPAP. The authors also noted that an additional feature of APAP is that it does not require initial titration.

Ayas et al. (2004) conducted a meta-analysis of the use of APAP vs. standard CPAP for the treatment of OSA that included nine trials and 282 patients. The authors reported that APAP is associated with a reduction in mean pressure of 2.2 cm of water pressure throughout the night. Post-treatment AHI, subjective sleepiness and adherence were similar in both groups. The authors stated that there are important differences in the various APAP models, depending on the manufacturer. The algorithms by which they detect events and the manner in which they increase or decrease applied pressure vary, and the effectiveness and tolerability of one APAP model compared to another may be very different. In addition, newer devices with advanced technology may be more effective than older models. Pooling the results of studies using various devices and time periods may therefore be overly simplistic. The authors state that although APAP should not be considered a first-line therapy in most patients with OSA, there are likely subgroups of patients who may do better with APAP rather than CPAP. These may include younger patients, patients intolerant of CPAP, or patients with position-dependent or sleep stage-dependent OSA.

Massie et al. (2003) conducted a multisite, randomized, single-blind cross-over study to determine whether CPAP use and outcomes can be improved by an APAP device in patients with OSA who require higher CPAP pressures (i.e., 10 centimeters or more of water pressure). A total of 44 patients were randomized to six weeks at laboratory-determined fixed pressure and six weeks on APAP. Average nightly use was greater in automatic mode (306 versus 271 minutes), and median and 95th percentile pressures in automatic mode were lower. APAP resulted in better SF-36 (short form health survey) vitality scores (65 ± 20 vs. 58 ± 23) and mental health scores (80 vs. 75), but no significant difference in Epworth Sleepiness Scale (ESS) scores. During automatic therapy, patients reported more restful sleep, better quality sleep, less discomfort from pressure, and less trouble getting to sleep for both the first week of therapy and for the averaged scores for weeks two through six. The authors reported that patients who require higher fixed CPAP used APAP more and reported greater benefit from this therapy.

Berry et al. (2002) conducted an AASM systematic review of the literature on the use of APAP for treatment of OSA in adults. The authors stated that in the evaluated studies, including 16 randomized controlled trials, APAP reduced the AHI to acceptable levels (i.e., $AHI < 10$ /hour) in greater than 80–95% of patients studied. Although this was considered acceptable treatment, a reduction to an $AHI < 5$ may be needed for reversal of sleepiness in some patients. A total of three nonrandomized and 11 randomized controlled trials were reviewed to determine whether APAP can reduce the AHI as well as conventional CPAP. The APAP mean or median pressure was lower in nine studies and slightly higher in one study. The authors also reported that a number of studies report improved sleep quality, defined as a treatment arousal index of ≤ 20 events per hour or an increase in either slow wave or REM sleep, or both. In general, the literature supports the idea that improvements in sleep quality with APAP and CPAP are similar. The authors also reported no significant differences between APAP and CPAP in measures of oxygen desaturation or in sleepiness as measured by the ESS.

Smith and Lasserson conducted a Cochrane systematic review (2009) to determine the efficacy of pressure level modifications and additional humidification in increasing CPAP usage. The analysis included 45 studies ($n=184$) that assessed interventions to improve CPAP compliance (i.e., APAP, BPAP, C-Flex, and humidification.). There was a statistically significant difference in machine usage of 0.21 hours per night in favor of APAP from crossover studies, although this difference was considered to be of questionable clinical significance. There was no statistically significant difference between APAP and CPAP in terms of Epworth Sleepiness Scores (ESS) in parallel studies, but there was an overall reduction in ESS scores in cross-over studies in favor of APAP. More patients preferred APAP to fixed CPAP when this was measured. No significant difference in usage was noted when CPAP was compared to BPAP or C-Flex, and there were conflicting findings between the studies in the use of humidification. The authors stated that further studies are needed to assess the evidence for Bi-PAP, C-Flex, and humidification. The authors noted that included studies were characterized by high machine usage in the control groups, and low withdrawal rates. Future studies should consider the effects of treatment in participants with more mild disease, and those who struggle to accept therapy despite persistent symptoms.

AASM practice parameters for the use of auto-titrating CPAP devices for titrating pressures and treating adult patients with OSA include the following recommendations (Morgenthaler, et al., 2007). Recommendations are classified as follows: Standard: a generally accepted patient care strategy that reflects a high degree of clinical certainty; Guideline: a patient care strategy that reflects a moderate degree of clinical certainty, and Option: a patient care strategy that reflects uncertain clinical use.

- APAP is not recommended to diagnose OSA (Standard)
- Patients with the following conditions are not currently candidates for APAP titration or treatment: (Standard)
 - Congestive heart failure
 - Lung disease, such as chronic obstructive pulmonary disease
 - Patients expected to have nocturnal arterial oxyhemoglobin desaturation due to conditions other than OSA (e.g., obesity, hypoventilation syndrome)
 - Patients who do not snore, either due to palate surgery or naturally
- APAP devices are not currently recommended for split-night titration (Standard)
- Certain APAP devices may be used during attended titration with PSG to identify a single pressure for use with standard CPAP for treatment of moderate to severe OSA. (Guideline)

- Certain APAP devices may be used in an unattended way to determine a fixed CPAP pressure for patients with moderate to severe OSA without significant comorbidities (CHG, COPD, central sleep apnea syndrome, or hypoventilation syndromes) (Option)
- Patients being treated with fixed CPAP on the basis of APAP titration or being treated with APAP must have close clinical follow-up to determine treatment effectiveness and safety. This is especially important during the first few weeks of PAP use. (Standard)
- A re-evaluation and, if necessary, a standard attended CPAP titration should be performed if symptoms do not resolve or the CPAP or if the APAP treatment otherwise appears to lack efficacy. (Standard)

Home PAP Titration

As discussed in the PSG section above, PAP pressures may be titrated during the second portion of a split-night PSG when a diagnosis of OSA has been established during the initial diagnostic portion of the exam. PAP pressures may also be titrated during a full-night PSG that follows a diagnostic PSG in which the diagnosis of OSA is established. Home titration using APAP has also been utilized as an alternative to in-laboratory PSG titration.

Mulgrew et al. (2007) conducted a randomized controlled trial to test the utility of a diagnostic algorithm in conjunction with ambulatory CPAP titration in the initial management of OSA. A total of 68 patients with a high probability of moderate to severe OSA were randomly assigned to PSG or to ambulatory titration using a combination of APAP and overnight oximetry. Patients in the PSG group received an overnight diagnostic PSG, and final CPAP was determined during a titration PSG performed the following night. The ambulatory group received an APAP device set to auto-titrate at pressures between 4 and 20 cm H₂O. After one week of use, the APAP device was interrogated. The 95th percentile pressure was taken as the initial effective pressure if no residual sleep-disordered breathing was identified, and patients continued at this pressure in fixed CPAP mode for another week. On days six and 13, overnight oximetry was performed, and CPAP settings were adjusted if oxygen desaturation was observed either night or if residual respiratory events were noted on device interrogation. The final CPAP pressure was set on day 14. After three months, there was no difference between the two groups in the primary outcome, AHI on CPAP. Adherence to CPAP therapy was good in both groups, with slightly higher compliance in the ambulatory group (median 6.0, range 5.1–7.1 hours per night) than in the PSG group (median 5.4, range 3.7–6.4 hours per night). The difference between the two groups was statistically significant ($p=0.021$). The authors acknowledged that the interventions of overnight oximetry, CPAP downloads, and CPAP adjustments may have contributed to better adherence in the ambulatory group, however.

Cross et al. (2006) conducted a randomized, single-blind, parallel-group controlled trial in Scotland to evaluate whether APAP titration in the home produced patient outcomes equal to those following laboratory-based APAP titration. A consecutive series of CPAP-naïve patients with OSA were randomized to laboratory-based APAP titration ($n=100$) or home titration ($n=100$). Patients assigned to hospital titration had a single-night study attended by two sleep nurses, and were continuously monitored by the APAP device and by video, sound, oxygen saturation, and position monitoring. The home patients had APAP titration for three nights, with telephone access to the sleep center on the first titration night. In addition, the home patients received calls from clinical staff at the sleep center after the first two nights. On day three, patients returned to the sleep center with the APAP unit for downloading of data. After titration, all patients were issued a CPAP device set at the fixed pressure determined from the titration study. The CPAP titration pressures were similar in both groups (hospital 10.6 ± 0.2 cm H₂O, home 10.4 ± 0.2 cm H₂O, $p=.19$). At three months, there was no significant difference between the two groups in CPAP use, the primary outcome measure (hospital 4.39 ± 0.25 , home 4.38 ± 0.25 , $p>.9$). At three months, improvements in ESS scores were similar in both groups, and health status questionnaires showed no statistical difference.

The AASM practice parameters on the use of APAP for titrating pressures, discussed above (Morgenthaler et al., 2008), state, "Certain APAP devices may be used in an unattended way to determine a fixed CPAP pressure for patients with moderate to severe OSA without significant comorbidities (CHG, COPD, central sleep apnea syndrome, or hypoventilation syndromes). (Option)." The writing committee noted that the evidence was specific to each device, including the particular version of software and device version, and the pressure determination should be made by experienced sleep specialists after examining the raw pressure titration data for each patient. For these reasons, the authors did not find that the available evidence supported a guideline recommendation. The use of APAP for titrating pressures was considered an option, meaning that this is a patient care strategy that reflects uncertain clinical use and implies inconclusive or conflicting evidence, or conflicting expert opinion.

Although PSG-directed titration remains the standard method for determination of effective CPAP pressure, unattended titration using an APAP device may be a reasonable option for patients diagnosed with moderate or severe OSA without significant comorbidities.

BPAP: BPAP is a noninvasive respiratory device that delivers different levels of inspiratory and expiratory pressure. A lower pressure is applied during the expiratory phase so that the total pressure applied to the airway can be reduced. BPAP devices have additional flow and pressure delivery methods to meet the needs of patients with various respiratory conditions, such as those requiring ventilatory support, and have been shown to be therapeutic for OSA. Reported advantages of BPAP include decreasing the work of breathing, lowering mean treatment pressure, and creating a more physiologic breathing pattern. BPAP is applied via a nasal mask or full-face interface.

BPAP is a reasonable treatment option for patients who cannot tolerate CPAP or APAP as well as for patients with chronic obstructive pulmonary disease or hypoventilation syndromes. BPAP is not considered a first-line treatment, however, since it has not been demonstrated to be superior to traditional CPAP in terms of adherence to therapy, treatment outcomes, nasal discomfort or complaints regarding therapy (ICSI, 2005).

AASM practice parameters on the use of CPAP and BPAP state that BPAP is an optional therapy in some cases where high pressure is needed and the patient experiences difficulty exhaling against a fixed pressure. BPAP may also be indicated when coexisting central hypoventilation is present (Kushida, et al., 2006).

Oral Appliances

Various oral appliances have been developed for the treatment of OSA. Most of these devices are designed based on the principal that advancing the mandible and holding it forward during sleep improves upper airway patency and/or decreases upper airway collapsibility. The appliance is attached to the upper and lower dental arches and allows for incremental advancement of the mandible. Studies using cephalometry have shown that these mandibular repositioning appliances (MRAs) lower the tongue position, reduce the mandibular plane-to-hypoid distance, advance the mandible and widen the upper oropharynx (retropalatal and retroglossal) in some patients. MRAs, also referred to as mandibular advancement appliances (MAA) or mandibular advancement splints (MAS) may be custom-made based on dental impressions or may consist of a prefabricated appliance adapted to the patient's dimensions. Side effects reported with the use of MRAs include discomfort in the temporomandibular joint (TMJ), tooth and facial musculature discomfort, bite change, excessive salivation, and mouth dryness. Contraindications to MRA therapy include moderate to severe TMJ disorders, an inadequate protrusive ability, and lack of an adequate number of healthy teeth in the upper and lower dental arch. Significant bruxism may also be a contraindication, since damage to the appliance or increased pain may result. Patients with full dentures are generally unable to use an MRA but may be treated with a tongue-retaining appliance (TRA).

TRAs, also referred to as tongue-retaining devices (TRD), hold the tongue forward and affect genioglossus muscle activity in patients with OSA. The effect on other upper airway muscles has not been evaluated, however. TRAs may be custom-made or fitted by the patient. There are few studies on the use of TRAs, and these devices are generally only used in patients with contraindications to the use of an MRA.

Lam et al. (2006) conducted a randomized trial to compare three nonsurgical treatments for mild to moderate OSA (n=101). Inclusion criteria consisted of AHI > 5 and ≤ 40, and ESS score of > 9 for those with AHI of 5–20. Patients were assigned to one of three groups: conservative measures (i.e., sleep hygiene) (n=33), CPAP in addition to conservative measures (n=34), and oral appliance in addition to conservative measures (n=34). All overweight patients were referred to a weight reduction class. Assessment included ESS, the Health-Related Quality of Life (HRQOL) portion of the SF-36, and Sleep Apnea Quality of Life Index (SAQLI). At ten weeks, hypoxemia and AHI were significantly improved in both the CPAP and appliance group, but an AHI of < 5 was achieved only in the CPAP group, and improvement in arousal index was only significant in the CPAP group. The conservative measures group showed no significant change in AHI. The difference in the improvement in AHI between the groups was statistically significant. Of the 91 patients who completed the study and underwent weight measurement, 15 patients in each treatment group had a decrease in weight. There was a linear relationship between changes in body weight and changes in AHI. There was a decrease in ESS scores in all three groups at ten weeks, with a greater improvement in the CPAP group than the appliance or

conservative treatment group. Overall, CPAP produced the greatest improvement in physiological, symptomatic and HRQOL measures, while the oral appliance was slightly less effective.

A Cochrane systematic review (Lim, et al., 2004) was conducted to determine the effects of oral appliances in the treatment of sleep apnea in adults. Twelve randomized controlled trials (n=509) that compared MRAs to control or other treatments were evaluated. Reviewers concluded that there is increasing evidence suggesting that oral appliances improve subjective sleepiness and indices of sleep-disordered breathing compared to inactive control treatment. CPAP and MRAs both led to improvements in AHI compared to baseline, but the magnitude of improvement favored CPAP. The authors stated that data are limited, however, with relatively small numbers of patients studied in trials that contained methodological weaknesses. The authors therefore recommended that MRA therapy be offered to patients with mild symptomatic OSA and those who are unwilling or unable to comply with CPAP.

AASM practice parameters for the use of oral appliances (Kushida, et al., 2006) based on a literature review conducted by Ferguson et al. (2006) include the following recommendations:

- The presence or absence of OSA must be determined before initiating treatment with oral appliances to identify patients at risk due to complications of sleep apnea and to provide a baseline to establish the effectiveness of subsequent treatment.
- For patients with OSA, the desired outcome of treatment includes the resolution of the clinical signs and symptoms of OSA and the normalization of the AHI and oxyhemoglobin saturation.
- Although not as efficacious as CPAP, oral appliances are indicated for use in patients with mild to moderate OSA who prefer oral appliances to CPAP, or who do not respond to CPAP, are not appropriate candidates for CPAP, or who fail treatment attempts with CPAP or behavioral measures such as weight loss or sleep position change.
- Patients with severe OSA should have an initial trial of CPAP because greater effectiveness has been shown with this intervention than with the use of oral appliances. Upper airway surgery (including tonsillectomy and adenoidectomy, craniofacial operations and tracheostomy) may also supersede use of oral appliances in patients for whom these operations are predicted to be highly effective in treating the OSA.
- To ensure satisfactory therapeutic benefit from oral appliances, patients with OSA should undergo PSG or attended cardiorespiratory (Type III) sleep study with the oral appliance in place after final adjustments of fit have been performed.

Surgical Treatment

Patients with OSA who fail or cannot comply with conservative treatment may be candidates for surgical interventions. The surgical techniques used to treat OSA specifically modify either the retropalatal or retrolingual region of the pharyngeal airway, or, in the case of tracheostomy, bypass the pharyngeal portion of the upper airway. The goals of surgical intervention in the treatment of OSA include resolution of clinical signs and symptoms of OSA and normalization of sleep quality, AHI, and oxyhemoglobin saturation levels.

Numerous upper airway procedures have been developed that may be used alone or in combination with other procedures to treat OSA. Palatal surgery procedures include uvulopalatopharyngoplasty (UPPP) and laser-assisted uvulopalatoplasty (LAUP). Additional palatal stiffening procedures introduced recently include cautery-assisted palatal stiffening operation (CAPSO) and radiofrequency energy (Coblation[®], Somnoplasty[®]).

Palatal surgical procedures alone are not successful in achieving adequate reductions in AHI in most patients. The following procedures may be performed either alone or following palatal surgery when an unacceptable AHI persists: tracheostomy; inferior sagittal mandibular osteotomy (ISO) and genioglossal advancement with hyoid myotomy and suspension (GAHN); and maxillomandibular osteotomy and advancement (MMO). Several additional tongue-base procedures have been proposed for the treatment of OSA, including suture suspension with the Repose System (Influence Corp; San Francisco, CA), and base-of-tongue Somnoplasty.

Tracheostomy may be considered when other options do not exist, have failed or are refused, or when deemed necessary by clinical urgency

Franklin et al. (2009) conducted a systematic review to evaluate the efficacy and adverse effects of surgery for snoring and OSA. The review included four randomized controlled trials of surgery vs. either sham surgery or conservative treatment in adults. The trials included outcome measures of daytime sleepiness, quality of life, AHI, and snoring. There was no significant effect on daytime sleepiness and quality of life after laser-assisted uvulopalatoplasty (LAUP) and radiofrequency ablation. The AHI and snoring were reduced in one trial after LAUP but not in another. Subjective snoring after radiofrequency ablation was reduced in one trial. No trials of other surgical procedures met the inclusion criteria for evaluation of efficacy. A total of 45 observational studies were also reviewed to evaluate adverse effects following surgical treatment. Persistent side-effects occurred after uvulopalatopharyngoplasty (UPPP) and uvulopalatoplasty (UPOP), with difficulty swallowing, globus sensation, and voice changes commonly observed.

Uvulopalatopharyngoplasty (UPPP): UPPP increases the area of the retro-palatal airway by resection of the free edge of the uvula and soft palate in patients with collapse of the oropharyngeal and hypopharyngeal airways, or with some other anatomical impediment such as small retrolingual airways. UPPP may be combined with tonsillectomy and may also be performed sequentially with other surgical procedures. The success of UPPP is variable, with positive results most often seen in patients whose obstruction is limited to the retropalatal airway (Sher, et al., 1996; Sundaram, et al., 2005).

A Cochrane systematic review assessed the results of any surgery in the treatment of OSA in adults (Sundaram, et al., 2005). UPPP was one of several procedures evaluated. The authors concluded that available studies do not provide evidence to support the use of surgery in OSA because overall significant benefit has not been demonstrated. Long-term follow-up of patients who undergo surgical treatment is required to determine whether surgery is curative or whether the signs and symptoms of OSA tend to recur, requiring further treatment.

Sher (1996) conducted a systematic literature review with meta-analysis to provide an overview of the surgical treatment of OSA to provide the basis for the AASM practice parameters on this subject. Studies included in the meta-analysis provided preoperative and postoperative PSG data on at least nine patients treated with UPPP for OSA. Analysis of the UPPP studies revealed that this procedure is, at best, effective in treating less than 50% of patients with OSA. AASM practice parameters based on this review state that UPPP, with or without a tonsillectomy, may be appropriate for patients with narrowing or collapse in the retropalatal region. The recommendations also state that effectiveness of UPPP is variable, and the procedure should only be performed when nonsurgical treatment options, such as PAP, have been considered.

Laser-Assisted Uvulopalatoplasty (LAUP)/Uvulectomy: LAUP differs from UPPP in that much less palatal tissue is removed, the tonsils and pharyngeal pillars are not altered, and a carbon dioxide laser is used rather than a scalpel. Vertical transpalatal laser incisions measuring approximately one cm are made bilaterally through the soft palate lateral to the base of the tongue, followed by partial vaporization of the uvula. Up to seven separate treatment sessions may be required.

Well-designed trials evaluating the safety and efficacy of LAUP are lacking. The AASM (Littner, et al., 2000) conducted a review of studies of LAUP in order to update guidelines for this procedure originally published in 1994. One reviewed study (Finkelstein, et al., 1997) provided evidence that LAUP may result in structural modifications of the upper airway that decrease airway resistance and result in further narrowing during inspiration and collapse of the upper airway at the level of the tongue base. A second reviewed study (Terris, et al., 1996) found worsening of the AHI and a significant decrease in the cross-sectional area of the airway as determined by videoendoscopy between 48 and 72 hours after surgery. The authors of another included study (Berger, et al., 1999) on histopathological changes of the soft palate after LAUP found extensive thermal-induced changes, including diffuse fibrosis, oral epithelia ulceration, and a patchy inflammatory reaction, which the authors proposed may be responsible for worsening of OSA.

AASM updated practice parameters on the use of LAUP include the following recommendations:

- LAUP is not recommended for the treatment of sleep-related breathing disorders, including OSA.
- LAUP is not recommended as a substitute for UPPP in the treatment of sleep-related breathing disorders, including OSA.

Uvulectomy has been proposed as a surgical treatment for snoring and mild obstructive sleep apnea. There are no well-designed studies in the peer-reviewed medical literature that evaluate uvulectomy for the treatment of obstructive sleep apnea. Based on the available evidence, it is not possible to determine the safety and efficacy of this procedure compared to established medical and surgical treatment. Uvulectomy performed as a separate procedure is not addressed in relevant published specialty society guidelines.

(Note: This Coverage Policy is not intended to address uvulectomy when performed for other indications (e.g., acute inflammation/angioedema of the uvula).

Cautery-Assisted Palatal Stiffening Operation (CAPSO): CAPSO is an office-based procedure in which a midline strip of soft palate mucosa is removed, and the wound is left to heal by secondary intention. The procedure has been proposed as a treatment for OSA based on the premise that the resulting midline palatal scar stiffens the palate and eliminates palatal snoring.

Wassmuth et al. (2000) conducted a case series (n=25) to evaluate the ability of CAPSO to treat OSA. PSG was performed preoperatively and at three months following the procedure on all patients. Patients with a reduction in the AHI of 50% or more and an AHI of 10 or less were classified as responders. Based on these criteria, 40% of patients were considered to have responded to CAPSO. Mean AHI improved from 25.1 ± 12.9 to 16.6 ± 15.0 . The ESS improved from 12.7 ± 5.6 to 8.8 ± 4.6 . The authors concluded that CAPSO is as effective as other palatal surgeries in the management of OSA.

Although this case series reported promising results, there is insufficient evidence in the published medical literature to demonstrate the safety, efficacy, and long-term outcomes of CAPSO in the treatment of OSA. Data from well-designed trials with adequate numbers of patients that compare this procedure with other treatments of OSA are lacking.

Pillar™ Palatal Implant System: The Pillar Palatal Implant System (Restore Medical, St. Paul, MN) received FDA 510(k) approval on December 18, 2002, for the treatment of snoring. On June 7, 2004, FDA approval of the Pillar System was expanded to include treatment of OSA. According to the FDA summary, the Pillar System consists of an implant and delivery tool, and is designed to stiffen the tissue of the soft palate to reduce the incidence of snoring in some patients and to reduce the incidence of airway obstruction in patients with mild to moderate OSA. The implant is a cylindrical-shaped segment of braided polyester filaments. The delivery tool consists of a handle and needle assembly that allows for positioning and placement of the implant in the submucosa of the soft palate.

Friedman et al. (2007) conducted a retrospective review to assess subjective and objective improvement in 145 patients with mild to moderate OSA treated with a single-stage multilevel minimally invasive technique. All patients were treated with nasal surgery, palatal stiffening by Pillar implants, and radiofrequency volume reduction of the tongue base. Of 145 patients, 122 had a minimum follow-up of six months and complete data available for review. The primary outcome measure was change from baseline in AHI. The mean AHI decreased from 28.2 ± 7.6 preoperatively to 14.5 ± 10.2 postoperatively ($p < .0001$). Mean Epworth Sleepiness Scale (ESS) decreased from 9.7 ± 3.9 to 7.0 ± 3.3 ($p < .0001$). It is difficult to draw conclusions from this study due to its retrospective design, lack of long-term outcomes, and the inability to determine the individual impact of each procedure on short-term outcomes.

Nordgard et al. (2006) conducted a prospective nonrandomized study of 25 patients with untreated OSA with an AHI of 10–30, as determined by preoperative PSG, and BMI ≤ 30 . Three permanent implants were placed in the soft palate of each patient in an office setting under local anesthesia. A repeat PSG showed a mean decrease in AHI from 16.2 to 12.1 for the study group. Twenty of 25 patients demonstrated a reduced AHI, and 12 of 25 patients demonstrated an AHI of 10 or less 90 days post-implant. The mean ESS score decreased from 9.7 to 5.5. The authors concluded that palatal implants can significantly improve AHI and other sleep-related parameters in patients with mild to moderate OSA and BMI ≤ 30 , with short-term results comparable to those reported for UPPP. The authors acknowledged the lack of long-term outcomes in this study and the limited number of patients. As with other palatal procedures, reduction in effectiveness over time may be expected. The authors further concluded that while short-term durability and effectiveness have been established, longer-term research needs to be conducted.

A multicenter non-comparative study was conducted by Walker et al. (2006) to evaluate the safety and effectiveness of the Pillar Palatal Implant System (n=53). Primary inclusion criteria were primary palatal contribution to OSA as determined by the investigator, an AHI of 10–30 events per hour, BMI \leq 32 kg/m², age 18 or greater, and soft palate length adequate to accommodate a 28-mm implant. Each patient had three implants placed in the soft palate in an office procedure under local anesthesia. The primary outcome measure was AHI. PSG was performed prior to and 90 days following Pillar implantation. The AHI decreased from 25.0 \pm 13.9 to 22.0 \pm 14.8 events/hour (p=0.05). ESS scores, a secondary outcome measure, decreased from 11.0 \pm 5.1 to 6.9 \pm 4.5 (p<0.001). The AHI was reduced to below 10 in 12 patients (23%), and the AHI increased in 18 patients (34%). There were no serious complications. The most common adverse event was partial extrusion. Of 202 implants, 20 became partially exposed through the mucosa of the soft palate. All were removed and, in most cases, the implant was replaced.

Guidance issued by the National Institute for Health and Clinical Excellence (NICE, United Kingdom) in 2007 states that the current evidence on soft palate implants for OSA raises no major safety concerns, but there is inadequate evidence that the procedure is efficacious in the treatment of this potentially serious condition for which other treatments exist. The guidance states that soft palate implants should therefore not be used to treat this condition.

There is insufficient evidence in the published medical literature to demonstrate the safety, efficacy, and long-term outcomes of the Pillar System in the treatment of OSA.

Radiofrequency Volumetric Tissue Reduction (RFVTR): RFVTR (e.g., Coblation[®], Somnoplasty[®]) is a procedure used to remove redundant tissue in the upper airway. Although the procedure has been used to remove tissue from the turbinates and tonsils, recent studies of RFA in the treatment of OSA have limited the procedure to the soft palate, uvula and tongue base.

The ENTec[™] ReFlex[™] Wand (ArthroCare Corp., Sunnyvale, CA) received FDA approval through the 510(k) process on February 4, 2000, for ablation and coagulation of soft tissue in otolaryngological (ENT) surgery, including tissue in the uvula/soft palate for the treatment of snoring and submucosal palatal shrinkage. The ReFlex Wand is used to perform Coblation[®] treatment using radiofrequency energy. In 2002, the ENTec Plasma Wand received 510(k) approval for ablation, resection, and coagulation of soft tissue and hemostasis of blood vessels in ENT surgery, including tissue of the uvula/soft palate for the treatment of snoring.

The Somnoplasty system (Somnus Medical Technologies, Sunnyvale, CA) received FDA 510(k) approval on July 17, 1997, for coagulation of soft tissue, including the uvula/soft palate. The 510(k) summary states that the Somnoplasty system may reduce the severity of snoring in some individuals. An expanded approval on November 2, 1998, states that the system is intended for the reduction of the incidence of airway obstruction in patients with upper airway resistance syndrome and OSA. The Somnoplasty system is comprised of an RF generator and tissue coagulating electrodes. The procedure is usually performed on an outpatient basis with local anesthesia.

National Institute for Health and Clinical Excellence (NICE, United Kingdom) issued interventional procedure guidance on radiofrequency ablation of the soft palate in 2005, stating that current evidence suggests that, although there are no major safety concerns associated with the procedure as a treatment for snoring, evidence on the short-term efficacy is limited and long-term outcomes are uncertain. The NICE guidance states that this procedure should not be used without special arrangements for audit, consent and research.

There is insufficient evidence in the published medical literature to demonstrate the safety, efficacy, and long-term outcomes of RFVTR (e.g., Somnoplasty, Coblation) in the treatment of OSA.

Inferior Sagittal Mandibular Osteotomy (ISO) and Genioglossal Advancement with Hyoid Myotomy and Suspension (GAHM): The two components of the procedure, ISO and GAHM, create an enlarged retrolingual airway. The genoid tubercle of the mandible, which acts as the anterior attachment of the tongue, is advanced by a limited mandibular osteotomy. In the initial version of this procedure, the hyoid bone was advanced and suspended from the mandible by a fascial strip. Modifications of the procedure include stabilization of the hyoid bone anteriorly and inferiorly by attachment to the thyroid cartilage. ISO and GAHM may be performed with UPPP or may be performed following a UPPP procedure.

AASM practice parameters for the surgical treatment of OSA (Thorpy, et al., 1999) state that, of the procedures directed at enlarging the retrolingual region, inferior sagittal mandibular osteotomy and genioglossal advancement with or without hyoid myotomy and suspension, appears to be most promising. The procedure is listed as an option, rather than a guideline or standard, because the success of ISO and GAHM is dependent on the surgeon's skills and expertise, and few centers have experience with this procedure.

Maxillomandibular Osteotomy and Advancement (MMO): MMO is a surgical technique that modifies the airway space by advancing the maxilla, the mandible, and therefore the tongue. MMO is used to correct retrolingual or hypopharyngeal obstruction and is usually performed after a failed UPPP. MMO may also be employed as the sole procedure for OSA patients with mandibular skeletal deformities associated with a narrowed posterior airway space and tongue base obstruction (Sher, et al., 1996).

The AASM practice parameters for the surgical treatment of OSA state that a stepwise approach to surgical management is acceptable if the patient is advised of the likelihood of success of each procedure and that multiple operations may be necessary. In selected patients for whom UPPP and other surgical procedures have failed, MMO may be successful in effectively treating the OSA. MMO is not generally considered as initial therapy, however.

AASM practice parameters also recommend that patients with preoperatively symptomatic or moderate to severe OSA undergo a follow-up evaluation, including objective measures of the presence and severity of OSA and sleep disruption, to assess the presence of residual disease.

The Repose™ Bone Screw System: The Repose Bone Screw System (Influence, Inc., San Francisco, CA) received FDA 510(k) approval on August 27, 1999. The Repose System is used to perform anterior tongue base suspension by fixation of the soft tissue of the tongue base to the mandible bone using a bone screw with pre-threaded sutures. It is indicated for the treatment of OSA and/or snoring. The Repose System has been proposed as a sole treatment of OSA and has also been used in conjunction with UPPP and radiofrequency ablation.

Kuhnel et al. (2005) conducted a prospective nonrandomized study (n=28) to demonstrate the efficacy of tongue base suspension with the Repose System in the treatment of OSA. PSG was performed before as well as three and 12 months after surgery. Lateral cephalometric radiography and videoendoscopy of the pharynx were performed preoperatively and postoperatively to identify morphological changes in the posterior airway space. A suspension suture anchored intraorally at the mandible was passed submucosally in the body of the tongue, with suture tightness adjusted individually. The posterior airway space was widened by at least 2 mm in 60% of cases. Daytime sleepiness improved subjectively in 67% of patients, and the RDI improved postoperatively in 55% of patients. The correlation between posterior airway space widening and the improvements in daytime sleepiness and respiratory disturbance index was not significant. The authors concluded that surgical intervention in obstructive sleep apnea syndrome with the Repose System does not result in permanent anatomical change in the posterior airway space.

Miller et al. (2002) conducted a retrospective analysis of the Repose System for the treatment of OSA to describe preliminary experience using the system in conjunction with UPPP in the multilevel surgical approach. The authors evaluated 19 consecutive patients undergoing UPPP and the Repose System tongue base suspension for the management of OSA during a one-year period (1998 through 1999). Fifteen patients had complete preoperative and postoperative PSG data. A 46% reduction in RDI was demonstrated at a mean of 3.8 months after surgery. The apnea index demonstrated a 39% reduction. The authors concluded that the Repose System in conjunction with UPPP has been shown to produce significant reductions in the RDI and apnea index, as well as a significant increase in oxygen saturation. Despite the improvement in these objective parameters, the overall surgical cure rate was only 20% (three of 15 patients) in this retrospective series. Further research is warranted to define the role of the Repose System in the management of obstructive sleep apnea patients.

There is insufficient evidence in the published medical literature to support the safety, efficacy, and long-term outcomes of the use of the Repose System in the treatment of OSA.

Tracheotomy: Tracheotomy is a surgical procedure in which a percutaneous opening into the trachea is created. The tracheostomy (i.e., diameter of the stoma) is usually stented and maintained by the insertion of a rigid hollow tube that extends to the body surface. The tracheostoma enters the airway distal to the pharynx and

larynx, bypassing the portion of the airway that narrows or collapses with OSA. Because of the cosmetic effects and morbidity associated with tracheotomy, it is rarely performed for the treatment of OSA. According to AASM practice parameters, tracheotomy is the only operation shown to be consistently effective as a sole procedure in the treatment of OSA, but should only be considered when other options do not exist, have failed or are refused, or when deemed necessary by clinical urgency.

Other Devices and Procedures

Provent™ Device: The Provent Professional Sleep Apnea Therapy device (Ventus Medical, Inc., Belmont, CA) received U.S. Food and Drug Administration (FDA) Approval through the 510(k) process on February 6, 2008 for use in the treatment of OSA. The Provent device consists of a single-use nasal insert composed of soft foam surrounding a valve body constructed of a urethane polymer. The valve body contains a silicone valve mechanism that acts to increase the expiratory pressure by creating expiratory resistance, resulting in airway positive back pressure during expiration. A device is inserted into each nostril and held in place by adhesive tape.

In a pilot evaluation of the Provent nasal expiratory resistance device, Colrain et al. (2008) recruited 24 patients with an AHI > 5 and six patients with primary snoring. Exclusion criteria included basal metabolic index (BMI) > 35. Patients were evaluated with PSG on two consecutive nights; PSG alone was performed on one night, and PSG was combined with the Provent device on the alternate night. The AHI and oxygen desaturation both decreased significantly with use of the device ($p < 0.001$ and $p < 0.1$, respectively). The percentage of time spent above 90% oxygen saturation also increased significantly with device use ($p < 0.05$). There were no significant changes in measures of sleep architecture. Because of the study design, small number of participants, and data from a single night of treatment, conclusions cannot be drawn from this pilot study;

There is insufficient evidence in the published medical literature to support the safety, efficacy, and long-term outcomes of the use of the Provent nasal expiratory resistance device in the treatment of OSA.

Electrosleep Therapy: Electrosleep therapy consists of the application of short duration, low-amplitude pulses of direct current to the patient's brain via externally placed occipital electrodes. It has been used in the treatment of chronic insomnia, anxiety, and depression, but has also been used in disorders with possible psychosomatic components, such as asthma, spastic colitis, or tension headache, and for organic disorders, including essential hypertension. Scientific assessment of this technique has not been completed, and its efficacy in the treatment of OSA has not been established.

Injection Snoreplasty: Injection Snoreplasty is a nonsurgical treatment for snoring that involves the injection of a hardening agent into the upper palate. Sodium tetradecyl sulfate is the most common hardening agent used. Following the injection, scar tissue is reported to pull the uvula forward to eliminate palatal flutter associated with snoring. There is no evidence in the published medical literature to demonstrate the safety and efficacy of injection Snoreplasty in the treatment of OSA.

Atrial Overdrive Pacing: Atrial overdrive pacing by means of an implantable cardiac pacemaker has been proposed as a treatment for central sleep apnea patients and in certain OSA patients with some degree of heart failure. Atrial overdrive pacing consists of pacing at a rate higher than the mean nocturnal sinus rate. Investigators theorized that atrial overdrive pacing would improve vagal tone and increase upper airway muscle activity in patients with OSA.

Weng et al. conducted a meta-analysis of eight randomized controlled trials to determine the effects of atrial overdrive pacing on sleep apnea syndrome ($n=129$). Atrial overdrive pacing, as compared to non-pacing, reduced the apnea-hypopnea index (AHI) and increased the minimum arterial oxygen saturation (SaO₂) significantly in the central sleep apnea-predominant trials. No statistically significant increase in minimum SaO₂ was observed in the obstructive sleep apnea syndrome-predominant trials, however, and it was unclear whether AHI was reduced in these patients. The authors concluded that the role of atrial overdrive pacing in obstructive sleep apnea syndrome remains unclear.

Pepin et al. (2005) conducted a randomized controlled trial to assess the ability of overdrive atrial pacing to reduce OSA severity. A total of 17 patients who had received permanent atrial-synchronous ventricular pacemakers for symptomatic bradyarrhythmias and were not known to have central apnea or OSA were studied. Patients received overdrive pacing only during sleep periods, identified by a specific algorithm included

in the pacemaker. Patients underwent three overnight PSG evaluations one month apart. The first was performed for baseline evaluation. The patients were then randomly assigned to either one night in spontaneous rhythm or to one night in pacing mode with atrial overdrive. Two patients refused to continue the study after the first PSG evaluation. OSA was highly prevalent in this population: 10 of the 15 patients exhibited an AHI of > 30. The nocturnal spontaneous rhythm was 59 ± 7 beats per minute at baseline, compared to 75 ± 10 beats per minute with atrial overdrive pacing. The AHI was 46 in spontaneous rhythm, compared to 50 with atrial overdrive pacing. Overdrive pacing changed none of the respiratory indices, sleep fragmentation or sleep structure parameters. The authors concluded that atrial overdrive pacing has no significant effect on obstructive sleep apnea.

Guidelines for device-based therapy published by the American College of Cardiology (ACC) and the American Heart Association (AHA) state that, a variety of heart rhythm disturbances may occur in OSA. Sinus bradycardia or pauses may occur during hypopneic episodes, and atrial tachyarrhythmias may also be observed, especially following an apnea episode. The guideline states that although a small retrospective trial demonstrated a decrease in central or OSA without reducing the total sleep time, subsequent randomized trials have not validated a role for atrial overdrive pacing in OSA (Epstein et al., 2008).

There is insufficient evidence to demonstrate the safety and efficacy of atrial overdrive pacing in the treatment of OSA.

Pediatric Obstructive Sleep Apnea

The etiology, clinical manifestations and treatment of OSA in the pediatric population differ from those in adults. OSA in children is described in a clinical statement by the ATS (1999) as a disorder of breathing during sleep characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction (obstructive apnea) that disrupts normal ventilation during sleep and disrupts normal sleep patterns. In children, obstructive apneas of any length are considered abnormal, and children with OSA may demonstrate obstructive hypoventilation or continuous hypopnea associated with hypercapnia, as opposed to discrete obstructive apnea events as seen in adults. During these episodes, increased respiratory effort as evidenced by retractions and/or paradoxical chest movements may be seen. Hypercapnia or oxyhemoglobin desaturation usually accompany these periods of obstructive hypoventilation. The episodes may terminate spontaneously or by arousal from sleep but may last continuously throughout the night.

Diagnosis: The American Academy of Pediatrics (AAP) voiced agreement with this definition in a technical report on the diagnosis and management of childhood OSA and provided additional elaboration of symptoms. Childhood OSA is associated with habitual (nightly) snoring, sleep difficulties, and/or daytime neurobehavioral problems, choking sounds, abnormal motor activity, arousal from sleep, nightmares, heavy sweating and bedwetting at an inappropriate age. According to the AAP statement, studies generally show a nearly threefold increase in behavior and neurocognitive abnormalities in children with sleep-disordered breathing. Most studies did not differentiate between primary snoring and snoring linked to OSA, so the true prevalence of these factors is not clear. It may be possible that primary snoring, even in the absence of diagnosed OSA, may place children at risk (Schechter, AAP, 2002).

Although snoring is one of the major signs of OSA, this is rarely the reason pediatricians are initially consulted. Children are frequently seen instead because of parents' concerns about eating, behavioral or learning difficulties. History and physical examinations are poor predictors of OSA in children. There is general agreement that in-laboratory PSG is the diagnostic test of choice, since it is the only technique shown to quantify the ventilatory and sleep abnormalities associated with sleep-disordered breathing. It is difficult to assess the accuracy of PSG for OSA in children, however, since performance and interpretation have not been well standardized for the pediatric population. An obstructive apnea index of one is often selected as the cutoff for normalcy, and while this is statistically significant in terms of normative data, it is not known what level actually constitutes clinical significance (AAP, 2002).

According to consensus recommendations published in an ATS statement on standards and indications for cardiopulmonary sleep studies in children (1995), PSG is indicated for the following:

- to differentiate benign or primary snoring from pathological snoring
- for evaluating the child with disturbed sleep patterns, excessive daytime sleepiness, cor pulmonale, failure to thrive, or polycythemia unexplained by other factors or conditions

- when the physician is uncertain whether the clinical observation of obstructed breathing is sufficient to warrant surgery, or if a child needs intensive postoperative monitoring following adenotonsillectomy or other pharyngeal surgery
- for children previously diagnosed with OSA who exhibit persistent snoring or other symptoms of sleep-disordered breathing (If the child's clinical condition permits, this repeat PSG should be deferred until at least four weeks following surgery to allow resolution of postoperative edema.)
- for titrating CPAP levels and periodically reevaluating the appropriateness of CPAP settings

The ATS consensus recommendations also state that in a child under one year, or a child with severe OSA based on clinical symptoms, number of obstructive events, or severe desaturation episodes, follow-up PSG should be considered to assure resolution of clinically significant abnormalities.

(Note: The ATS statement referenced above includes additional indications for PSG unrelated to OSA not listed here.)

Several procedures have been proposed as alternatives to overnight in-laboratory PSG for the diagnosis of pediatric OSA. Unattended home PSG, also referred to as cardiorespiratory monitoring, has been found to be comparable to the diagnostic accuracy of in-laboratory PSG at high apnea index levels but serves primarily as a screen for lower apnea levels. Home audiotaping and videotaping to supplement clinical evaluation has been explored as an alternative to PSG but has been inadequately investigated. Additional studies are necessary before statements regarding the validity of this method can be made. Overnight pulse oximetry and nap PSG have been shown to have high specificity but low sensitivity. Negative test results would therefore require confirmation by PSG (AAP, 2002).

An ECRI Short Technology Assessment, Diagnosis of Sleep Disorders in Children, evaluated research published since the AAP guideline and technical report of 2002. The report states that several good quality studies and a systematic review confirm the finding that history and physical are clearly insufficient to make a diagnosis of OSA. The literature on alternatives to PSG still indicates that PSG is the gold standard procedure for the diagnosis of OSA, although this literature is sparse and generally of poor quality because the alternatives have not been compared directly with PSG. The report states that questionnaires, audiotaping, and pulse oximetry may be useful screening methods prior to the use of PSG but much more research is needed here, and that home PSG in children must be compared to laboratory PSG before a definitive statement can be made about the use of home PSG (ECRI, 2007).

Treatment: In contrast to adult patients, the vast majority of children with OSA have hypertrophy of the tonsils and adenoids, and the current first-line treatment therefore is adenotonsillectomy. Additional risk factors for OSA in children include neuromuscular disease, obesity, and genetic syndromes, especially those associated with midface hypoplasia, small nasopharynx, or micrognathia, such as Down syndrome and Pierre Robin sequence. Studies evaluated in the AAP OSA technical report demonstrated adenotonsillectomy to be curative in 75–100% of cases, even in children who are obese. Children may be candidates for CPAP when adenotonsillectomy is unsuccessful or when definitive surgery is indicated but must await complete dental and facial development. Commercially available nasal masks fit children from infancy through adolescence, and the flow rates and pressures delivered by currently marketed devices appear to be appropriate and safe for children (ATS, 1994; AAP, 2002).

Currently available CPAP devices are FDA approved for home use for children who weigh more than 30 kilograms (66 pounds). Limited data is available on CPAP compliance in children. A small prospective study by Marcus et al. (2006) randomly assigned 29 children, ages two to 16, to six months of CPAP vs. BPAP. One third of the children dropped out before six months. Of the remaining 21 children for whom adherence data could be downloaded, the mean nightly use was 5.3 ± 2.5 hours. Parental assessment of adherence was considerably higher than actual use. PAP was highly effective, with a reduction of the AHI from 27 ± 32 /hour to 3 ± 5 /hour. Results were similar for children who received CPAP vs. BPAP. The authors concluded that PAP is effective in children with OSA, but adherence is an important issue. The authors suggested that additional research be conducted to develop methods to improve adherence and to develop other treatment alternatives for children who do not respond to tonsillectomy and adenoidectomy and are unable to tolerate CPAP.

Summary

In-laboratory polysomnography (PSG) is broadly accepted as the definitive diagnostic tool for evaluating OSA. A number of other methods have been proposed as alternatives to PSG, but none have demonstrated the specificity and sensitivity of PSG in the diagnosis of OSA. Unattended/home sleep studies using a Type II or Type III device that records, at a minimum, airflow, respiratory effort, and blood oxygenation may be a reasonable alternative to in-laboratory PSG in carefully selected patients.

Positive airway pressure (PAP) is the most effective and widespread treatment for OSA, and has been demonstrated to be effective in reducing or abolishing apneic episodes in patients with OSA and in alleviating associated symptoms. C-Flex and auto-titrating PAP (APAP) are modified versions of CPAP that may be considered for patients who cannot tolerate conventional CPAP. Bi-Level PAP (BPAP) is a treatment option when high pressures are needed and the patient experiences difficulty exhaling against a fixed pressure and may be an option for patients with chronic obstructive pulmonary disease or hypoventilations syndromes. Mandibular repositioning appliances (MRA) have been demonstrated to be effective in the treatment of OSA, although comparative studies demonstrate a greater improvement with PAP when compared to MRA. Tongue-retaining appliances (TRAs) are generally used only in patients with contraindication to the use of MRAs.

Patients who fail or cannot comply with conservative treatment may be candidates for surgical interventions, including uvulopalatopharyngoplasty (UPPP), inferior sagittal mandibular osteotomy (ISO) and genioglossal advancement with hyoid myotomy and suspension (GAHN); and maxillomandibular osteotomy and advancement (MMO), and tracheostomy. Numerous additional procedures and devices have been proposed for the treatment of OSA, including laser assisted uvulopalatoplasty (LAUP), uvulectomy, cautery-assisted palatal stiffening operation (CAPSO), Pillar™ Palatal Implant System, radiofrequency volumetric tissue reduction (RFVTR) (Somnoplasty®), the Repose™ Bone Screw System, the Provent™ Professional Sleep Apnea Therapy Device, electrosleep therapy, Injection Snoreplasty, and atrial overdrive pacing. There is insufficient evidence in the published medical literature to demonstrate the safety, efficacy and long-term outcomes of these devices/procedures in the treatment of OSA.

There is general agreement that in-laboratory PSG is the diagnostic test of choice in children. The vast majority of children with OSA have hypertrophy of the tonsils and adenoids, and the current first-line treatment, adenotonsillectomy, has been shown to be curative in 75–100% of cases. Children may be candidates for CPAP when adenotonsillectomy is unsuccessful, is contraindicated, or when definitive surgery is indicated but must await complete dental and facial development.

Coding/Billing Information

Note: This list of codes may not be all-inclusive.

Diagnostic Testing

Covered when medically necessary for the diagnosis of obstructive sleep apnea:

CPT®* Codes	Description
95806	Sleep study, unattended, simultaneous recording of, heart rate, oxygen saturation, respiratory airflow, and respiratory effort (eg, thoracoabdominal movement)
95807	Sleep study, simultaneous recording of ventilation, respiratory effort, ECG or heart rate, and oxygen saturation, attended by a technologist
95808	Polysomnography; sleep staging with 1-3 additional parameters of sleep, attended by a technologist
95810	Polysomnography; sleep staging with 4 or more additional parameters of sleep, attended by a technologist
95811	Polysomnography; sleep staging with 4 or more additional parameters of sleep, with initiation of continuous positive airway pressure therapy or bi-level ventilation, attended by a technologist

HCPCS Codes	Description
G0398	Home sleep study test (HST) with type II portable monitor, unattended; minimum of 7 channels: EEG, EOG, EMG, ECG/heart rate, airflow, respiratory effort and oxygen saturation
G0399	Home sleep test (HST) with type III portable monitor, unattended; minimum of 4 channels: 2 respiratory movement/airflow, 1 ECG/heart rate and 1 oxygen saturation

ICD-9-Diagnosis Codes	Description
327.23	Obstructive sleep apnea (adult) (pediatric)

In-laboratory polysomnography (PSG) or home/portable sleep studies are experimental, investigational, or unproven and not covered for the following diagnoses, in the absence of suspected obstructive sleep apnea:

ICD-9-Diagnosis Codes	Description
327.30-327.39	Circadian rhythm sleep disorder
296.3	Major depressive disorder, recurrent episode
311	Depressive disorder, not elsewhere classified
496	Chronic airway obstruction, not elsewhere classified
780.52	Insomnia, unspecified

Covered when medically necessary for evaluation of suspected narcolepsy:

CPT® Codes	Description
95805	Multiple sleep latency or maintenance of wakefulness testing, recording, analysis and interpretation of physiological measurements of sleep during multiple trials to assess sleepiness

ICD-9-Diagnosis Codes	Description
347.00	Narcolepsy, without cataplexy

Experimental/Investigational/Unproven/Not Covered for any indication:

CPT® Codes	Description
95803	Actigraphy testing, recording, analysis, interpretation, and report (minimum of 72 hours to 14 consecutive days of recording) (New code effective 1/1/09)
0203T	Sleep study, unattended, simultaneous recording; heart rate, oxygen saturation, respiratory analysis (eg, by airflow or peripheral arterial tone) and sleep time
0204T	Sleep study, unattended, simultaneous recording; minimum of heart rate, oxygen saturation, and respiratory analysis (eg, by airflow or peripheral arterial tone)

ICD-9-Diagnosis Codes	Description
	All codes

Treatment of Obstructive Sleep Apnea

Covered when medically necessary for the treatment of obstructive sleep apnea:

CPT® Codes	Description
21198	Osteotomy mandible segmental
21199	Osteotomy, mandible, segmental; with genioglossus advancement
21206	Osteotomy, maxilla, segmental (eg, Wassmund or Schuchard)
21685	Hyoid myotomy and suspension
31600	Tracheostomy, planned (separate procedure);
31601	Tracheostomy, planned (separate procedure); younger than two years
42145	Palatopharyngoplasty (e.g., uvulopalatopharyngoplasty, uvulopharyngoplasty)
42820	Tonsillectomy and adenoidectomy; younger than age 12
42821	Tonsillectomy and adenoidectomy; age 12 or over
42825	Tonsillectomy, primary or secondary; younger than age 12
42826	Tonsillectomy, primary or secondary; age 12 or over
42830	Adenoidectomy, primary; younger than age 12
42831	Adenoidectomy, primary; age 12 or over
42835	Adenoidectomy, secondary; younger than age 12
42836	Adenoidectomy, secondary; age 12 or over
94660	Continuous positive airway pressure ventilation (CPAP), initiation and management

HCPCS Codes	Description
E0470	Respiratory assist device, bi-level pressure capability, without back-up rate feature, used with noninvasive interface, e.g., nasal or facial mask (intermittent assist device with continuous positive airway pressure device)
E0471	Respiratory assist device, bi-level pressure capability, with back-up rate feature, used with noninvasive interface, e.g., nasal or facial mask (intermittent assist device with continuous positive airway pressure device)
E0472	Respiratory assist device, bi-level pressure capability, with backup rate feature, used with invasive interface, e.g., tracheostomy tube (intermittent assist device with continuous positive airway pressure device)
E0485	Oral device/appliance used to reduce upper airway collapsibility, adjustable or non-adjustable, prefabricated, includes fitting and adjustment
E0486	Oral device/appliance used to reduce upper airway collapsibility, adjustable or non-adjustable, custom fabricated, includes fitting and adjustment
E0601	Continuous airway pressure (CPAP) device
S8262	Mandibular orthopedic repositioning device, each

ICD-9-Diagnosis Codes	Description
327.23	Obstructive sleep apnea (adult) (pediatric)

Experimental/Investigational/Unproven/Not Covered for the treatment of obstructive sleep apnea:

CPT®* Codes	Description
41512	Tongue base suspension, permanent suture technique (new code effective 1/1/09)
41530	Submucosal ablation of the tongue base, radiofrequency, one or more sites, per session (new code effective 1/1/09; replaces 0088T)
42140 [†]	Uvulectomy, excision of the uvula

HCPCS Codes	Description
G0400	Home sleep test (HST) with type IV portable monitor, unattended; minimum of 3 channels
S2080 ^{††}	Laser-assisted uvulopalatoplasty (LAUP)

ICD-9- Diagnosis Codes	Description
327.23	Obstructive sleep apnea (adult) (pediatric)

Treatment of Snoring:

Not Medically Necessary/Not Covered for the treatment of snoring:

CPT®* Codes	Description
21198	Osteotomy mandible segmental
21199	Osteotomy, mandible, segmental; with genioglossus advancement
21206	Osteotomy, maxilla, segmental (eg, Wassmund or Schuchard)
21685	Hyoid myotomy and suspension
31600	Tracheostomy, planned (separate procedure);
31601	Tracheostomy, planned (separate procedure); younger than two years
41512	Tongue base suspension, permanent suture technique (new code effective 1/1/09)
41530	Submucosal ablation of the tongue base, radiofrequency, one or more sites, per session (new code effective 1/1/09; replaces 0088T)
42145	Palatopharyngoplasty (e.g., uvulopalatopharyngoplasty, uvulopharyngoplasty)
42140 [†]	Uvulectomy, excision of the uvula
42820	Tonsillectomy and adenoidectomy; younger than age 12
42821	Tonsillectomy and adenoidectomy; age 12 or over
42825	Tonsillectomy, primary or secondary; younger than age 12
42826	Tonsillectomy, primary or secondary; age 12 or over
42830	Adenoidectomy, primary; younger than age 12
42831	Adenoidectomy, primary; age 12 or over
42835	Adenoidectomy, secondary; younger than age 12
42836	Adenoidectomy, secondary; age 12 or over
94660	Continuous positive airway pressure ventilation (CPAP), initiation and management

HCPCS Codes	Description
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E0470	Respiratory assist device, bi-level pressure capability, without back-up rate feature, used with noninvasive interface, e.g., nasal or facial mask (intermittent assist device with continuous positive airway pressure device)
E0471	Respiratory assist device, bi-level pressure capability, with back-up rate feature, used with noninvasive interface, e.g., nasal or facial mask (intermittent assist device with continuous positive airway pressure device)
E0472	Respiratory assist device, bi-level pressure capability, with backup rate feature, used with invasive interface, e.g., tracheostomy tube (intermittent assist device with continuous positive airway pressure device)
E0485	Oral device/appliance used to reduce upper airway collapsibility, adjustable or non-adjustable, prefabricated, includes fitting and adjustment
E0486	Oral device/appliance used to reduce upper airway collapsibility, adjustable or non-adjustable, custom fabricated, includes fitting and adjustment
E0601	Continuous airway pressure (CPAP) device
G0400	Home sleep test (HST) with type IV portable monitor, unattended; minimum of 3 channels
S2080	Laser-assisted uvulopalatoplasty (LAUP)
S8262	Mandibular orthopedic repositioning device, each

ICD-9-Diagnosis Codes	Description
786.09	Symptoms involving respiratory system and other chest symptoms, other (snoring)

[†]Note: This Coverage Policy is not intended to address uvulectomy performed for other indications (e.g., acute inflammation/angioedema of the uvula).

*Current Procedural Terminology (CPT®) ©2010 American Medical Association: Chicago, IL.

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Policy History

<u>Pre-Merger Organizations</u>	<u>Last Review Date</u>	<u>Policy Number</u>	<u>Title</u>
CIGNA HealthCare	9/15/2008	0158	Obstructive Sleep Apnea Diagnosis and Treatment Services
Great-West Healthcare	3/14//2006	04.260.02	Continuous Positive Airway Pressure (CPAP) and Bi-Level Positive Airway Pressure (BPAP)
	01/04/2006	97.259.06	Sleep Apnea Treatment

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Connecticut General Life Insurance Company has acquired the business of Great-West Healthcare from Great-West Life & Annuity Insurance Company (GWLA). Certain products continue to be provided by GWLA (Life, Accident and Disability, and Excess Loss). GWLA is not licensed to do business in New York. In New York, these products are sold by GWLA's subsidiary, First Great-West Life & Annuity Insurance Company, White Plains, N.Y.