Molina Clinical Policy
Expiratory Positive Airway Pressure (EPAP) for Obstructive Sleep Apnea: Policy No. 145

Last Approval: 12/14/2022
Next Review Due By: December 2023

DISCLAIMER

This Molina Clinical Policy (MCP) is intended to facilitate the Utilization Management process. Policies are not a supplementation or recommendation for treatment; Providers are solely responsible for the diagnosis, treatment and clinical recommendations for the Member. 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 (e.g., 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 MCP and provide the directive for all Medicare members. References included were accurate at the time of policy approval and publication.

OVERVIEW

Obstructive Sleep Apnea (OSA) is a breathing disorder that is defined by 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. (Kryger et al., 2021; Paruthi, 2021; Badr, 2021).

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. (Kryger et al., 2021; Paruthi, 2021; Badr, 2021).

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; patients frequently complain of the intrusive nature of the device, resulting in lack of acceptance or partial adherence. (Patil, et al., 2019).

Expiratory Positive Airway Pressure (EPAP) uses an air-valve-type of device that is placed over each nostril. Small exit holes in the device provide a positive airway pressure (PAP), also known as a back pressure, that pushes backward into the patient's airway to maintain it open as the patient exhales. EPAP refers to PAP caused by the patient's own expiration of air. EPAP is currently provided by one device, Provent (Ventus Medical Inc.). 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 designed to treat mild, moderate, and severe OSA. The device is typically prescribed by a sleep medicine specialist and is used at home by the patient. (Patil, et al., 2019).

Regulatory Status

The Food and Drug Administration (2010) Center for Devices and Radiological Health (CDRH) classified the Provent Sleep Apnea Therapy (Ventus Medical Inc.) as an intranasal expiratory resistance valve for OSA and regulated as a Class II device, classified under the Product Code OHP.

COVERAGE POLICY

EPAP devices (including but not limited to nasal dilators [Provent]) **are considered experimental and investigational** due to insufficient clinical evidence supporting the safety and efficacy for treating OSA.

SUMMARY OF MEDICAL EVIDENCE

Results from early studies indicate that therapeutic response was variable among participants and small sample sizes. 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. More recently, Liu et al. (2019) published a study on the efficacy and safety of EPAP – there were no considerable differences between the use of EPAP over CPAP. Below is a summary of studies and trials published between 2009 and 2014.

A small randomized, double-blind, placebo-controlled, crossover pilot study was performed by Kureshi et al. (2014). Candidates ages 8-16 underwent nasal expiratory positive airway (NEPAP) and placebo polysomnograms. In conclusion, NEPAP devices are a potential alternative therapy for obstructive sleep apnea syndrome (OSAS) in a small subset of children. Due to variability in individual responses, efficacy of NEPAP should be evaluated with PSG.

Rossi et al. (2013) evaluated the efficacy of the Provent nasal device for preventing the recurrence of OSA following CPAP withdrawal among 67 patients with OSA who were receiving CPAP. The goal of the study was to determine if OSA patients could occasionally substitute the Provent device for CPAP. For the Active Provent vs. Placebo Provent groups, primary outcomes included OSA severity, oxygen desaturation index (ODI), AHI, and Epworth Sleepiness Scale (ESS) score. Secondary outcomes for the Active Provent vs. Placebo Provent groups included: ODI from ambulatory pulse oximetry and blood pressure. For the CPAP vs. Active Provent groups, or CPAP vs. Placebo Provent groups, secondary outcomes included: ODI, AHI, ESS, and blood pressure.

Rossi et al. also assessed compliance by patient diaries – CPAP usage data was downloaded from the devices. OSA recurred in the Provent (ODI 35.8, SD 17.4) and placebo Provent (ODI 28.2, SD 18.3) groups; there was no significant difference in ODI, AHI and ESS between the Provent and Placebo Provent groups at two weeks. ODI from ambulatory pulse-oximetry and blood pressure at two weeks were not different in the Provent vs. Placebo Provent groups. ODI, AHI and blood pressure (but not ESS) were significantly higher in the Provent and Placebo Provent groups compared with the CPAP group. In conclusion, Provent cannot be recommended as an alternative short-term therapy for patients with moderate to severe OSA already using CPAP.

Berry et al. (2011) performed a prospective, multicenter, sham-controlled, parallel-group, randomized, double-blind clinical trial to investigate the efficacy of nasal EPAP device as a treatment for OSA. The trial included individuals with OSA and 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 was performed on 2 non-consecutive nights (random order: device-on, device-off) at week one and after three months of treatment. Analysis of an intention at week one found the median AHI value (device-on versus device-off) was significantly lower with EPAP. The decrease in the AHI (median) was greater for the ITT group. At month three, the percentage decrease in the AHI was 42.7% (EPAP) and 10.1% (sham), P<0.0001. Over three months of EPAP treatment, the ESS decreased, and the median percentage of reported nights used (entire night) was 88.2%. 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.

Kryger et al. (2011) conducted a prospective, multicenter, single-arm, open-label extension to a three-month EPAP vs sham randomized clinical trial. The goal was to evaluate the long-term durability of treatment response and safety of a nasal EPAP device used to treat OSA. The trial included OSA patients in the EPAP arm of the EPAP vs. sham randomized study who used the EPAP device inclusion criteria was defined as use of a EPAP device ≥ four hours per night, ≥ 5 nights per week on average during months one- and two- of the three-month trial. and had ≥ 50% reduction in AHI or AHI reduction to <10 documented by PSG, comparing the three-month device-on PSG to the week one device-off PSG. Treatment with a nasal EPAP device (N = 41) for 12 months was performed. 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%. The median proportion of sleep time with snoring was reduced by 74%. Over 12 months of EPAP treatment, the ESS decreased, and the median percentage of reported nights used (entire night) was 89%. In conclusion, 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 three of the EPAP vs. sham study.

Walsh et al. (2011) evaluated tolerability, short-term efficacy, and adherence of an 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 one week of home use, 47 patients (80%) underwent a screening/baseline polysomnogram (PSG1). Forty-three patients met AHI entry criteria and underwent a treatment polysomnogram (PSG2) within 10 days of PSG1. Twenty-four patients (56%) met pre-specified efficacy criteria and underwent PSG3 which was performed 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. Improvements in AHI and ESS scores were noted combined with the high degree of treatment adherence observed – this suggests that the EPAP device tested may 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.

Patel et al. (2011) studied a one-way nasal device at the New York University Sleep Disorders Center, using EPAP to identify appropriate patients for treatment. Pilot data provided 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. Nasal valve device produced improvement in sleep disordered breathing in 75% of patients with OSA of varying severity; 50% of patients reached a clinically significant reduction in RDI. While the study was not able to establish predictors of success or a definitive mechanism of action, it helps define a restricted list of candidates for further investigation. A potential for bias exists due to manufacturer sponsorship of the study.

Rosenthal et al. (2009) performed a multicenter, prospective study of nasal EPAP device in the treatment of OSA. Study objectives were to evaluate the efficacy of a novel device placed in the nares that imposes an expiratory resistance for the treatment of OSA and evaluate adherence to the device over a 30-day in-home trial period. Participants reported using the device all night long for 94% of nights during the in-home trial. The authors concluded that treatment was well tolerated and accepted by participants. An overall reduction in AHI was documented however, therapeutic response was variable. Further research is required to identify ideal candidates for this therapeutic option.

The first study using the Provent device for the treatment of OSA was conducted at the Stanford Research Institute International. Colrain et al. (2008) examined 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; 24 had at least mild OSA (AHI >5) and six were primary snorers. Participants underwent two nights of polysomnographic evaluation, one with and one without a new nasal resistance device (with the order of nights counterbalanced across participants). Standard PSG was conducted to compare participants sleep ~~both~~ with and without the device, with the scoring conducted blind to treatment condition. Measurement of AHI and oxygen desaturation (O2DI) indices both significantly decreased – the percentage of the night spent above 90% saturation significantly increased with device use. 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.

Systematic Reviews

Riaz et al. (2015) performed a systematic review and meta-analysis to quantify the effectiveness of nasal EPAP devices or Provent as treatment for OSA. Eighteen studies (920 patients) were included. Pre- and post-nasal EPAP means ± standard deviations (M ± SD) for AHI in 345 patients decreased from 27.32 ± 22.24 to 12.78 ± 16.89 events/hr (relative reduction = 53.2%). Nasal EPAP (Provent) reduced AHI by 53%, ODI by 41% and improved LSAT by three oxygen saturation points. There were no clear characteristics (e.g., demographic factors, medical history, physical exam finding) that predicted favorable response to these devices. 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 Provent.

National and Specialty Organizations

The **American Academy of Sleep Medicine (AASM)** guidelines (2019) issued two practice statements for appropriate and effective management of patients with OSA treated with PAP:

1. OSA treatment with PAP therapy should be based on a diagnosis of OSA which is confirmed by objective sleep apnea testing; and
2. Adequate follow-up should include monitoring objective efficacy and device data to confirm treatment and adherence; this should happen after initiation of PAP therapy and during OSA treatment.

AASM also provided the following recommendations:

1. PAP should be used, compared to no therapy, for treatment of OSA in adults with excessive sleepiness, impaired sleep-related quality of life, or comorbid hypertension.
2. PAP therapy can begin using automatic PAP (APAP) at home or in-laboratory PAP titration in adults with OSA and no significant comorbidities.
3. CPAP or APAP is recommended for ongoing treatment of OSA in adults.
4. CPAP or APAP over bilevel PAP (BPAP) is recommended as the routine treatment of OSA in adults.
5. Educational interventions should be given at the start of PAP therapy in adults with OSA.
6. Behavioral interventions should be given during the onset of PAP therapy in adults with OSA.
7. Telemonitoring-guided interventions are recommended during the onset of PAP therapy in adults with OSA.

The **American Academy of Pediatrics (AAP)** guidelines published in 2012 for the diagnosis and management of childhood OSAS. The guidelines 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 or objective evidence of OSAS persists after adenotonsillectomy or if adenotonsillectomy is not performed.

The **American College of Physicians (ACP)** published the clinical practice guideline *Management of Obstructive Sleep Apnea in Adults* with the following recommendations:

1. Patients who are overweight and obese with a diagnosis of OSA should be encouraged to lose weight.
2. CPAP treatment is an initial therapy for patients with OSA.
3. Mandibular advancement devices are considered an alternative therapy to CPAP treatment for patients with OSA with a preference to these types of devices. The devices may also be considered for patients with adverse effects due to CPAP treatment.

SUPPLEMENTAL INFORMATION

None.

CODING & BILLING INFORMATION

CPT Code

CPT	Description
94799	Unlisted pulmonary service or procedure (when used for EPAP)

HCPCS Code

HCPCS	Description
E1399	Durable medical equipment, miscellaneous (EPAP)

CODING DISCLAIMER. Codes listed in this policy are for reference purposes only and may not be all-inclusive. Deleted codes and codes which are not effective at the time the service is rendered may not be eligible for reimbursement. Listing of a service or device code in this policy does not guarantee coverage. Coverage is determined by the benefit document. Molina adheres to Current Procedural Terminology (CPT®), a registered trademark of the American Medical Association (AMA). All CPT codes and descriptions are copyrighted by the AMA; this information is included for informational purposes only. Providers and facilities are expected to utilize industry standard coding practices for all submissions. When improper billing and coding is not followed, Molina has the right to reject/deny the claim and recover claim payment(s). Due to changing industry practices, Molina reserves the right to revise this policy as needed.

APPROVAL HISTORY

12/14/2022	Policy reviewed, no changes to the criteria, references updated.
12/8/2021	Policy reviewed, no changes to coverage criteria. Summary of Medical Evidence section condensed; updated AASM and AAP guidelines. References updated.
12/9/2020	Policy reviewed, no changes to the criteria.
12/10/2019	Policy reviewed, no changes to the criteria. No new evidence-based studies or guidelines found. IRO Peer Review. Policy reviewed on October 25, 2019 by a practicing, board-certified physician in the area of Sleep Medicine.
7/10/2018	Policy reviewed, no changes to the criteria.
9/19/2017	Policy reviewed, no changes to the criteria.
11/8/2016	Policy reviewed, no changes to the criteria. Summary of Medical Evidence and Reference sections updated.
12/16/2015	Policy reviewed, no changes to the criteria.

REFERENCES

Government Agencies

1. Centers for Medicare and Medicaid Services (CMS). Medicare coverage database (search "continuous positive airway pressure therapy for obstructive sleep apnea 240.4"). Available from CMS. Effective March 13, 2008. Accessed November 2022.
2. Food and Drug Administration (FDA). Provent professional sleep apnea therapy (Provent 80) and Provent professional sleep apnea therapy (Provent 50): Device classification. Available from FDA. Published 2010. Accessed November 2022.

Evidence Based Reviews and Publications

1. Badr MS. Central sleep apnea: Treatment. Available from UpToDate. Updated September 29, 2021. Accessed November 2022. Registration and login required.
2. Kryger MH, Malhorta A. Management of obstructive sleep apnea in adults. Available from UpToDate. Updated August 11, 2022. Accessed November 2022. Registration and login required.
3. Paruthi S. Evaluation of suspected obstructive sleep apnea in children. Available from UpToDate. Updated April 15, 2022. Accessed November 2022. Registration and login required.
4. Paruthi S. Management of suspected obstructive sleep apnea in children. Available from UpToDate. Updated January 14, 2021. Accessed November 5, 2022. Registration and login required.

National and Specialty Organizations

1. Patil S, Ayappa I, Caples SM, Kimoff RJ, Patel SR, Harrod CG. Treatment of adult obstructive sleep apnea with positive airway pressure: An American Academy of Sleep Medicine clinical practice guideline. J Clin Sleep Med. 2019 Feb 15;15(2):335-343. doi: 10.5664/jcsm.7640.

Peer Reviewed Publications

1. Berry RB, Kryger MH, Massie CA. A novel nasal expiratory positive airway pressure (EPAP) device for the treatment of obstructive sleep apnea: A randomized controlled trial. Sleep. 2011 Apr 1;34(4):479-85. doi: 10.1093/sleep/34.4.479.
2. Colrain IM, Brooks S, Black J. A pilot evaluation of a nasal expiratory resistance device for the treatment of obstructive sleep apnea. J Clin Sleep Med. 2008 Oct 15;4(5):426-33. PMID: 18853699; PMCID: PMC2576328.
3. Kryger MH, Berry RB, Massie CA. Long-term use of a nasal expiratory positive airway pressure (EPAP) device as a treatment for obstructive sleep apnea (OSA). J Clin Sleep Med. 2011 Oct 15;7(5):449-53B. doi: 10.5664/JCSM.1304.
4. Kureshi SA, Gallagher PR, McDonough JM, Cornaglia MA, Maggs J, Samuel J, et al. Pilot study of nasal expiratory positive airway pressure devices for the treatment of childhood obstructive sleep apnea syndrome. J Clin Sleep Med. 2014 Jun 15;10(6):663-9. doi: 10.5664/jcsm.3796.
5. Liu Y, Ying Y, Pandu JS, Wang Y, Dou S, Li Y, Ma D. Efficacy and safety assessment of expiratory positive airway pressure (EPAP) mask for OSAHS therapy. Auris Nasus Larynx. 2019 Apr;46(2):238-245. doi: 10.1016/j.anl.2018.08.013.
6. Patel AV, Hwang D, Masdeu MJ, Chen GM, Rapoport DM, Ayappa I. Predictors of response to a nasal expiratory resistor device and its potential mechanisms of action for treatment of obstructive sleep apnea. J Clin Sleep Med. 2011;7(1):13-22.
7. Patil SP, Ayappa IA, Caples SM, Kimoff RJ, Patel SR, Harrod CG. Treatment of adult obstructive sleep apnea with positive airway pressure: An American Academy of Sleep Medicine systematic review, meta-analysis, and GRADE assessment. J Clin Sleep Med. 2019 Feb 15;15(2):335-343. doi: 10.5664/jcsm.7640.
8. Qaseem A, Holty JEC, Owens DK, Dallas P, Starkey M, Shekelle P, Clinical Guidelines Committee of the American College of Physicians. Management of obstructive sleep apnea in adults: A clinical practice guideline from the American College of Physicians. Ann Intern Med. 2013 Oct 1;159(7):471-83. doi: 10.7326/0003-4819-159-7-201310010-00704.
9. Riaz M, Certal V, Nigam G, Abdullatif J, Zaghi S, Kushida CA, et al. Nasal expiratory positive airway pressure devices (Provent) for OSA: A systematic review and meta-analysis. Sleep Disord. 2015;2015:734798. doi: 10.1155/2015/734798.
10. Rosenthal L, Massie CA, Dolan DC, et al. A multicenter, prospective study of a novel nasal EPAP device in the treatment of obstructive sleep apnea: Efficacy and 30-day adherence. J Clin Sleep Med. 2009 Dec 15;5(6):532-7. PMID: 20465019; PMCID: PMC2792968.
11. Rossi VA, Winter B, Rahman NM, et al. The effects of Provent on moderate to severe obstructive sleep apnea during continuous positive airway pressure therapy withdrawal: A randomised controlled trial. Thorax. 2013 Sep;68(9):854-9. doi: 10.1136/thoraxjnl-2013-203508.
12. Walsh JK, Griffin KS, Forst EH, et al. A convenient expiratory positive airway pressure nasal device for the treatment of sleep apnea in patient's non-adherent with continuous positive airway pressure. Sleep Med. 2011 Feb;12(2):147-52. doi: 10.1016/j.sleep.2010.06.011.

APPENDIX

Reserved for State specific information. *Information includes, but is not limited to, State contract language, Medicaid criteria and other mandated criteria.*