Cigna Medical Coverage Policy

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

Effective Date **10/15/2013**
Next Review Date **10/15/2014**
Coverage Policy Number **0158**

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Coverage Policy

In certain markets, delegated vendor guidelines may be used to support medical necessity and other coverage determinations.

HOME/PORTABLE SLEEP STUDY AND IN-FACILITY POLYSOMNOGRAPHY (PSG) ADULT:

Cigna covers a sleep study as medically necessary for the diagnosis of suspected obstructive sleep apnea (OSA) in an adult (age 18 or older) when BOTH of the following criteria are met. (Refer to the sections below to determine whether in-facility PSG or home/portable testing is indicated):

- evidence of daytime sleepiness (e.g., excessive sleepiness not explained by other factors, non-refreshing sleep, sleep fragmentation)
- **ANY** of the following additional symptoms or risk factors of OSA:
  - witnessed apneas
  - gasping or choking at night
  - disruptive snoring
  - increased neck circumference (i.e., > 17 inches in men, > 16 inches in women)
  - obesity (i.e., body mass index ≥ 30)

Home/Portable Study:

Cigna covers a home/portable sleep study* as medically necessary for the diagnosis of obstructive sleep apnea (OSA) in an adult (age 18 or older) when ALL of the following criteria are met:

- study/test equipment meets the minimum definition described in at least one of the following Current Procedural Terminology (CPT) or Health Care Procedure Coding System (HCPCS) codes:
    - 95800: Sleep study, unattended, simultaneous recording: heart rate, oxygen saturation, respiratory analysis (eg, by airflow or peripheral arterial tone) and sleep time)
    - 95801: Sleep study, unattended, simultaneous recording: minimum of heart rate, oxygen saturation, and respiratory analysis (e.g., by airflow or peripheral arterial tone)
    - 95806: Sleep study, unattended, simultaneous recording of heart rate, oxygen saturation, respiratory airflow, and respiratory effort (eg, thoracoabdominal movement)
    - 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 study test (HST) with type III portable monitor, unattended; minimum of 4 channels: 2 respiratory movement/airflow, 1 ECG/heart rate and 1 oxygen saturation
- medical necessity criteria for a sleep study for suspected OSA as outlined above have been met
- absence of significant comorbid condition that would be expected to degrade the accuracy of a home/portable study, such as any of the following
    - moderate to severe pulmonary disease, such as chronic obstructive pulmonary disease (COPD)
    - moderate to severe neuromuscular/neurodegenerative disorder causing restrictive lung diseases (e.g., kyphoscoliosis, myasthenia gravis, amyotropic lateral sclerosis (ALS), polio, Polymyositis, Guillian Barre syndrome)
    - congestive heart failure Class III or IV
    - obesity hypoventilation syndrome, previously documented
    - pulmonary hypertension
- no sleep disorder other than OSA is suspected (e.g., central sleep apnea, periodic limb movement disorder, parasomnias, narcolepsy, REM behavior sleep disorder)

*__Note; A home/portable study is considered to be one study, whether performed during a single night or during two or more consecutive nights.__

Cigna covers a home/portable sleep study when the diagnosis of OSA has been established in an adult (age 18 or older) when ALL of the following criteria are met:

- testing is to be performed for **ANY** of the following:
    - confirmation of therapeutic benefit following final adjustment of a mandibular repositioning appliance (MRA)
    - assessment of results following surgical treatment for OSA
    - clinical response is insufficient or symptoms return despite a good initial response to a mandibular repositioning appliance
- no significant oxygen desaturation during diagnostic sleep study
- absence of comorbid sleep disorder or significant comorbid medical condition, as described above, that would be expected to degrade the accuracy of a home/portable study

Cigna covers home titration using auto-titrating PAP (APAP to determine a fixed CPAP pressure for ongoing treatment when ALL of the following criteria are met:

- individual meets the criteria for PAP (detailed in PAP section below)
- individual does not have a comorbid condition that would be expected to degrade the accuracy of auto-titration, such as any of the following:
    - congestive heart failure Class III or IV
    - significant lung disease [e.g., COPD)
    - prior diagnosis of central sleep apnea
- no evidence of nocturnal oxygen (O_2) desaturations caused by a condition other than OSA (e.g. obesity hypoventilation syndrome), as indicated by ANY of the following results of initial sleep study::
    - O_2 saturation < 80% for > 1% of recording time during prior diagnostic home study
    - O_2 saturation < 80% for > 1% of sleep time during prior diagnostic facility-based study
    - O_2 saturation < 90% for > 22% of recording time during prior diagnostic facility-based PSG
    - O_2 saturation < 90% for > 22% of recording time during prior diagnostic home study

Cigna covers follow-up home titration using APAP when ALL of the following criteria are met:

- no comorbid condition that would be expected to degrade the accuracy of auto-titration
- no evidence of nocturnal oxygen desaturation caused by a condition other than OSA (as described above)
- procedure to be performed for ANY of the following:
  - to determine whether pressure adjustment is needed when clinical response is insufficient or symptoms return despite a good initial response to PAP
  - substantial weight loss (e.g., 10% of body weight) to determine if adjustment of PAP pressure is indicated
  - substantial weight gain (e.g., 10% of body weight) with return of symptoms despite continued use of CPAP, to determine if adjustment of PAP pressure is indicated

Cigna does not cover a Type IV home-portable sleep study (HCPCS code G0400) for any indication because it is considered experimental, investigational or unproven.

Cigna does not cover a home/portable sleep study for any other indication because it is considered not medically necessary.

<u>In-Facility Polysomnography (PSG)-Full-Night:</u>

Cigna covers full night in-facility polysomnography (PSG) (CPT codes 95808, 95810) as medically necessary in an adult (age 18 or older) when BOTH of the following criteria are met:

- medical necessity criteria for a sleep study for suspected obstructive sleep apnea (OSA) as outlined above have been met
- **ANY** of the following:
  - significant comorbid condition that would be expected to degrade the accuracy of a home/portable study such as any of the following
    - moderate to severe pulmonary disease, such as chronic obstructive pulmonary disease (COPD)
    - moderate to severe neuromuscular/neurodegenerative disorder causing restrictive lung diseases (e.g., kyphoscoliosis, myasthenia gravis, amyotropic lateral sclerosis (ALS), polio, polymyositis, Guillian Barre syndrome)
    - congestive heart failure (moderate to severe)
    - obesity hypoventilation syndrome, previously documented
    - pulmonary hypertension
  - sleep disorder other than OSA is suspected (e.g., central sleep apnea, periodic limb movement disorder, parasomnias, narcolepsy, REM behavior sleep disorder) that is corroborated by the clinical documentation
  - recent home/portable testing proved to be technically inadequate or failed to establish the diagnosis of OSA in an individual with high pretest likelihood of OSA
  - individual and caregiver/companion incapable of operating home testing equipment

Cigna covers full night in-facility polysomnography (PSG) (CPT codes 95808, 95810) as medically necessary prior to a planned multiple sleep latency test (MSLT) in an adult (age 18 or older) with suspected narcolepsy.

<u>In-Facility Polysomnography (PSG) with Initiation of Positive Airway Pressure (PAP) (Split-Night Study):</u>

Cigna covers split-night in-facility polysomnography (PSG) (CPT code 95811), in which the initial diagnostic portion of the PSG is followed by positive airway pressure (PAP) titration, as medically necessary in an adult (age 18 or older) when ALL of the following criteria are met:

- medical necessity criteria for a sleep study for suspected obstructive sleep apnea (OSA) as outlined above have been met

- apnea/hypopnea index (AHI) of 15 or higher during initial diagnostic portion of split-night study, or AHI > 5 with symptoms indicative of significant OSA (e.g., repetitive obstructions, significant oxygen desaturation)
- **ANY** of the following:
  - significant comorbid condition that would be expected to degrade the accuracy of a home/portable study. such as any of the following
    - moderate to severe pulmonary disease, such as chronic obstructive pulmonary disease (COPD)
    - moderate to severe neuromuscular/neurodegenerative disorder causing restrictive lung diseases (e.g., kyphoscoliosis, myasthenia gravis, amyotropic lateral sclerosis (ALS), polio, polymyositis, Guillian Barre syndrome)
    - congestive heart failure (moderate to severe)
    - obesity hypoventilation syndrome, previously documented
    - pulmonary hypertension
  - sleep disorder other than OSA is suspected (e.g., central sleep apnea, periodic limb movement disorder, parasomnias, narcolepsy, REM behavior sleep disorder) and is corroborated by the clinical documentation
  - recent home/portable testing proved to be technically inadequate or failed to establish the diagnosis of OSA in an individual with high pretest likelihood of OSA
  - individual and caregiver/companion incapable of operating home testing equipment

<u>In-Facility Polysomnography (PSG)-Positive Airway Pressure (PAP) Titration:</u>

Cigna covers in-facility PSG (CPT code 95811) for PAP titration, following a prior diagnostic study as medically necessary in an adult (age 18 or older) when BOTH of the following criteria are met:

- AHI ≥ 15 documented on prior PSG or home/portable study, or AHI ≥ 5 and < 15, with symptoms of OSA (e.g., excessive daytime sleepiness, impaired cognition, mood disorders or insomnia, or with hypertension, ischemic heart disease or history of stroke
- EITHER of the following:
  - a comorbid sleep disorder (e.g., central sleep apnea, periodic limb movement disorder, parasomnias, narcolepsy, REM behavior sleep disorder) corroborated by the clinical documentation
  - a significant comorbid condition that would be expected to degrade the accuracy of a home/portable study, such as any of the following
    - moderate to severe pulmonary disease, such as chronic obstructive pulmonary disease (COPD)
    - moderate to severe neuromuscular/neurodegenerative disorder causing restrictive lung diseases (e.g., kyphoscoliosis, myasthenia gravis, amyotropic lateral sclerosis (ALS), polio, Polymyositis, Guillian Barre syndrome)
    - congestive heart failure (moderate to severe)
    - obesity hypoventilation syndrome, previously documented
    - pulmonary hypertension

Cigna covers in-facility PSG (CPT code 95811) for re-titration of PAP as medically necessary in an adult (age 18 or older) when BOTH of the following criteria are met:

- clinical response to PAP is insufficient or symptoms return despite compliance with PAP therapy
- individual with significant oxygen desaturation during diagnostic sleep study, or presence of a comorbid sleep disorder or significant comorbid medical condition as described above

Cigna does not cover <u>adult</u> in-facility PSG for any other indication because it is considered not medically necessary.

Cigna does not cover an abbreviated cardiorespiratory sleep study to acclimate an individual to PAP (e.g., PAP-Nap study, CPT code 95807-52) because it is considered experimental, investigational or unproven.

HOME/PORTABLE SLEEP STUDY AND IN-FACILITY POLYSOMNOGRAPHY-CHILD:

In-Facility Polysomnography:

Cigna covers pediatric in-facility polysomnography (PSG) (CPT codes 95782, 95783, 95808, 95810, 95811) as medically necessary for ANY the following indications:

- obstructive sleep apnea (OSA) suspected based on clinical assessment
- following adenotonsillectomy in a child with mild preoperative OSA with residual symptoms of OSA
- following adenotonsillectomy to assess for residual OSA in child with preoperative evidence of moderate to severe OSA, obesity, craniofacial anomalies that obstruct the upper airway, or neurologic disorder (e.g., Down syndrome, Prader-Willi syndrome, myelomeningocele)
- titration of positive airway pressure (PAP) in a child with OSA
- suspected congenital central alveolar hypoventilation syndrome or sleep related hypoventilation due to neuromuscular disorders or chest wall deformities
- primary apnea of infancy
- evidence of a sleep related breathing disorder in infant who has experienced an apparent life threatening event (ALTE).
- child being considered for adenotonsillectomy to treat OSA
- follow-up for child on chronic PAP support, to determine whether pressure requirements have changed due to growth and development; if symptoms recur while on PAP; or if additional or alternate treatment is instituted
- assessment of response to treatment with an oral appliance
- noninvasive positive pressure ventilation (NIPPV) titration in child with other sleep-related breathing disorder (SRBD)
- evaluation of child treated with mechanical ventilation for adjustment of ventilator settings.
- evaluation prior to decannulation in child treated with tracheostomy for SRBD
- clinical suspicion of an accompanying sleep related breathing disorder in a child with chronic asthma, cystic fibrosis, pulmonary hypertension, bronchopulmonary dysplasia, or chest wall abnormality (e.g., kyphoscoliosis)

Cigna does not cover pediatric in-facility PSG for any other indication because it is considered not medically necessary.

Home/Portable Testing:

Cigna does not cover a home/portable sleep study for the diagnosis of OSA in a child because it is considered experimental, investigational or unproven.

Cigna does not cover an in-facility polysomnography (PSG) or home/portable sleep study in an adult or child for any of the following indications because each is 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

OTHER DIAGNOSTIC TESTS:

Cigna covers multiple sleep latency testing (MSLT) or maintenance of wakefulness testing (MWT) (CPT code 95805) as medically necessary for the evaluation of suspected narcolepsy when other sleep disorders have been ruled out by prior PSG.

Cigna does not cover MSLT or MWT (CPT code 95805) for the diagnosis of OSA because it is considered not medically necessary.

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

- SleepStrip™
- Actigraphy (CPT code 95803)

Coverage of, testing for, and the treatment of obstructive sleep apnea and other sleep disorders is subject to the terms, conditions and limitations as described in the applicable benefit plan's schedule of copayments. Please refer to the applicable benefit plan document and schedules to determine benefit availability and the terms, conditions and limitations of coverage particularly around coverage for testing required for employment, insurance coverage, or government license purposes. Even when there is no exclusion in the benefit plan for such coverage, Cigna considers screening for or the evaluation of obstructive sleep apnea or other sleep disorder to be not medically necessary when required for employment, insurance or government license purposes in the absence of symptoms suggestive of obstructive sleep apnea or other sleep disorder.

NONSURGICAL TREATMENT

Coverage for continuous positive airway pressure (CPAP), 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 (CPT code E0601) or auto-titrating PAP (APAP) (HCPCS code E0601) with or without a humidifier (HCPCS codes E0561, E0562) for an initial 90 day period as medically necessary for the treatment of OSA in an adult (18 years or older) when EITHER of the following criteria is met:

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

Cigna covers CPAP (HCPCS code E0601) or auto-titrating PAP (APAP) (HCPCS code E0601) with or without a humidifier (HCPCS codes E0561, E0562) for an initial 90 day period as medically necessary for the treatment of OSA in a child 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 bi-level positive airway pressure (BPAP) (HCPCS codes E0470, E0471, E0472) with or without a humidifier (HCPCS codes E0561, E0562) for an initial 90 day period as medically necessary for the treatment of OSA for an individual with coexisting central hypoventilation or for an individual who requires, but proves intolerant to, high pressures of CPAP or APAP.

Cigna covers continued CPAP, APAP, or BPAP therapy beyond the initial 90 day period when adequate adherence to therapy is demonstrated.

Cigna covers CPAP, APAP, or BPAP loaner rental for up to 30 days when BOTH of the following criteria are met:

- demonstrated compliant use of the device
- description of malfunction and documentation that equipment has been sent for repair/assessment

Cigna does not cover PAP with expiratory pressure relief (e.g., C-Flex, C-Flex +, A-Flex, Bi-Flex) [Respironics, Inc., Murrysville, PA]) because it is considered not medically necessary.

Cigna does not cover positive airway pressure (PAP) treatment (i.e., CPAP, APAP, BPAP) for any other indication because it is considered experimental, investigational or unproven.

Cigna does not cover oral pressure therapy (e.g., Winx® Sleep Therapy System) because it is considered experimental, investigational or unproven.

Cigna covers ANY ONE of the following interfaces for use with CPAP, APAP, or BPAP as medically necessary:

- nasal mask (HCPCS code A7027)
- nasal pillows/prongs (HCPCS code A7034)
- full face mask (HCPCS code A7030)
- Oracle™ Oral Mask (Payne & Raykel Healthcare, Irvine, CA) (HCPCS code A7044)

Cigna covers a replacement of any of the above interfaces for use with CPAP, APAP, or BPAP as medically necessary at a frequency of no more often than every three months.

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 (HCPCS codes E0485, E0486, S8262)), also referred to as mandibular advancement appliance or mandibular advancement splint, as medically necessary for an individual with mild or moderate OSA when EITHER of the following criteria is met:

- apnea/hypopnea index (AHI) ≥ 15 and < 30, as documented by polysomnography (PSG) or home/portable sleep study
- AHI ≥ 5 and < 15 as documented by PSG or home/portable sleep study, 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 (HCPCS codes E0485, E0486, S8262) as medically necessary for an individual with severe OSA (i.e., AHI ≥ 30) who is 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) or home/portable sleep study.

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
- for mild or moderate OSA, consideration has also 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 multi-level or stepwise surgery (MLS) (e.g., UPPP and/or genioglossus advancement and hyoid myotomy (GAHM), maxillary and mandibular advancement osteotomy [MMO]) as a combined procedure or as stepwise multiple procedures as medically necessary for the treatment of OSA when ALL of the following criteria are met:

- narrowing of multiple sites in the upper airway
- criteria for PAP met and individual has proved intolerant to or failed a trial of PAP
- a mandibular repositioning appliance (MRA) or tongue-retaining appliance has been considered and found to be ineffective or undesirable

Cigna covers maxillo-mandibular advancement as medically necessary for the treatment of severe OSA when ALL of the following criteria are met:

- criteria for PAP met and individual has proved intolerant to or failed a trial of PAP`
- a mandibular repositioning appliance (MRA) or tongue-retaining appliance has been considered and found to be ineffective or undesirable
- individual has craniofacial disproportion or deformities

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)
- cautery-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 (e.g., AIRVance System)
- transpalatal advancement pharyngoplasty
- 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 ≥ 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.

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 (cricothryroid 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.

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.

Diagnosis of OSA: Adult
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).

The term upper airway resistance syndrome (UARS) was introduced by Guilleminault to describe patients who did not meet the criteria for OSA but experience excessive daytime sleepiness and other somatic complaints.. UARS is based on the theory that one could experience an event with a progressive increase in upper airway resistance and hence, in snoring, that resulted in arousal, but did not meet the definition of hypopnea. More sensitive nasal pressure measurements demonstrated that some of these upper airway resistance events were actually hypopneas. Subsequent publications describe UARS as an abnormally sensitive reaction to the normal increase in upper airway resistance that occurs during sleep, is more common in women, and presents with symptoms of fatigue and other somatic symptoms. The term respiratory effort-related arousals (RERA) was

introduced to capture upper airway resistance events, defined as a sequence of breaths over at least ten seconds with increasing respiratory effort, terminating in arousal. A RERA may be detected by the use of esophageal pressure manometry which demonstrates a pattern of progressively increasing negative esophageal pressure followed by an arousal. The concept of UARS remains controversial. Identification of the syndrome would require recording esophageal pressure via an indwelling catheter, a procedure not routinely performed. . With UARS, air flow is maintained by increased respiratory effort despite partial airway obstruction (Ishman, 2010; Goroll, 2009; Pien, 2010)

Methods used to treat OSA (e.g. PAP, oral appliances) have also been proposed for the treatment of UARS. There is insufficient evidence in the published medical literature to determine whether such treatments are effective or result in improved outcomes in patients with UARS.

Polysomnography (PSG) and Home/Portable Sleep Studies
Polysomnography is the collective process of monitoring and recording physiologic data during sleep. Full-night in-laboratory PSG is considered by most experts as the reference method for evaluating OSA. Based on 1994 American Sleep Disorders Association (now American Academy of Sleep Medicine [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.

In a split-night PSG, the initial diagnostic portion of the PSG is followed by positive airway pressure (PAP) titration, based on the apnea-hypopnea index (AHI) during the initial portion of the test. A follow-up PSG may be performed when a diagnosis of OSA is confirmed during a prior full-night PSG, or when confirmed during a split-night study when the PAP titration portion of the study is insufficient.

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).

As stated above, in 1994 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. Type IV devices provide limited information; they do not measure sleep time and cannot distinguish between obstructive and central apneas.

The 1994 classification system was based on the number and type of “leads” used, and was closely aligned with existing Current Procedural Terminology (CPT) codes. Since then, there has been a proliferation of devices that measure various parameters, and many devices do not fall within this classification scheme. In 2011, an AASM task force proposed a more specific and inclusive method of classifying and evaluating sleep testing devices other than PSG (refer to AASM section on page 12). Also in 2011, AASM published Standards for Accreditation of Out of Center Sleep Testing that state that OCST equipment must meet the minimum definitions described in at least one of the specified sleep testing Current Procedural Terminology (CPT) or Health Care Procedure Coding System (HCPCS) codes currently in use.

Although facility-based PSG is considered by most experts to be the reference method for evaluation of OSA, this does not mean that it is an error-free “gold standard” for the diagnosis of OSA. Such a gold standard would consist of a set of criteria or measurements that distinguish patients with OSA from those without, with small

misclassification errors, PSG indices alone, however, are not adequate to classify individuals as those with and without OSA. An AHI suggestive of OSA is not sufficient for the diagnosis of the condition, since the severity of symptoms must be accounted for, and other conditions that affect sleep must be excluded. A gold-standard would also have inherent prognostic ability, since patients with OSA have a different prognosis than those without OSA. AHI is not well correlated with response to CPAP therapy, or compliance with therapy. Thus the increased accuracy of the AHI obtained by facility-based PSG may not be predictive of outcomes (Agency for Healthcare Research and Quality [AHRQ], 2007).

Comparison of portable testing to PSG has been one approach taken to validate portable monitoring. Because there is not a direct correlation of PSG results with clinical symptoms and outcomes, however, determination of an accepted treatment threshold based solely on AHI or any other PSG measurement has not been possible. Lack of such a threshold prevents comparative studies of portable monitor testing to calculate sensitivity, specificity, and likelihood ratios. Simultaneous in-laboratory PSG and portable monitoring recordings may be compared to unattended portable monitoring in the home, but direct comparison of results from PSG and portable monitoring are not closely correlated. This may be due to differences in equipment and testing environments, intra-scorer reliability, and night-to-night variability of AHI.

Because of the limitations of studies directly comparing results of PSG to portable monitoring, comparative effectiveness studies have instead evaluated clinical outcomes of patients managed with portable monitoring at home vs. those managed with PSG. These non-inferiority or equivalency trials compare improvements in quality of life and other outcomes instead of directly comparing sleep test results (Kuna, 2010).

Kuna et al. (2011 conducted a randomized parallel noninferiority trial to determine whether patients with suspected OSA who received portable monitor testing at home have functional improvements and subsequent CPAP adherence that are not inferior to patients receiving in-laboratory PSG. Consecutive patients referred to two Pennsylvania Veterans Affairs facilities were randomized to either home testing (n=148) or standard in-laboratory testing (n=148). Home testing consisted of a type 3 portable monitor recording followed by at least three nights using an automatically adjusting PAP device. An in-laboratory PSG was performed for patients with an AHI less than 15 events/hour on home testing. Patients diagnosed with OSA were treated with CPAP for three months. The primary outcome measure was the score on the Functional Outcomes of Sleep Questionnaire (FOSQ), designed to assess the impact of disorders of excessive sleepiness on functional status. Of 296 patients, 260 (88%) were diagnosed with OSA, and 213 were initiated on CPAP. The mean functional outcome score improved 1.74 ± 2.81 in the home group ($p<0.0001$) and 1.85 ± 2.46 in the in-laboratory group ($p<0.0001$). Mean hours of daily CPAP adherence were 3.5 ± 2.5 hours/day in the home group and 2.9 ± 2.3 hours/day in the in-laboratory group ($p=0.08$). Patients with OSA randomized to home unattended testing had functional improvement and adherence to CPAP over a three month period that was not clinically inferior to those who receive standard in-laboratory PSG testing.

Skomro et al. (2010) conducted a randomized controlled trial to compare a home-based diagnostic and therapeutic strategy for OSA with the current standard practice of in-laboratory PSG. Adults with suspected OSA referred to three sleep medicine physicians were randomized to the monitoring home (HM) arm (n=44) or PSG arm (n=45). Patients in the HM arm underwent testing using a level III device measuring airflow, respiratory effort, oxygen saturation, heart rate and body position. Home testing was followed by one week of auto CPAP therapy and three weeks of fixed-pressure CPAP, based on the 95% pressure obtained from the auto CPAP device. After completion of HM, and prior to application of auto-CPAP, patients underwent an in-laboratory PSG. CPAP was applied during the PSG if the AHI was ≥ 15. If the AHI was > 5 but < 15, a repeat in-laboratory PSG with CPAP titration was performed. Patients in the PSG arm underwent overnight PSG in the sleep laboratory followed by one night of home monitoring. CPAP titration was performed during the split-night PSG if the AHI was >15, or during a second in-laboratory PSG, if the AHI was > 5 but <15. Patients were assessed one week after CPAP initiation using the Epworth Sleepiness Scale (ESS), and were evaluated for CPAP compliance. At week four, there was no significant difference in ESS (PSG 6.4 ± 3.8 vs. HM (6.5 ± 3.8, P = .71); Pittsburgh Sleep Quality Index (PSQI) (PSG 5.4 ± 3.1 vs. HM 6.2 ± 3.4, P = .30), Calgary Sleep Apnea Quality of Life Index (SAQLI) (PSG 4.5 ± 1.1 vs. HM 4.6 ± 1.1, P = .85); Standard Form 36 Health Survey (SF-36) vitality (PSG 62.2 ± 23.3 vs. HM 64.1 ± 18.4, P = .79), SF-36 mental health (PSG 84.0 ± 10.4 vs. HM 81.3 ± 14.9, P = .39), and blood pressure (PSG 129/84 ± 11/0 vs. HM 125/81 ± 13/9, P = .121). There was no difference in CPAP adherence (PSG 5.6 ± 1.7 h/night vs. HM 5.4 ± 1.0 h/night, P = .49). The authors concluded that, compared with the home-based protocol, diagnosis and treatment of OSA in the sleep laboratory does not lead to superior four-week outcomes in sleepiness scores, sleep quality, quality of life, blood pressure, and CPAP adherence.

A randomized controlled trial by Tonally de Oliveira et al. (2009) evaluated the accuracy of portable monitoring using a Type III device performed at home to diagnose OSA and its outcomes after first validating PM in the laboratory setting by comparing it to PSG (n=157). Patients were referred in random order to PM at the sleep lab concurrently with in-lab PSG or home PM. Diagnostic performance evaluation included sensitivity, specificity, positive and negative predictive values, positive likelihood ration (+ LR), and negative likelihood ratio (- LR). In-lab PM demonstrated sensitivity of 95.3%, specificity of 75%, + LR of 3.8, and – LR of 0.11. Home PM demonstrated sensitivity of 96%, specificity of 64%, + LR of 2.7, and -LR of 0.005. There was substantial correlation between PSG and PM results. The diagnostic performance of the in-lab PM was only marginally higher than the home PM, compared to PSG. The authors stated that the agreement of home PM with PSG is similar to that described between two typical PSG studies. Several clinical outcomes obtained from the Berlin Questionnaire for sleep apnea, the ESS, and systolic and diastolic blood pressure were correlated with PSG and home PM AHI. Correlation coefficients were similar; indicating the diagnostic ability of the three indexes is comparable.

Mulgrew et al. (2007) conducted a randomized controlled trial to test the utility of a diagnostic algorithm in conjunction with ambulatory CPAP titration in initial management of OSA. Standard PSG was compared to ambulatory CPAP titration in high-risk patients identified by a diagnostic algorithm. Sixty eight patients with a high pretest probability of moderate to severe OSA (AHI > 15 episodes per hour) were identified by sequential application of the ESS score, Sleep Apnea Clinical Score, and overnight oximetry, and were randomly assigned to PSG (n=35) or ambulatory titration (n=33). Ambulatory titration was completed by using a combination of auto PAP (APAP) and overnight oximetry. The PSG and ambulatory groups were similar in terms of median BMI, age, ESS score, and respiratory disturbance index. Each episode was determined by a computer algorithm based on analysis of oxygen saturation measured by pulse oximetry. At three months, there was no difference between the groups in the primary outcome, AHI on CPAP (median 3.2 vs. 2.5; difference, 0.8/hour, p=0.31), or in secondary outcome measures of Epworth Sleepiness Scale (p=.0.86) or Sleep Apnea Quality of Life Index (p=0.41). CPAP compliance was better in the ambulatory group than the PSG group (p=0.021). The algorithm yielded a probability of 0.94 for the diagnosis of moderate to severe OSA. One patient in the ambulatory group with Cheyne Stokes respiration was misdiagnosed as having OSA. The authors suggested a clinical algorithm for use in patients with a high probability of OSA, with the caveat that patients who don't fulfill the criteria for high probability, or who do not respond appropriately to CPAP, should undergo PSG.

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% CCI, 0.86–0.89). There was a significant degree of heterogeneity (p=000) regarding average sleep times. The authors stated that this result was heavily influenced by a single large study with narrow confidence internals. 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.

Centers for Medicare and Medicaid (CMS): A National Coverage Determination (NCD) for sleep testing for OSA issued in 2009 concluded that the evidence was sufficient to determine that the results of the sleep tests below can be used to diagnose OSA, that the use of such sleep testing technologies demonstrated improved health outcomes in Medicare beneficiaries who have OSA and receive the appropriate treatment, and that these tests are reasonable and necessary. The NCD provides the following coverage indications and limitations:

Nationally Covered Indications

- Type I PSG is covered when used to aid the diagnosis of OSA in beneficiaries who have clinical signs and symptoms indicative of OSA if performed attended in a sleep lab facility.
- Type II or Type III sleep testing devices are covered when used to aid the diagnosis of OSA in beneficiaries who have clinical signs and symptoms indicative of OSA if performed unattended in or out of a sleep lab facility or attended in a sleep lab facility.
- Type IV sleep testing devices measuring three or more channels, one of which is airflow, are covered when used to aid the diagnosis of OSA in beneficiaries who have signs and symptoms indicative of OSA if performed unattended in or out of a sleep lab facility or attended in a sleep lab facility.
- Sleep testing devices measuring three or more channels that include actigraphy, oximetry, and peripheral arterial tone, are covered when used to aid the diagnosis of OSA in beneficiaries who have signs and symptoms indicative of OSA if performed unattended in or out of a sleep lab facility or attended in a sleep lab facility.

Nationally Non-Covered Indications

Effective for claims with dates of services on and after March 3, 2009, other diagnostic sleep tests for the diagnosis of OSA, other than those noted above for prescribing CPAP, are not sufficient for the coverage of CPAP and are not covered.

SleepStrip™: The SleepStrip 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.

PAP-Nap Study: An abbreviated cardiorespiratory sleep study, referred to as a PAP-nap study, has been proposed as a method to acclimate patients to PAP and promote adherence to therapy. The PAP-nap study includes mask and pressure desensitization and therapy to overcome aversive emotional reactions, mental imagery, and physiologic exposure to PAP therapy during a nap period. There is insufficient evidence in the published medical literature to determine whether PAP-nap studies result in improved adherence to therapy of improved patient outcomes.

American Academy of Sleep Medicine (AASM): A task force was commissioned by the Board of the American Academy of Sleep Medicine (AASM) Collop et al., 2011) to determine a more specific and inclusive method of classifying and evaluating sleep testing devices other than PSG used as aids in the diagnosis of OSA in the out-of-center setting. The term out-of-center (OOC) sleep testing is used to describe portable monitoring/home sleep testing. The first widely used classification system published by AASM in 1994 placed devices into four categories based on the number and type of "leads" used, and this scheme closely aligned with available Current Procedural Terminology (CPT) codes. Since that time, a plethora of devices have been developed, and many do not fall within this classification scheme. The authors proposed a new classification method that details the types of signals measured. The proposed system categorizes OOC devices based on measurements of Sleep, Cardiovascular, Oximetry, Position, Effort, and Respiratory (SCOPER) parameters. Criteria for evaluating devices was also proposed; in patients with a high pre-test probability of having OSA, the OOC testing device has a positive likelihood ration of 5 or greater, coinciding with an in-lab PSG-generated AHI of ≥ 5, and an adequate sensitivity (at least 0.825). Using the above criteria, the authors reviewed peer-reviewed literature on FDA-approved devices that utilize more than one signal. Devices that do no include oximetry were excluded, since oximetry is a mandatory signal for scoring AHI using PSG. The literature was analyzed to answer six questions the address the adequacy of different respiratory and effort sensors and combinations to diagnose OSA. The task force provided the following conclusions in response to the six key questions:

- The literature is inadequate to state with confidence that a thermistor alone without any effort sensor is adequate to diagnose OSA. If a thermal sensing device is used as the only measure of respiration, two effort belts are required as part of the montage, and piezoelectric belts are acceptable in this context.
- Nasal pressure can be an adequate measurement of respiration with no effort measure with the caveat that this may be device specific
- Nasal pressure may be used in combination with either two piezo-electric or respiratory inductance plethysmographic (RIP) belts (but not one piezoelectric belt)
- There is insufficient evidence to state that both nasal pressure and thermistor are required to adequately diagnose OSA
- Regarding alternative devices to diagnose OSA:
    - The data indicate that peripheral arterial tonometry (PAT) devices are adequate for the proposed use
    - The device based on cardiac signals shows promise, but more study is required as it has not been tested in the home setting.
    - The device based on end-tidal CO_2 (ETC_2) appears to be adequate for a hospital population
    - For devices using acoustic signals, the data are insufficient to determine whether the use of acoustic signals with other signals, as a substitute for airflow, is adequate to diagnose OSA.

The taskforce stated that future studies for the evaluation of OOC testing devices would greatly benefit by the use of consistent outcome measures to allow direct comparisons and meta-analyses of studies. Standardized research is needed that report a positive likelihood ratio at the appropriate AHI (i.e., ≥ 5), and scored according to the recommended definitions, while using appropriate research reporting and methodology to minimize bias.

AASM Standards for Accreditation of Out of Center Sleep Testing (OCST) in Adult Patients states that the OCST equipment must meet the minimum definitions described in at least one of the CPT codes 95800, 95801 or 95806, or one of the HCPCS codes G0398, G0399 or G0400. (Designated in the guideline as Standard D3) (Refer to the Coding/Billing Information Section of this Coverage Policy for a description of these codes).

The equipment also must have the capability to meet the following accreditation standards:

- OCST Reports–OCST reports must include at minimum:
    - An RDI (an estimate of the apnea and hypopneas per unit time)
    - Evaluation of oxygen saturation during recording period
    - Recording duration of test
    - Technical adequacy of test
- OCST recording Equipment:

- Equipment must provide an RDI based on measures that approximate an AHI based on full polysomnography.
- Equipment must also measure oxygen saturation and heart rate and meet the criteria for the codes designated in Standard D-3 (described above)
- Equipment must allow for the display of raw data for manual scoring or editing.

The guideline also includes quality assurance requirements for the sleep center entity, including a quality assurance program, quality assurance reporting and quality improvement.

A Clinical Guideline for the Evaluation, Management and Long-Term Care of Obstructive Sleep Apnea in Adults (Epstein, et al., 2009) states that the presence of absence and severity of OSA must be determined before initiating treatment in order to identify those at risk for developing complications of sleep apnea, guide treatment, and provide a baseline to evaluate the effectiveness of subsequent treatment. Diagnostic criteria are based on clinical signs and symptoms established during a comprehensive sleep evaluation, which includes a sleep oriented history and physical examination, and findings established by sleep testing. A comprehensive sleep history should include an evaluation for snoring, witnessed apneas, gasping/choking episodes, excessive sleepiness not explained by other factors, including assessment of sleepiness severity by the Epworth Sleepiness Scale, total sleep amount, nocturia, morning headaches, sleep fragmentation/sleep maintenance, insomnia, and decreased concentration and memory. The guideline also states that particular attention should be paid to the presence of obesity, signs of upper airway narrowing, or the presence of other disorders that can contribute to the development of OSA. Features to be evaluated that may suggest the presence of OSA include increased neck circumference (> 17 I men, and > 16 in women), body mass index (BMI) ≥ 30, and various physiologic abnormalities that may compromise respiration (e.g., retrognathia, macroglossia, tonsillar hypertrophy, elongated/enlarged uvula).

The 2009 AASM guideline (Epstein et al.) reaffirmed recommendations provided in a 2007 guideline on the use of unattended portable monitoring (Collop et al.). Recommendations are based on a review of the literature and consensus: The guideline states that portable monitoring may be used in the unattended setting as an alternative to PSG for the diagnosis of OSA in patients with a high pretest probability of moderate to severe OSA and no comorbid sleep disorder or major comormid medical disorders when all the following parameters are met:

- 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.
- A PM should, at a minimum, 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 portable monitors, and include an oronasal thermal sensor to detect apneas, a nasal pressure transducer to measure hypopneas, oximetry, and ideally, inductance plethysmography for respiratory effort.
- 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.
- 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
- A quality/performance improvement program for PM, including inter-scorer reliability must be in place to assure accuracy and reliability.
- 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.

Agency for Healthcare Research and Quality (AHRQ): An AHRQ comparative effectiveness review was conducted in 2011 to systematically review the evidence on OSA diagnosis and treatment in adults. The key questions focused on OSA screening and diagnosis, treatments, associations between apnea-hypopnea index

(AHI) and clinical outcomes, and predictors of treatment compliance. Of the 234 studies that met eligibility criteria, 46 evaluated diagnostic tests. The authors concluded that portable monitors and questionnaires may be effective screening tools, but assessments with clinical outcomes are necessary to prove their value over PSG. This conclusion was based on the following two Key Questions that addressed OSA diagnosis:

Key Question 1
How do different available tests compare in their ability to diagnose sleep apnea in adults with symptoms suggestive of disordered sleep? How do these tests compare in different subgroups of patients, based on race, sex, body mass index, existing non-insulin dependent diabetes mellitus, existing cardiovascular disease, existing hypertension, clinical symptoms, previous stroke, or airway characteristics?

To address this Key Question, three types of comparisons were evaluated: portable monitoring devices (Types II, III, and IV) versus PSG, questionnaires versus PSG or portable monitors, and clinical prediction models versus PSG or portable monitors. Studies included in the 2007 Technology Assessment (discussed below) were not reevaluated. The authors provided the following conclusions:

Portable monitors vs. PSG: The strength of evidence is moderate among 15 quality A, 45 quality B, and 39 quality C studies that Type III and IV monitors may have the ability to accurately predict AHI suggestive of OSA with high positive likelihood ratios and low negative likelihood ratios for various AHI cutoffs in PSG. Type III monitors perform better than Type IV monitors at AHI cutoffs of 5, 10, and 15 events per hour. Analysis of difference vs. average analyses plots suggest that substantial differences in the measured AHI may be encountered between PSG and both Type III and Type IV monitors. Large differences compared to in-laboratory PSG cannot be excluded for all portable monitors. The evidence is insufficient to adequately compare specific monitors to each other.

No recent studies compared Type II monitors with PSG. The prior Technology Assessment concluded that based on three quality B studies, type II monitors used at home may identify AHI suggestive of OSA with high positive likelihood ratios and low negative likelihood ratios, although substantial differences in the AHI may be encountered between type II monitors and facility-based PSG.

Questionnaire vs. PSG
The strength of evidence is low that the Berlin Questionnaire is moderately accurate (sensitivity and specificity generally < 90%) to screen for OSA. The strength of evidence is insufficient to evaluate other questionnaires (TOP, STOP-Bang, ASA Checklist, Epworth Sleepiness Scale, Hawaii Sleep questionnaires).

Clinical Prediction Rules vs. PSG
The strength of evidence is low that some clinical prediction rules may be useful in the prediction of a diagnosis of OSA. Ten different clinical prediction rules have been described (e.g., oropharyngeal morphometric model, pulmonary function data model). While all the models were internally validated, external validation for these predictive rules had not been conducted in the vast majority of the studies.

Key Question 2:
How does phased testing (screening tests or battery followed by full test) compare to full testing alone?
The strength of evidence is insufficient to determine the utility of phased testing, followed by full testing when indicated, to diagnose sleep apnea. Only one study met the inclusion criteria, and this study did not fully analyze the phased testing. The sensitivity and specificity of this phased strategy could not be calculated due to a verification bias; not all participants received PSG testing.

In a discussion of OSA diagnosis, the authors stated that, in theory, OSA is relatively simple to diagnose. PSG, the standard diagnostic test, is inconvenient, resource-intensive, and may not be representative of a typical night's sleep. In addition, there are variations across laboratories in definitions of OSA and in the way results are read and interpreted. AHI, which is used as the single metric to define OSA, can also vary from night to night and does not take into account symptoms, comorbidities, or response to treatment. Numerous portable monitors (evaluated in 99 studies) have been developed for use in non-laboratory settings. These use fewer "channels", or specific physiologic measures than typical 16-channel PSG. Although most of the tested portable monitors fairly accurately predict OSA, it is unclear whether any of these monitors can replace laboratory-based PSG. The evidence suggests that the measured AHI from portable monitors is variable compared with PSG-derived

AHI, but he source of this variability is unclear. No studies have evaluated the predictive ability for clinical outcomes or response to treatment by portable monitors.

Future studies of the accuracy or bias of diagnostic tests should focus more on head-to-head comparisons of portable monitors, questionnaires, and predictive rules to determine the optimal tool for use in a primary care stetting to maximize initial evaluation of OSA and triage high risk patients for prompt PSG. Direct comparisons among existing alternatives to PSG are more important that the current focus on developing new diagnostic tests.

An AHRQ 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 rations. 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: Facility-Based PSG and Portable Monitoring/Home Sleep Studies
Although facility-based PSG has been considered the standard method for evaluation of OSA, it cannot be considered the “gold standard”, since a true gold standard would include a defined set of criteria or measurements to distinguish patients with OSA from those without OSA. An AHI suggestive of OSA is not sufficient for the diagnosis of the condition, since the severity of symptoms must be accounted for, and other conditions that affect sleep must be excluded. A gold-standard would also have prognostic ability, since patients with OSA have a different prognosis than those without OSA. Although the published evidence comparing PSG with home/portable testing has demonstrated that PSG more accurately measures AHI, AHI is not well correlated with response to CPAP therapy or compliance with therapy. The increased accuracy of the AHI obtained by facility-based PSG therefore may not be predictive of outcomes. In addition, the precise accuracy of

PSG may be impacted by several factors, including inter-reader variability, use of different test instruments, an individual's night to night variability, and ability to sleep in a non-home setting (AHRQ, 2007, CMS, 2009).

As diagnostic tests, PSG and HST would not be expected to directly change health outcomes, but would affect outcomes through changes in disease management by actions taken in response to the test results. The usefulness of a test result is constrained somewhat by the available treatment options. The number of practical treatment options for OSA is limited; most patients are treated with CPAP, and a small number are treated with oral appliances or surgery (CMS, 2009).

There is adequate evidence to demonstrate that portable monitoring/home sleep studies accurately predict AHI suggestive of OSA with high positive likelihood ratios and low negative likelihood ratios in patients with a high pretest probability of OSA. Comparative effectiveness studies that have evaluated clinical outcomes of patients managed with home testing vs. those managed with PSG demonstrated similar outcomes in terms of functional improvement (e.g., sleepiness scores, activity level, vigilance, productivity), and CPAP adherence. Home sleep studies are not indicated, however, for individuals with significant comorbid medical conditions that may degrade the accuracy of portable testing, including moderate to severe pulmonary disease, neuromuscular disease, obesity-hypoventilation syndrome, or heart failure. Home testing has not been evaluated for, and/or does not include the diagnostic data necessary for those suspected of having other sleep disorders.

Most studies of home sleep testing have evaluated Type III devices that measure two respiratory variables (eg, respiratory movement and airflow), a cardiac variable (eg, heart rate or an electrocardiogram), and arterial oxyhemoglobin saturation via pulse oximetry. Some devices also include signals that can detect snoring, determine body position, or detect movement. Type IV devices or continuous single or dual bioparameter recording, generally use oximetry and may employ a second airflow assessment parameter. Type IV devices provides limited information; they do not measure sleep time and cannot distinguish between obstructive and central apneas. There is insufficient evidence in the published medical literature to determine the diagnostic accuracy of Type IV studies.

A full night or split night facility-based PSG may be indicated when recent portable monitoring was technically inadequate or failed to establish the diagnosis in an individual with a high pretest probability of OSA; when a sleep disorder other than OSA is suspected, or when a significant comorbid medical condition exists, including moderate to severe pulmonary disease, neuromuscular disease, obesity-hypoventilation syndrome, or heart failure. In-facility PSG may also be indicated for PAP titration; when the PAP titration portion of a prior split-night study was insufficient; or prior to a planned multiple sleep latency test (MSLT) when narcolepsy is suspected.

Subsequent in-facility PSG or home/portable testing may be indicated when the diagnosis of OSA has been established, in order to assess outcomes following OSA treatment or to evaluate a return of symptoms or inadequate clinical response to treatment.

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.

Non-Surgical Treatment

Agency for Healthcare Research and Quality (AHRQ)
An AHRQ Comparative Effectiveness Review, Diagnosis and Treatment of Obstructive Sleep Apnea in Adults (discussed in the diagnosis section above) included the following key questions and conclusions regarding treatment with PAP and mandibular advancement devices (MAD):

Key Question: What is the comparative effect of different treatments for obstructive sleep apnea in adults?

- Despite no evidence or weak evidence on clinical outcomes, given the large magnitude of effect on the important intermediate outcomes AHI, ESS and other sleep study measures, the strength of evidence is moderate that CPAP is an effective treatment for OSA. However, the strength of evidence is insufficient to determine which patients might benefit most from treatment.
- Despite no or weak evidence on clinical outcomes, overall there is moderate strength of evidence that autoCPAP and fixed CPAP result in similar compliance and treatment effects for patients with OSA.
- The strength of evidence is low of no substantial difference in compliance or other outcomes between C-Flex and CPAP.

- The strength of evidence is insufficient regarding comparisons of different CPAP devices or modifications
- Despite no evidence or weak evidence on clinical outcomes, given the large magnitude of effect on the important intermediate outcomes AHI, ESS and other sleep study measures, overall the strength of evidence is moderate that MAD is an effective treatment for OSA in patients without comorbidities (including periodontal disease) or excessive sleepiness. However, the strength of evidence is insufficient to address which patients might benefit most from treatment.
- The strength of evidence is insufficient regarding comparisons of different oral devices.
- Despite no evidence or weak evidence on clinical outcomes, overall the strength of evidence is moderate that the use of CPAP is superior to MAD. However, the strength of evidence is insufficient to address which patients might benefit most from either treatment.
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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.

Bakker and Marshall (2011) conducted a systematic review and meta-analysis to compare flexible and standard CPAP in adult patients with OSA. A meta-analysis of seven trials was performed to quantify improvements in objective compliance and symptoms as measured by the Epworth Sleepiness Scale (ESS), Maintenance of Wakefulness Test (MWT), and the Psychomotor Vigilance Task (PVT). Flexible pressure did not improve

compliance in either the parallel trials (p=.21) or the crossover trials (p=.39). Flexible pressure compared to CPAP cause no improvement in any secondary outcome (ESS, MWT, PVT, and residual OSA, all p> .05). The systematic review and meta-analysis found no significant evidence that C-Flex provides any benefit over standard CPAP in terms of compliance, subjective or objective sleepiness, or psychomotor vigilance in patients with moderate to severe OSA.

Bakker et al. conducted a double-blind, parallel-arm randomized controlled trial to compare compliance with C-Flex and CPAP and to analyze measures of sleepiness and vigilance. Consecutive patients with severe OSA (mean AHI 60.2 ± 32.9; Epworth Sleepiness Scale [ESS] 13.6 ± 4.5/24) were randomized to C-Flex (n=39) or CPAP (n=41). Patients underwent titration with C-Flex/CPAP. Median compliance after three months of treatment was 5.51 hours per night for C-Flex vs. 5.89 hours for CPAP (p=0.82). There was no significant difference between the groups in terms of psychomotor vigilance tasks (PVT) reaction time, subjective sleepiness, sleep quality, health-related quality of life, or treatment comfort. In patients with severe OSA, both CPAP and C-Flex resulted in substantial improvements, but neither treatment appeared superior.

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 it has been proposed that the addition of expiratory pressure relief (e.g., C-Flex) may result in increased comfort and compliance, there is insufficient evidence to demonstrate that the addition of expiratory pressure relief to CPAP, APAP or BPAP, results in improved treatment adherence or clinical outcomes compared to PAP alone.

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 (CHF, 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)

Oral Pressure Therapy
Oral pressure therapy has also been proposed for the treatment of OSA. The Winx® Sleep Therapy System (ApriCure, Inc., Redwood City CA) received FDA approval through the 510(k) process on May 22, 2013. An earlier version of the device was approved in 2012. The Winx Sleep Therapy System consists of a small electronic bedside console, a soft polymer mouthpiece, a flexible polymer tube that connects the mouthpiece to the console, and a physician's software application. The mouthpiece is an intraoral device that is worn during sleep. The system is designed to increase airway patency and decrease airway obstruction by delivering a gentle negative pressure into the oral cavity and holding the tongue and soft palate out of the airway. Published evidence evaluating the use of this device is limited to feasibility studies and a small case series (Colrain et al., 2013). There is insufficient evidence to determine the safety and efficacy of this system for the treatment of OSA.

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, or during a full-night PSG that follows a diagnostic PSG in which the diagnosis of OSA is established. In-facility PSG, rather than home/portable testing, is indicated only for patients who are not suitable candidates for home testing due to medical comorbidities, or when sleep disorders other than OSA are suspected. When a diagnosis of OSA is established following a home/portable study, home titration to determine a fixed CPAP pressure can be effectively completed using auto-titrating positive airway pressure.

Gao et al. (2011) conducted a systematic review to evaluate the effect of automatic titration compared to manual titration prior to CPAP treatment in OSA patients. The authors evaluated APAP in identifying an effective pressure and the improvement of AHI and somnolence, change in sleep quality, and the acceptance and compliance of CPAP treatment compared to manual titration. Ten randomized controlled trials (849 patients)

met the inclusion criteria. Studies were pooled to yield odds ratios (OR) or mean differences (MD) with 95% confidence intervals (CI). Automatic titration improved the AHI (MD=0.03/h, 95% CI=4.48-4.53) and Epworth sleepiness scale (SMD=0.02, 95% CI=0.34-0.31) as effectively as manual titration. There was no difference in sleep architecture between auto titration and manual titration. There was also no difference in acceptance of CPAP treatment or compliance with treatment. The authors concluded that automatic titration is as effective as standard manual titration in terms of improvement in AHI, somnolence, and sleep quality, as well as acceptance and adherence to CPAP.

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 H_20. After one week of use, the APAP device was interrogated. The 95^{th} 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 H_20, home 10.4 ± 0.2 cm H_20, 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 (CHF, 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).

Adherence to PAP Therapy
The ability of PAP to reverse the repetitive upper airway obstruction of sleep apnea is dramatic. PAP has been demonstrated to normalize sleep architecture, reduce daytime sleepiness, enhance daily functioning, elevate mood, reduce auto accidents, and decrease blood pressure and cardiovascular events. Despite the efficacy of CPAP, studies evaluating adherence report high rates of non-adherence. Adherence to PAP therapy is usually defined as ≥ four hours of CPAP usage for ≥ 70% of the nights monitored, based on a 1993 prospective study by Kribbs et al., evaluating patterns of CPAP use. Patient reports of the frequency and duration of CPAP use frequently overestimate actual use. The average duration of CPAP use is approximately five hours per night, as reported in numerous studies. The available evidence indicates that CPAP used for more than six hours per night results in normal levels of objectively measured and subjectively reported daytime sleepiness, and improved daily functioning (Kribbs et al., 1993; Gay et al., 2006; Weaver and Grundstein, 2008).

As stated above, adherence to PAP therapy is usually defined as ≥ four hours of CPAP usage for ≥ 70% of the nights monitored. Patients with borderline adherence to PAP therapy (e.g., 55%-69% of nights for at least three hours but less than four hours per night, may require intervention to evaluate barriers to treatment. According to the AASM Clinical Guideline, Evaluation, Management, and Long-Term Care of Obstructive Sleep Apnea in Adults (Epstein et al., 2009) CPAP usage should be objectively monitored with time meters to help assure utilization. The guidelines also recommend close follow-up for PAPA usage and problems by appropriately trained health care providers to establish effective utilization patterns and remediate problems, if needed. This is especially important during the first few weeks of PAP use.

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. An MRA, also referred to as mandibular advancement appliances (MAA) mandibular advancement device (MAD) or mandibular advancement splint (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**.**

A randomized controlled crossover trial was conducted by Phillips et al. (2013) to evaluate the health outcomes of optimal CPAP therapy compared to use of a mandibular advancement device (MAD). A total of 126 patients with moderate to severe OSA were randomly assigned to a treatment order and 108 completed the trial with both devices. The reduction in AHI was greater with CPAP than with MAD (CPAP AHI, 4.5 ± 6.6/hour; MAD AHI 11.1 ± 12.1/hour, $p < 0.01$), but compliance was higher with MAD (6.50 ± 1.3 hours/night vs. 5.20 ± 2 hours/night, $p<0.00001$). The 24-hour mean arterial pressure was not inferior on treatment with MAD compared to CPAP. Neither treatment improved blood pressure. Sleepiness, driving simulator performance, and disease-specific quality of life improved on both treatments by similar amounts, but MAD was superior to CPAP for improving four general quality of life domains. The authors stated that the similar results in terms of important health outcomes may be explained by greater efficacy of CPAP being offset by inferior compliance compared to MAD.

Aarab et al. (2010) conducted a randomized placebo-controlled trial to compare treatment effects of oral appliance therapy and CPAP in patients with OSA. Patients with mild to moderate OSA were randomized to one of three parallel groups: a titrated mandibular advancement device (MAD) (n=20); nasal CPAP (n=18) or an intraoral placebo device (n=19), In-hospital PSG was performed in all patients prior to treatment and after approximately six months of treatment initiation. No differences in the AHI were found between the MAD and CPAP groups ($p=0.092$). The change in AHI in these groups was significantly greater than in the placebo group. The authors concluded that there is no clinically relevant difference between MAD and CPAP in the treatment of mild to moderate OSA when both treatment modalities are titrated objectively.

A European Respiratory Society (ERS) task force report evaluated non-CPAP therapies, including mandibular advancement devices (MADs), for the treatment of OSA (Randerath et al., 2011). The report states that MADs reduce sleep apneas and subjective daytime sleepiness and improve quality of life compared to control treatments. CPAP is more effective at reducing the number of sleep apneas, but the positive effects on symptoms and health are similar, and patients generally prefer MAD over CPAP. The device should be custom-made, evaluated, and should advance the mandible at least 50% of maximal protrusion. The authors noted that a titration procedure is essential, since the improvement in symptoms is not a precise indicator of treatment success, and long-term follow-up should be performed. Tongue retaining devices (TRD), however, were not recommended for patients with OSA. They may be used, however, in selected patients with mild to moderate OSA when other treatments have failed or are not possible. Patients may have a trial with the device if treatment effect is monitored and strict follow-up is performed.

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 tracheotomy, 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: tracheotomy; 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 tongue base suspension with the AIRVance System (Influence Corp; San Francisco, CA), and base-of-tongue Somnoplasty.

American Academy of Sleep Medicine (AASM): Practice Parameters for the Surgical Modification of the Upper Airway for Obstructive Sleep Apnea in Adults (Aurora et al., 2010), based on a systematic review of the literature (Caples et al, 2010) updated earlier practice parameters published in 1996.

Recommendations are classified as Standard, Guideline, or Option, in descending order based on the benefits vs. harms and the quality of evidence. Recommendations for individual procedures are included in the relevant sections below.

<u>Standard:</u>
- The presence and severity of obstructive sleep apnea (OSA) must be determined before initiating surgical therapy

- The patient should be advised about potential surgical success rates and complications, the availability of alternative treatment options such as nasal positive airway pressure and oral appliances, and the levels of effectiveness and success rates of these alternative treatments.
- The desired outcomes of treatment include resolution of the clinical signs and symptoms of OSA and the normalization of sleep quality, the apnea-hypopnea index, and oxyhemoglobin saturation levels.

Option

- Maxillo-mandibular advancement (MMA) is indicated for surgical treatment of severe OSA in patients who cannot tolerate or who are unwilling to adhere to positive airway pressure therapy, or in whom oral appliances, which are more often appropriate in mild and moderate OSA patients, have been considered and found ineffective or undesirable.
- Uvulopalatopharyngoplasty (UPPP) as a sole procedure, with or without tonsillectomy, does not reliably normalize the apnea hypopnea index (AHI) when treating moderate to severe OSA syndrome. Therefore, patients with severe OSA should initially be offered positive airway pressure (PAP) therapy, while those with moderate OSA should initially be offered either PAP therapy or oral appliances.
- Use of multi-level or stepwise surgery (MLS), as a combined procedure or as stepwise multiple operations, is acceptable in patients with narrowing of multiple sites in the upper airway, particularly if they have failed UPPP as a sole treatment.
- Laser-assisted uvulopalatoplasty (LAUP) is not routinely recommended as a treatment for obstructive sleep apnea syndrome.
- Radiofrequency ablation (RFA) can be considered as a treatment in patients with mild to moderate OSA who cannot tolerate or who are unwilling to adhere to PAP therapy, or in whom oral appliances have been considered and found ineffective or undesirable.
- Palatal implants may be effective in some patients with mild OSA who cannot tolerate or who are unwilling to adhere to PAP therapy, or in whom oral appliances have been considered and found ineffective or undesirable.

An AHRQ comparative effectiveness review was conducted in 2011 to systematically review the evidence on OSA diagnosis and treatment in adults (discussed above in the diagnosis section). The review provided the following conclusions regarding surgical treatment of OSA:

Key Question: What is the comparative effect of different treatments for OSA in adults?

- The strength of evidence is insufficient to determine the relative merits of surgical treatments versus CPAP.
- The strength of evidence is insufficient regarding the relative merit of mandibular advancement devices versus surgery in the treatment of OSA.

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).

The recommendation for UPPP in the 2010 AASM practice parameters for surgical modification of the upper airway (Aurora, et al.), discussed above, states that UPPP does not reliably normalize the AHI in moderate to severe OSA; patients with severe OSA should therefore initially be offered PAP therapy, while those with moderate OSA should initially be offered either PAP therapy or an oral appliance. This recommendation differs from the previously published guideline that recommended UPPP for patients with narrowing or collapse of the retropalatal area.

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). The AHI and snoring were reduced in one trial after LAUP but not in another. 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 (UPP), with difficulty swallowing, globus sensation, and voice changes commonly observed.

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.

Tracheostomy: AASM practice parameters (Aurora et al., 2011), discussed above state that tracheostomy has been shown to be an effective single intervention to treat OSA. This operation should be considered only when other options do not exist, have failed, are refused, or when this operation is deemed necessary by clinical urgency. This recommendation is considered an Option; although tracheostomy is nearly always successful in bypassing the upper airway obstruction and normalizing AHI, it is not recommended as primary therapy based on placing a high value on patient safety, autonomy, and quality of life.

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.

AASM 2010 Practice Parameters for the Surgical Modification of the Upper Airway for OSA, as noted above, state that LAUP is not routinely recommended. The evidence was judged to be low quality LAUP does not generally normalize the AHI, and the literature does not demonstrate significant improvement in secondary outcomes. Two studies performed since the last review in 2001 actually reported worsening of the overall AHI.

Uvulectomy: 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.

A meta-analysis of the efficacy of the Pillar implant in the treatment of snoring and OSA was conducted by Choi et al. (2013). Efficacy for snoring (seven studies) and for mild to moderate OSA (seven studies) was analyzed separately. For patients with mild to moderate OSA, the Pillar implant significantly reduced the Epworth Sleepiness Scale (p<.001) and AHI (p=.002) compared to pre-procedure values. The authors noted that these results indicate that the Pillar implant has a moderate effect on snoring and mild to moderate OSA, but more studies with a high level of evidence are needed to arrive at a definite conclusion.

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 ≤ 32 kg/m^2, 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 ± 13.9 to 22.0 ± 14.8 events/hour (p=0.05). ESS scores, a secondary outcome measure, decreased from 11.0 ± 5.1 to 6.9 ± 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.

AASM Practice Parameters for the Surgical Modification of the Upper Airway for OSA (Aurora et al., 2011) discussed above, state that palatal implants may be effective in some patients with mild obstructive sleep apnea who cannot tolerate or are unwilling to adhere to PAP therapy, or in whom oral appliances have been considered and found ineffective or undesirable. Evidence is of very low quality, and while this procedure may be an alternate mode of therapy for mild OSA, it is difficult to predict if it will ultimately be found to be a reliably effective intervention.

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 otolarygological (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.

AASM practice parameters discussed above (Aurora et al., 2010) state that RFA can be considered in patients with mild to moderate OSA who cannot tolerate or are unwilling to adhere to PAP therapy, or in whom oral appliances have been considered and found ineffective or undesirable. This is noted to be a new recommendation based on very low quality evidence. The average post-procedure AHI was found in 7 case series and one randomized controlled trial to be 14.9, consistent with residual mild OSA. The authors noted that RFA studies have shown improvement in subjective sleepiness and, in one study, quality of life. Because cardiovascular complications of OSA are associated with even lower values of AHI, patients treated with RFA should receive follow-up assessments for residual AHI, even if symptoms have improved. The authors also note that long-term sequelae of RFA are not published.

The systematic review by Franklin et al. (2009) to evaluate the efficacy and adverse effects of surgery for snoring and OSA, discussed above, concluded that there was no significant effect on daytime sleepiness and quality of life after radiofrequency ablation.

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.

Multi-Level or Stepwise surgery (MLS): This category includes a wide array of combined procedures that address narrowing of multiple upper airway sites. MLS often consists of phase I, utilizing UPPP and/or genioglossus advancement and hyoid myotomy (GAHM). Phase II procedures, consisting of maxillary and mandibular advancement osteotomy (MMO), may be considered for patients who fail phase I surgeries (Aurora et al., 2011).

AASM Practice Parameters for the Surgical Modification of the Upper Airway for OSA (Aurora et al, 2011) discussed above state that use of multi-level or stepwise surgery (MLS), as a combined procedure or as stepwise multiple operations, is acceptable in patients with narrowing of multiple sites in the upper airway, particularly if they have failed UPPP as a sole treatment. Although a large volume of literature addressing MLS exists, the evidence is of low quality, consisting of observational case series or comparative studies without randomization. While a multilevel approach may eventually result in significant improvement in AHI, available data are heterogeneous, clinical outcomes such as cardiovascular events are not well studies, and multiple procedures could be associated with increased morbidity and mortality.

Maxillomandibular Advancement (MMA): Maxillomandibular advancement is a surgical procedure that involves the simultaneous advancement of the maxilla and mandible through sagittal split osteotomies. The procedure provides enlargement of the retrolingual airway, and some advancement of the retropalatal airway (Aurora et al., 2011).

Holty and Guilleminault (2010) conducted a systematic review and meta-analysis of 22 studies (627 patients) to evaluate the clinical efficacy and safety of maxillomandibular advancement for the treatment of OSA. The mean AHI decreased from 63.9/hour to 9.5/hour ($p<0.001$) following surgery. The pooled surgical success and cure (AHI<5) rates were 86.0% and 43.2%, respectively. Younger age, lower preoperative weight and AHI, and greater degree of maxillary advancement were predictive of increased surgical success. The major and minor complication rates were 1.0% and 31%, respectively. Long-term surgical success was maintained at a mean follow-up of 44 months. Statistically significant improvements in quality of life measures, OSA symptomatology (i.e., excessive daytime sleepiness) and blood pressure control were reported after MMA. The authors concluded that MMA appears to be a safe and highly effective treatment for OSA, but further research is needed to assess clinical outcomes of MMA more thoroughly in long-term cohort studies, and to identify which OSA patients would benefit most from MMA.

AASM Practice Parameters for the Surgical Modification of the Upper Airway for OSA (Aurora et al., 2011), discussed above, state that MMA is indicated for surgical treatment of severe OSA in patients who cannot tolerate or who are unwilling to adhere to positive airway pressure therapy, or in whom oral appliances, which are more often appropriate in mild and moderate OSA patients, have been considered and found ineffective or undesirable. The evidence was considered to be very low quality, consisting of nine case series, but did tend to demonstrate consistent effectiveness in severe OSA. In the published series, AHI was reduced to at least 10/hour in most patients, but PAP remains more effective in normalizing AHI, and improvement in other measures such as sleepiness and quality of life are well supported for PAP but are lacking for MMA. PAP or oral appliance therapy therefore should be suggested ahead of MMA in appropriate candidates.

Traditional “stepped” care frequently utilizes MMA as a final approach for surgical treatment of OSA, but MMA may be considered as an initial or sole approach in treating OSA. The authors recommended multidisciplinary evaluation to identify which patients would benefit from MMA as initial or sole therapy. There is a need for further clarification regarding the relative risks and benefits of MMA compared with other treatment modalities,

The AIRVance System: The Repose Bone Screw System (Influence, Inc., San Francisco, CA) received FDA 510(k) approval on August 27, 1999. The device name was changed to AIRVance in 2011, and is marketed by MedTronic. The 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 AIRVance System has been proposed as a sole treatment of OSA and has also been use 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.

Transpalatal Advancement Pharyngoplasty
Transpalatal Advancement Pharyngoplasty has been proposed as an alternative to traditional methods of reconstructing the upper pharyngeal airway. The procedure enlarges and stabilizes the upper pharyngeal airway by altering bone and soft tissue attachments of the posterior maxilla. The concept of transpalatal advancement pharyngoplasty is based on OSA pathophysiology in which the primary craniofacial predictor of OSA severity is maxillary constriction. The procedure increases the retropalatal airway size by combining a posterior maxillectomy with soft palate mobilization. Evidence evaluating this technique is limited, consisting primarily of retrospective reviews. There is insufficient evidence in the published medical literature to determine the safety and efficacy of this procedure or to determine how it compares to available treatment options for OSA.

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.

Berry et al. (2010) conducted a randomized controlled trial to evaluate treatment with expiratory positive airway pressure (EPAP). Patients with OSA with an AHI of ≥ 10 were assigned to treatment with the Provent device (n=127) or a similar-appearing sham device (n=123) for three months. During the first week of treatment, after at least three nights of device use, PSG was performed on two non-consecutive nights once with and once without the device. After three months of treatment, patients were re-evaluated, and two additional PSGs were performed on non-consecutive nights (device on, device off). At week one, the median AHI (device on vs. off) was significantly lower with EPP (5.0 vs. 13.8 events/hour, $p<0.0001$) but not with sham treatment (11.6 vs. 11.1

events/hour). Over three months of treatment, Epworth Sleepiness Scores decreased from 9.9 ± 4.7 to 7.2 ± 4.2, p<0.0001), and the median percentage of reported nights used for the entire night was 88.2%.The authors acknowledged limitations of the study, including the large number of exclusion criteria that prevent generalizing the results of this study to less selected populations, and the fact that adherence determination was based on patient reporting rather than objective data.

Although EPAP with the use of the Provent device is a promising treatment option, additional well-designed studies are needed to determine how this device compares to currently available options in the treatment of OSA in terms of safety, efficacy, and long-term outcomes.

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 my 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.

Diagnosis of OSA-Child:
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.

American Academy of Pediatrics (AAP): An AAP Clinical Practice Guideline, Diagnosis and Management of Childhood Obstructive sleep Apnea Syndrome, was published in 2012 (Marcus et al.). The guideline focuses on uncomplicated childhood OSA, i.e., the OSA associated with adenotonsillar hypertrophy and/or obesity in an otherwise healthy child being treated in the primary care setting. The guideline defines OSA in children, consistent with the ATS definition above) 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 normal sleep patterns", accompanied by the following symptoms or signs:

- History
  - Frequent snoring (≥3 nights/wk)
  - Labored breathing during sleep
  - Gasps/snorting noises/observed episodes of apnea
  - Sleep enuresis (especially secondary enuresis)after at least 6 mo of continence
  - Sleeping in a seated position or with the neck hyperextended
  - Cyanosis
  - Headaches on awakening
  - Daytime sleepiness
  - Attention-deficit/hyperactivity disorder
  - Learning problems
- Physical examination
  - Underweight or overweight
  - Tonsillar hypertrophy
  - Adenoidal facies
  - Micrognathia/retrognathia
  - High-arched palate
  - Failure to thrive
  - Hypertension

Evidence grading used in the Key Action Statements ranges from A (randomized controlled trials or diagnostic studies on relevant population) to D (expert opinion, case reports, reasoning from first principles), with an additional category of X (exceptional situations where validating studies cannot be performed and there is a clear preponderance of benefit or harm). Recommendations are designated as strong recommendation, recommendation, option, or no recommendation.

The guideline includes the following key action statements regarding testing for OSA:

<u>Polysomnography</u>
If a child or adolescent snores on a regular basis and has any of the [above] complaints or findings, clinicians should either

- obtain a polysomnogram (Evidence Quality A, Key Action strength: Recommendation) OR

- refer the patient to a sleep specialist or otolaryngologist for a more extensive evaluation (Evidence quality D, Key Action strength: Option). (Evidence Quality: Grade A for polysomnography; Grade D for specialist referral, Recommendation Strength: Recommendation.)

Alternative Testing

- If polysomnography is not available, then clinicians may order alternative diagnostic tests, such as nocturnal video recording, nocturnal oximetry, daytime nap polysomnography, or ambulatory polysomnography. (Evidence Quality: Grade C, Recommendation Strength: Option.)

American Academy of Sleep Medicine (AASM): Practice Parameters for the Respiratory Indications for Polysomnography in Children, based on a systematic review of the literature. (Aurora et al., 2011), classifies recommendations as follows:

- Standard: A generally accepted patient-care strategy that reflects a high degree of clinical certainty and generally implies the use of Level 1 evidence or overwhelming Level 2 evidence.
- Guideline: A patient-care strategy that reflects a moderate degree of clinical certainty and implies the use of Level 2 evidence or a consensus of Level 3 evidence
- Option: A patient care strategy that reflects uncertain clinical use and implies inconclusive or conflicting evidence or conflicting expert opinion.

Recommendations for PSG use include the following:

Standard

1. Polysomnography in children should be performed and interpreted in accordance with the recommendations of the AASM Manual for the Scoring of Sleep and Associated Events.
2. Polysomnography is indicated when the clinical assessment suggests the diagnosis of obstructive sleep apnea syndrome (OSAS) in children.
3. Children with mild OSAS preoperatively should have clinical evaluation following adenotonsillectomy to assess for residual symptoms. If there are residual symptoms of OSAS, polysomnography should be performed.
4. Polysomnography is indicated following adenotonsillectomy to assess for residual OSAS in children with preoperative evidence for moderate to severe OSAS, obesity, craniofacial anomalies that obstruct the upper airway, and neurologic disorders (e.g., Down syndrome, Prader-Willi syndrome, and myelomeningocele).
5. Polysomnography is indicated for positive airway pressure (PAP) titration in children with obstructive sleep apnea syndrome.

Guideline

1. Polysomnography is indicated when the clinical assessment suggests the diagnosis of congenital central alveolar hypoventilation syndrome or sleep related hypoventilation due to neuromuscular disorders or chest wall deformities. It is indicated in selected cases of primary sleep apnea of infancy.
2. Polysomnography is indicated when there is clinical evidence of a sleep related breathing disorder in infants who have experienced an apparent life-threatening event (ALTE).
3. Polysomnography is indicated in children being considered for adenotonsillectomy to treat obstructive sleep apnea syndrome.
4. Follow-up PSG in children on chronic PAP support is indicated to determine whether pressure requirements have changed as a result of the child's growth and development, if symptoms recur while on PAP, or if additional or alternate treatment is instituted.

Option

1. Polysomnography is indicated after treatment of children for OSAS with rapid maxillary expansion to assess for the level of residual disease and to determine whether additional treatment is necessary.
2. Children with OSAS treated with an oral appliance should have clinical follow-up and polysomnography to assess response to treatment.
3. Polysomnography is indicated for noninvasive positive pressure ventilation (NIPPV) titration in children with other sleep related breathing disorders.

4. Children treated with mechanical ventilation may benefit from periodic evaluation with polysomnography to adjust ventilator settings.
5. Children treated with tracheostomy for sleep related breathing disorders benefit from polysomnography as part of the evaluation prior to decannulation. These children should be followed clinically after decannulation to assess for recurrence of symptoms of sleep related breathing disorders.
6. Polysomnography is indicated in the following respiratory disorders only if there is a clinical suspicion for an accompanying sleep related breathing disorder: chronic asthma, cystic fibrosis, pulmonary hypertension, bronchopulmonary dysplasia, or chest wall abnormality such as kyphoscoliosis.

Recommendations against PSG Use:

1. Nap (abbreviated) polysomnography is not recommended for the evaluation of obstructive sleep apnea syndrome in children. (Option)
2. Children considered for treatment with supplemental oxygen do not routinely require polysomnography for management of oxygen therapy. (Option)

Regarding home/portable testing, the guideline states, "Unattended testing outside the sleep laboratory in children has been used predominantly in research settings. There is a paucity of research comparing it to traditional in-laboratory attended sleep studies or other objective clinical outcomes, and there are insufficient data upon which to base reliable clinical recommendations for children at this time."

The authors concluded that current evidence in pediatric sleep medicine indicates that PSG has clinical utility in the diagnosis and management of sleep related breathing disorders, and that accurate diagnosis in the pediatric population is best accomplished by integration of PSG findings with clinical evaluation.

American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS): A Clinical Practice Guideline: Polysomnography for Sleep-Disordered Breathing Prior to Tonsillectomy in Children (Roland et al., 2011) provides recommendations for using PSG in assessing children, aged 2 to 18 years, who are candidates for tonsillectomy, with or without adenoidectomy. Recommendations pertaining to indications for PSG include the following:

- Indications for PSG: Before performing tonsillectomy, the clinician should refer children with sleep disordered breathing (SDB) for PSG if they exhibit any of the following: obesity, Down syndrome, craniofacial abnormalities, neuromuscular disorders, sickle cell disease, or mucopolysaccharidoses. This recommendation is based on observational studies with a preponderance of benefit over harm.
- Advocating for PSG: The clinician should advocate for PSG prior to tonsillectomy for SDB in children without any of the comorbidities listed above for whom the need for surgery is uncertain or when there is discordance between tonsillar size on physical examination and the reported severity of SDB. This recommendation is based on observational and case-control studies with a preponderance of benefit over harm.
- Unattended PSG with portable monitoring device: In children for whom PSG is indicated to assess SDB prior to tonsillectomy, clinicians should obtain laboratory-based PSG, when available. This recommendation is based on diagnostic studies with limitations and a preponderance of benefit over harm.

Alternatives to PSG in Children: 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
There is adequate evidence to demonstrate that most portable monitoring/home sleep studies accurately predict AHI suggestive of OSA with high positive likelihood ratios and low negative likelihood ratios in patients with a high pretest probability of OSA. Comparative effectiveness studies that have evaluated clinical outcomes of patients managed with home testing vs. those managed with PSG demonstrate similar outcomes in terms of functional improvement (e.g., sleepiness scores, activity level, vigilance, productivity), and adherence to positive airway pressure (PAP) treatment. There is insufficient evidence in the published medical literature to determine the diagnostic accuracy of Type IV studies, however. Home sleep studies are not indicated for individuals with significant co-morbid medical conditions that may degrade the accuracy of portable testing, however, Home testing has not been evaluated for, and/or does not include the diagnostic data necessary for those suspected of having other sleep disorders.

Full-night or split-night facility-based PSG may be indicated for those with suspected OSA who have significant medical comorbidities that could degrade the accuracy of home/portable testing, or are suspected to have sleep disorders other than OSA. Facility-based PSG may also be indicated when portable monitoring is technically inadequate or fails to establish the diagnosis in an individual with a high pretest probability of OSA, or when the individual and caregiver/companion is incapable of operating home testing equipment.

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. 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), maxillomandibular advancement (MMA), and multi-level or stepwise surgery (MLS). 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®, tongue-base suspension using the AIRVance System, transpalatal advancement pharyngoplasty, 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: 1) This list of codes may not be all-inclusive.
2) Deleted codes and codes which are not effective at the time the service is rendered may not be eligible for reimbursement

DIAGNOSTIC TESTING

Polysomnography (PSG)

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

CPT®* Codes	Description
95782	Polysomnography; younger than 6 years, sleep staging with 4 or more additional parameters of sleep, attended by a technologist (New code effective 01/01/2013)
95783	Polysomnography; younger than 6 years, 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 (New code effective 01/01/2013)
95808	Polysomnography; any age, sleep staging with 1-3 additional parameters of sleep, attended by a technologist
95810	Polysomnography; age 6 years or older, sleep staging with 4 or more additional parameters of sleep, attended by a technologist
95811	Polysomnography; age 6 years or older, 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

Home/Portable Testing

Covered when medically necessary for the diagnosis of obstructive sleep apnea in an adult (age 18 or older):

Note: Home/Portable Testing is Experimental/Investigational/Unproven/Not Covered in a child (less than age 18).

CPT®* Codes	Description
95800	Sleep study, unattended, simultaneous recording: heart rate, oxygen saturation, respiratory analysis (eg, by airflow or peripheral arterial tone) and sleep time
95801	Sleep study, unattended, simultaneous recording: minimum of heart rate, oxygen saturation, and respiratory analysis (e.g., by airflow or peripheral arterial tone
95806	Sleep study, unattended, simultaneous recording of, heart rate, oxygen

	saturation, respiratory airflow, and respiratory effort (eg, thoracoabdominal movement)

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

Experimental/Investigational/Unproven/Not Covered:

CPT®* Codes	Description
95807-52†	Sleep study, simultaneous recording of ventilation, respiratory effort, ECG or heart rate, and oxygen saturation, attended by a technologist (reduced services)
G0400	Home sleep test (HST) with type IV portable monitor, unattended; minimum of 3 channels

†**Note: Experimental/Investigational/Unproven/Not Covered when used to report an abbreviated cardiorespiratory sleep study to acclimate an individual to PAP (e.g., PAP-Nap study)**

Multiple Sleep Latency Testing, Maintenance of Wakefulness Testing

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

Actigraphy Testing

Experimental/Investigational/Unproven/Not Covered:

CPT®* Codes	Description
95803	Actigraphy testing, recording, analysis, interpretation, and report (minimum of 72 hours to 14 consecutive days of recording)

TREATMENT OF OBSTRUCTIVE SLEEP APNEA

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

Note: Not Medically Necessary/Not Covered for the treatment of snoring in the absence of obstructive sleep apnea:

CPT®* Codes	Description
21193	Reconstruction of mandibular rami, horizontal, vertical, C, or L osteotomy; without bone graft
21194	Reconstruction of mandibular rami, horizontal, vertical, C, or L osteotomy; with bone graft (includes obtaining graft)
21195	Reconstruction of mandibular rami and/or body, sagittal split; without internal

	rigid fixation
21196	Reconstruction of mandibular rami and/or body, sagittal split; with internal rigid fixation
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**
A4604	Tubing with integrated heating element for use with positive airway pressure device
A7027	Combination oral/nasal mask, used with continuous positive airway pressure device, each
A7028	Oral cushion for combination oral/nasal mask, replacement only, each
A7029	Nasal pillows for combination oral/nasal mask, replacement only, pair
A7030	Full face mask used with positive airway pressure device, each
A7031	Face mask interface, replacement for full face mask, each
A7032	Cushion for use on nasal mask interface, replacement only, each
A7033	Pillow for use on nasal cannula type interface, replacement only, pair
A7034	Nasal interface (mask or cannula type) used with positive airway pressure device, with or without head strap
A7035	Headgear used with positive airway pressure device
A7036	Chinstrap used with positive airway pressure device
A7037	Tubing used with positive airway pressure device
A7038	Filter, disposable, used with positive airway pressure device
A7039	Filter, nondisposable, used with positive airway pressure device
A7044	Oral interface used with positive airway pressure device, each
A7045	Exhalation port with or without swivel used with accessories for positive airway devices, replacement only
A7046	Water chamber for humidifier, used with positive airway pressure device, replacement, each
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
E0561	Humidifier, non-heated, used with positive airway pressure device
E0562	Humidifier, heated, used with positive airway pressure device
E0601	Continuous airway pressure (CPAP) device
S8262	Mandibular orthopedic repositioning device, each

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

CPT®* Codes	**Description**
30801	Ablation, soft tissue of inferior turbinates, unilateral or bilateral, any method (eg, electrocautery, radiofrequency ablation, or tissue volume reduction); superficial)
30802	Ablation, soft tissue of inferior turbinates, unilateral or bilateral, any method (eg, electrocautery, radiofrequency ablation, or tissue volume reduction); intramural (ie, submucosal)
41512	Tongue base suspension, permanent suture technique
41530	Submucosal ablation of the tongue base, radiofrequency, one or more sites, per session
42140†	Uvulectomy, excision of the uvula
42950	Pharyngoplasty (plastic or reconstructive operation on pharynx)

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

HCPCS Codes	**Description**
S2080	Laser-assisted uvulopalatoplasty (LAUP)

Experimental/Investigational/Unproven/Not Covered for the treatment of obstructive sleep apnea when used to report any non covered service/procedure outlined in this Coverage Policy:

CPT®* Codes	**Description**
42299	Unlisted procedure, palate, uvula
42999	Unlisted procedure, pharynx, adenoids, or tonsils

HCPCS Codes	**Description**
E1399	Durable medical equipment, miscellaneous

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