Subject: Expiratory Positive Airway Pressure (EPAP) for Obstructive Sleep Apnea		Original Effective Date: 10/30/13
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DISCLAIMER

This Molina Clinical Policy (MCP) is intended to facilitate the Utilization Management process. It expresses Molina's determination as to whether certain services or supplies are medically necessary, experimental, investigational, or cosmetic for purposes of determining appropriateness of payment. The conclusion that a particular service or supply is medically necessary does not constitute a representation or warranty that this service or supply is covered (i.e., will be paid for by Molina) for a particular member. The member's benefit plan determines coverage. Each benefit plan defines which services are covered, which are excluded, and which are subject to dollar caps or other limits. Members and their providers will need to consult the member's benefit plan to determine if there are any exclusion(s) or other benefit limitations applicable to this service or supply. If there is a discrepancy between this policy and a member's plan of benefits, the benefits plan will govern. In addition, coverage may be mandated by applicable legal requirements of a State, the Federal government or CMS for Medicare and Medicaid members. CMS's Coverage Database can be found on the CMS website. The coverage directive(s) and criteria from an existing National Coverage Determination (NCD) or Local Coverage Determination (LCD) will supersede the contents of this Molina Clinical Policy (MCP) document and provide the directive for all Medicare members. [1]

DESCRIPTION OF PROCEDURE/SERVICE/PHARMACEUTICAL

Obstructive Sleep Apnea

Obstructive sleep apnea (OSA) is a breathing disorder that is defined by either a decrease or complete cessation of airflow during sleep. Airflow obstruction arises when the muscles in the back of the throat fail to keep the airway open. OSA is characterized by repetitive pauses in breathing during sleep, despite the effort to breathe, and is usually associated with a reduction in blood oxygen saturation and is often portrayed by loud snoring, gasping, or choking, and by hypopnea or apnea during sleep. These pauses in breathing, called apneas, typically last 20 to 40 seconds. Hypopnea involves episodes of overly shallow breathing or an abnormally low respiratory rate. Hypopnea differs from apnea in that there remains some flow of air. Untreated OSA is associated with symptoms of sleep deprivation and excessive sleepiness, cognitive dysfunction, diminished quality of life and productivity, sexual dysfunction, mood changes, increased accident risk, and cardiovascular disease and stroke.

The results of polysomnogram (PSG) testing are reported in terms of the apnea-hypopnea index (AHI), or respiratory disturbance index (RDI). The AHI is determined by adding the total number of apneas and hypopneas during the sleep time and dividing that number by the total hours of sleep. RDI has been used synonymously with AHI, in addition to the number of apnea and hypopnea episodes, the RDI also includes the number of respiratory effort-related arousals (RERA).The severity of OSA is based on PSG results; an

AHI/RDI greater than or equal to 5 and less than 15 is mild, an AHI/RDI greater than or equal to 15 and less than or equal to 30 is moderate, and an AHI/RDI greater than 30 is severe. [27]

Treatment of OSA includes behavioral therapy (e.g., weight loss), drug therapy, continuous positive airway pressure (CPAP), oral appliances, palatal implants, and surgery. CPAP is the first-line treatment for patients with moderate to severe OSA, with a treatment success rate of nearly 100% when used properly. CPAP provides a constant flow of air delivered through a face mask worn while sleeping to keep the upper airway open; however, patients frequently complain of the intrusive nature of the device, resulting in lack of acceptance or partial adherence. [20-22]

Expiratory positive airway pressure (EPAP)

Expiratory positive airway pressure (EPAP) involves the use of an air-valve-type of device, which is placed over each nostril. Air is easily allowed through the valves when one breathes in, but when one breathes out, small exit holes in the device create a back pressure, called a positive airway pressure that pushes backward through one's airway to keep it open. Since this positive airway pressure is created by your own expiration of air, it is called expiratory positive airway pressure. There is currently one device called the Provent (Ventus Medical Inc.) that is used for EPAP. The device is equipped with small bidirectional valves worn just inside each nostril and secured to the outside of the nose with adhesive. The Provent device is intended for treatment of mild, moderate, and severe obstructive sleep apnea. The device is typically prescribed by a sleep medicine specialist and is used by the patient at home. [20-22]

Provent Sleep Apnea Therapy (Ventus Medical Inc.) is classified by the Center for Devices and Radiological Health (CDRH) as an intranasal expiratory resistance valve for OSA and regulated as a Class II device, classified under the Product Code OHP. [2]

INITIAL COVERAGE CRITERIA

Expiratory positive airway pressure (EPAP) devices that include nasal dilators (Provent) are considered experimental and investigational due to insufficient clinical evidence supporting the safety and efficacy for treating obstructive sleep apnea.

COVERAGE EXCLUSIONS

Expiratory positive airway pressure (EPAP) devices that include nasal dilators (Provent) are considered experimental and investigational due to insufficient clinical evidence supporting the safety and efficacy for treating obstructive sleep apnea.

SUMMARY OF MEDICAL EVIDENCE [3-19]

Overall the quality of the available evidence is low. Results from available studies indicate that therapeutic response is variable among the participants and sample sizes are small. Further research from larger, well-designed studies is needed to evaluate the effectiveness of the device compared with established treatments for OSA, to determine its long-term effectiveness and to determine which patients would benefit from this therapy.

Kureshi and colleagues (2014) performed a small randomized, double-blind, placebo-controlled, crossover pilot study was performed. CPAP candidates, 8-16 years old, underwent NEPAP and placebo polysomnograms. Subjects with ≥ 50% reduction in the apnea hypopnea index (AHI) from placebo to NEPAP night or AHI < 5/h on NEPAP night wore NEPAP at home for 30 days. Adherence was assessed by daily phone calls/emails and collecting used devices. Fourteen subjects (age 13.4 ± 1.9 years, BMI z-scores 2.2 ± 1 [mean ± SD]) were studied. There was significant improvement in the obstructive apnea index with NEPAP vs. placebo: 0.6 (0-21.1)/h vs. 4.2 (0-41.9)/h (median [range], p = 0.010) and trends for improvement in other polysomnographic parameters. However, responses were variable, with 3 subjects not improving and 2 worsening. Older children and those with less hypercapnia had a better response. Eight subjects were sent home with devices; one was lost to follow-up, and adherence in the remainder was 83% of nights; these subjects had a significant improvement in sleepiness and quality of life. In conclusion the authors suggested that NEPAP devices are a potential alternative therapy for OSAS in a small subset of children. Due to variability in individual responses, efficacy of NEPAP should be evaluated with polysomnography.[18]

Rossi and associates (2013) evaluated the efficacy of the Provent nasal device for preventing the recurrence of OSA following CPAP withdrawal in patients with moderate-to-severe OSA with well-established improvement while on CPAP. The goal of the study was to determine if OSA patients could occasionally substitute the Provent for their CPAP. This study, conducted in outpatient sleep clinics in the United Kingdom and Switzerland, randomized 67 patients with OSA receiving CPAP to 1 of 3 groups for 2 weeks: Continuing CPAP (n=23; mean age 64.4 years), Active Provent (n=22; mean age 63.2 years), or Placebo Provent (n=22; mean age 59.7 years). The three groups were similar at baseline and their mean AHI before CPAP treatment was 38 events per hour. Primary outcomes included for the Active Provent versus Placebo Provent groups were OSA severity (ODI), AHI, and ESS score. Secondary outcomes for the Active Provent versus Placebo Provent groups included ODI from ambulatory pulse oximetry and BP (blood pressure). For the CPAP versus Active Provent, or CPAP versus Placebo Provent groups, secondary outcomes included ODI/AHI, ESS, and BP. Compliance was assessed by patient diaries. CPAP usage data was downloaded from the machines. One patient in the Active Provent group withdrew from the study due to symptom recurrence while 3 patients (1 in the Active Provent group and 2 in the Placebo Provent group) were excluded from the study due to insufficient compliance leaving 63 patients in this per protocol analysis. OSA recurred in the Provent (ODI 35.8, SD 17.4) and placebo Provent (ODI 28.2, SD 18.3) groups, and there was no significant difference in ODI, AHI and ESS between Provent and placebo Provent at 2 weeks (mean difference ODI -1.0, 95% CI -10.0 to +12.0, p=0.85; AHI +3.2, 95% CI -7.7 to +14.1, p=0.52; and ESS -1.4, 95% CI -4.1 to +1.4, p=0.33). ODI from ambulatory pulse-oximetry and BP at 2 weeks were not different in the Provent versus placebo Provent groups. ODI, AHI and BP, but not ESS, were significantly higher in the Provent and placebo Provent groups compared with CPAP. The authors concluded that Provent cannot be recommended as an alternative short-term therapy for patients with moderate to severe OSA already on CPAP. [14]

Berry and colleagues (2011) performed a prospective, multicenter, sham-controlled, parallel-group, randomized, double-blind clinical trial to investigate the efficacy of nasal expiratory positive airway pressure (EPAP) device as a treatment for obstructive sleep apnea (OSA). Individuals with Obstructive sleep apnea with

a pre-study AHI≥10/hour were included. Treatment with a nasal EPAP device (N=127) or similar appearing sham device (N=123) for 3 months was completed. Polysomnography (PSG) was performed on 2 non-consecutive nights (random order: device-on, device-off) at week 1 and after 3 months of treatment. Analysis of an intention At week 1, the median AHI value (device-on versus device-off) was significantly lower with EPAP (5.0 versus 13.8 events/h, P<0.0001) but not sham (11.6 versus 11.1 events/h, P=NS); the decrease in the AHI (median) was greater (-52.7% vs. -7.3%, P<0.0001) for the ITT group. At month 3, the percentage decrease in the AHI was 42.7% (EPAP) and 10.1% (sham), P<0.0001. Over 3 months of EPAP treatment the Epworth Sleepiness Scale decreased (9.9±4.7 to 7.2±4.2, P<0.0001), and the median percentage of reported nights used (entire night) was 88.2%. to treat group (ITT) (patients completing week 1 PSGs) (EPAP N=119, sham N=110) was performed. The authors concluded that the nasal EPAP device significantly reduced the AHI and improved subjective daytime sleepiness compared to the sham treatment in patients with mild to severe OSA with excellent adherence. [7]

Another prospective, multicenter, single-arm, open-label extension to a 3-month EPAP vs sham randomized clinical trial was conducted by Kryger et al (2011). The goal was to evaluate the long-term durability of treatment response and safety of a nasal expiratory positive airway pressure (EPAP) device used to treat obstructive sleep apnea (OSA). The trial included OSA patients in the EPAP arm of the EPAP vs sham randomized study who used the EPAP device≥4 h per night,≥5 nights per week on average during months 1 and 2 of the 3-month trial and had≥50% reduction in AHI or AHI reduction to<10 documented by polysomnography, comparing the 3-month device-on PSG to the week-one device-off PSG. Treatment with a nasal EPAP device (N = 41) for 12 months was performed. Polysomnography (PSG) on the patients wearing the device was performed after 12 months of treatment. The month 12 device-on PSG data from the analyzable subject cohort (N = 34) was compared to the week 1 device-off PSG from the EPAP vs sham trial. Of the 51 patients eligible, 34 were still using the EPAP device at the end of 12 months. Median AHI was reduced from 15.7 to 4.7 events/h (week 1 device-off versus month 12 device-on). The decrease in the AHI (median) was 71.3% (p<0.001). The median proportion of sleep time with snoring was reduced by 74.4% (p<0.001). Over 12 months of EPAP treatment, the Epworth Sleepiness Scale decreased (11.1±4.2 to 6.0±3.2, p<0.001), and the median percentage of reported nights used (entire night) was 89.3%. The authors concluded that nasal EPAP significantly reduced the AHI, improved subjective daytime sleepiness and reduced snoring after 12 months of treatment. Long-term adherence to EPAP was excellent in those who had a positive clinical response at month 3 of the EPAP vs sham study. [8]

Walsh et al. (2011) evaluated tolerability, short-term efficacy and adherence of an expiratory positive airway pressure (EPAP) nasal device in 59 OSA patients who refused CPAP or used CPAP less than 3 hours per night. After demonstrating tolerability to the EPAP device during approximately 1 week of home use, 47 patients (80%) underwent a baseline polysomnogram (PSG1). Forty-three patients met AHI entry criteria and underwent PSG2 within 10 days of PSG1. Twenty four patients (56%) met pre-specified efficacy criteria and underwent PSG3 after 5 weeks of EPAP treatment. Compared to PSG1, mean AHI was significantly lower at both PSG2 and PSG3. For most patients AHI at PSG3 was similar to AHI at PSG2. Device use was reported an average of 92% of all sleep hours. The authors concluded that improvements in AHI and Epworth Sleepiness Scale (ESS) scores, combined with the high degree of treatment adherence observed, suggest that the EPAP device tested may become a useful therapeutic option for OSA. Limitations of the study include lack of randomization and

control, small sample size and short term follow-up. A potential for bias exists due to manufacturer sponsorship of the study. [11]

At the New York University Sleep Disorders Center, Patel et al. (2011) studied a one way nasal device using expiratory positive airway pressure (EPAP) to identify appropriate patients for the therapy and provide pilot data as to its potential mechanisms of action. Twenty patients with OSA underwent three nocturnal polysomnograms (NPSG) including diagnostic, therapeutic (with a Provent® nasal valve device) and CPAP. Nineteen of the 20 patients tolerated the device. The authors reported that the nasal valve device produced improvement in sleep disordered breathing in 75% of patients with OSA of varying severity, with 50% of patients reaching a clinically significant reduction in RDI. Although the study was not able to establish predictors of success or a definitive mechanism of action, the authors thought that it helps define a restricted list of candidates for further investigation. A potential for bias exists due to manufacturer sponsorship of the study. [13]

Rosenthal and colleagues (2009) performed another multicenter, prospective study of nasal EPAP device in the treatment of obstructive sleep apnea: Efficacy and 30-day adherence. Study objectives were to evaluate the efficacy of a novel device placed in the nares that imposes an expiratory resistance for the treatment of obstructive sleep apnea (OSA) and evaluate adherence to the device over a 30-day in-home trial period. Design: One diagnostic and 3 treatment polysomnograms were administered in a Latin-square design to identify the optimal expiratory resistance to be used during the 30-day in-home trial. Subjects had repeat polysomnography with the prescribed device at the end of the 30-day trial. Setting: Multicenter study. Participants included (N = 34; age 27 to 67) with a baseline apnea-hypopnea index (AHI) >= 5. The AHI was reduced from 24.5 +/- 23.6 (mean +/- SD) to an average of 13.5 +/- 18.7 ($p < 0.001$) across initial treatment nights. The AHI was 15.5 +/- 18.9 ($p = 0.001$) for the prescribed device at the end of the 30-day trial. Of 24 subjects with an AHI > 10 at baseline, 13 achieved an AHI <= 10 on the initial treatment nights; 10 had a similar response on the final treatment night. Percent of the night snoring decreased from 27.5 +/- 23.2 to 11.6 +/- 13.7 ($p < 0.001$) on initial treatment nights and 14.6 +/- 20.6 ($p = 0.013$) at the end of the trial; Epworth Sleepiness scores decreased from 8.7 +/- 4.0 at baseline to 6.9 +/- 4.4 ($p < 0.001$) at the end of the trial; the Pittsburgh Sleep Quality Index improved from 7.4 +/- 3.3 to 6.5 +/- 3.6 ($p = 0.042$). Mean oxygen saturation increased from 94.8 +/- 2.0 to 95.2 +/- 1.9 ($p = 0.023$) on initial treatment nights and 95.3 +/- 1.9 ($p = 0.003$) at the end of the trial. Sleep architecture was not affected. Participants reported using the device all night long for 94% of nights during the in-home trial. The authors concluded that treatment with this novel device was well tolerated and accepted by the participants. An overall reduction in AHI was documented; however, therapeutic response was variable among the participants. Further research is required to identify the ideal candidates for this new therapeutic option in the management of OSA. [9]

The first study using the nasal Provent device for the treatment of OSA was conducted at the Stanford Research Institute International by Colrain et al. (2008) to test the hypothesis that the application of expiratory resistance via a nasal valve device would improve breathing during sleep in subjects with OSA and in primary snorers. Thirty men and women were recruited for the study. Twenty-four had at least mild OSA (AHI >5), and 6 were

primary snorers. Subjects underwent 2 nights of polysomnographic evaluation, one with and one without a new nasal resistance device with the order of nights counterbalanced across participants. The device consisted of a small valve inserted into each nostril calibrated to provide negligible inspiratory resistance, but increased expiratory resistance. Standard polysomnography was conducted to compare participants' sleep both with and without the device, with the scoring conducted blind to treatment condition. The apnea-hypopnea (AHI) and oxygen desaturation (O2DI) indices both significantly decreased and the percentage of the night spent above 90% saturation significantly increased with device use. The results of this pilot study are suggestive of a therapeutic effect of expiratory nasal resistance for some OSA patients and indicate that this technique is worthy of further clinical study. A potential for bias exists due to manufacturer sponsorship of the study. [12]

Systematic reviews/meta-analysis:

Riaz and colleagues (2015) performed a systematic review and meta-analysis to quantify the effectiveness of nasal expiratory positive airway pressure (nasal EPAP) devices or Provent as treatment for obstructive sleep apnea (OSA). Eighteen studies (920 patients) were included. Pre- and post-nasal EPAP means ± standard deviations (M ± SD) for apnea-hypopnea index (AHI) in 345 patients decreased from 27.32 ± 22.24 to 12.78 ± 16.89 events/hr (relative reduction = 53.2%). Random effects modeling mean difference (MD) was -14.78 events/hr [95% CI -19.12, -10.45], p value < 0.00001. Oxygen desaturation index (ODI) in 247 patients decreased from 21.2 ± 19.3 to 12.4 ± 14.1 events/hr (relative reduction = 41.5%, p value < 0.00001). Lowest oxygen saturation (LSAT) M ± SD improved in 146 patients from 83.2 ± 6.8% to 86.2 ± 11.1%, MD 3 oxygen saturation points [95% CI 0.57, 5.63]. Epworth Sleepiness Scale (ESS) M ± SD improved (359 patients) from 9.9 ± 5.3 to 7.4 ± 5.0, MD -2.5 [95% CI -3.2, -1.8], p value < 0.0001. Nasal EPAP (Provent) reduced AHI by 53.2%, ODI by 41.5% and improved LSAT by 3 oxygen saturation points. Generally, there were no clear characteristics (demographic factors, medical history, and/or physical exam finding) that predicted favorable response to these devices. However, limited evidence suggests that high nasal resistance could be associated with treatment failure. Additional studies are needed to identify demographic and polysomnographic characteristics that would predict therapeutic success with nasal EPAP (Provent). [16]

Professional Organizations [20]

The American Academy of Sleep Medicine (AASM) 2009 guidelines recommends offering positive airway pressure therapy to all patients who have OSA on the basis of expert consensus. They define OSA as either an obstructive respiratory disturbance index (RDI) greater than 15 events per hour, or an obstructive RDI between 5 and 14 events per hour that is accompanied by daytime sleepiness, loud snoring, witnessed breathing interruptions, or awakenings due to gasping or choking. The obstructive RDI is the number of obstructive apneas, obstructive hypopneas, and respiratory effort related arousals per hour of sleep. The report summarizes the following:

- BPAP, pressure relief, or APAP can be considered in the management of OSA in CPAP-intolerant patients (Consensus).
- BPAP is an optional therapy in some cases where high pressure is needed and the patient experiences difficulty exhaling against a fixed pressure or coexisting central hypoventilation is present.

- Alternative therapies (eg, an oral appliance or upper airway surgery) may be offered to patients who decline positive airway pressure therapy and who have mild to moderate OSA, amenable upper airway anatomy, and a preference for such treatment.

American Academy of Pediatrics (AAP) 2012 guidelines for the diagnosis and management of childhood obstructive sleep apnea syndrome indicate that if a child is determined to have OSAS, has a clinical examination consistent with adenotonsillar hypertrophy, and does not have a contraindication to surgery, the clinician should recommend adenotonsillectomy as the first line of treatment. If the child has OSAS but does not have adenotonsillar hypertrophy, other treatment should be considered. Clinicians should refer patients for CPAP management if symptoms/signs or objective evidence of OSAS persists after adenotonsillectomy or if adenotonsillectomy is not performed. [6]

CODING INFORMATION: THE CODES LISTED IN THIS POLICY ARE FOR REFERENCE PURPOSES ONLY. LISTING OF A SERVICE OR DEVICE CODE IN THIS POLICY DOES NOT IMPLY THAT THE SERVICE DESCRIBED BY THIS CODE IS COVERED OR NON-COVERED. COVERAGE IS DETERMINED BY THE BENEFIT DOCUMENT. THIS LIST OF CODES MAY NOT BE ALL INCLUSIVE.

CPT	Description
94799	Unlisted pulmonary service or procedure (when used for Expiratory Positive Airway Pressure)

HCPCS	Description
E1399	Durable medical equipment, miscellaneous (when used for Expiratory Positive Airway Pressure device)

ICD-10	Description: [For dates of service on or after 10/01/2015]
G47.31	Primary central sleep apnea
G47.33	Obstructive sleep apnea (adult) (pediatric)
G47.37	Central sleep apnea in conditions classified elsewhere

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26. Advanced Medical Review (AMR):
    - Policy reviewed by MD Board certified in Internal Medicine, Pulmonary Disease, Critical Care, Sleep Medicine. 8/29/13
    - Policy reviewed by practicing MD Board certified in Sleep Medicine. 10//25/19

Review/Revision History:
12/16/15: Policy reviewed, no changes.
11/8/16: Policy was reviewed and updated and no changes were made to the criteria. The medical evidence summary and reference sections were updated.
9/19/17: Policy reviewed, no changes.
7/10/18: Policy reviewed, no changes.
12/10/19: Policy reviewed, no changes. No new evidence based studies or guidelines found, nasal EPAP remains experimental, investigational and unproven for the treatment of OSA.