Molina Clinical Policy
Implantable Neurostimulator (remedē System) for Central Sleep Apnea: Policy No. 340

Last Approval: 12/13/2023
Next Review Due By: December 2024

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

Central sleep apnea (CSA) refers to a heterogeneous group of sleep-related breathing disorders characterized by diminished or absent central respiratory drive. Central apneas occur through two pathophysiologic patterns: either post-hyperventilation or post-hypoventilation ([1]Badr 2023). CSA may be primary (e.g., idiopathic) or secondary (i.e., associated with an underlying medical cause) ([1-2]Badr 2023). The third edition of the International Classification of Sleep Disorders-Text Revision describes seven categories of sleep disorders grouped under CSA with the following categories affecting adults: primary CSA, CSA with Cheyne-Stokes breathing, CSA due to high altitude periodic breathing, CSA due to a medical disorder without Cheyne-Stokes breathing, CSA due to a medication or substance, and treatment-emergent CSA (previously referred to as “complex sleep apnea”) (Judd & Sateia 2023). The update to the third edition of the International Classification of Sleep Disorders-Text Revision requires that “the diagnosis of treatment-emergent CSA now requires the presence of signs or symptoms, in addition to central apneic events on polysomnography (Judd & Sateia 2023).”

The prevalence of CSA is not high in the general population. However, prevalence tends to be higher among older adults, males, and those with certain comorbid conditions, such as heart failure or stroke ([2]Badr 2023). Diagnosis of CSA generally requires a full-night polysomnographic evaluation, which includes both sleep measures and respiration measures. An overnight, in-laboratory polysomnogram is recommended in “patients with daytime sleepiness plus risk factors for CSA (e.g., heart failure, stroke, use of a long-acting opioid) or more than one symptom or sign of CSA (e.g., daytime sleepiness, insomnia, morning headaches, nocturnal angina, witnessed pauses in breathing during sleep) ([2]Badr 2023).” Polysomnogram also differentiates CSA from the more common obstructive sleep apnea, which can have a very similar presentation. CSA is characterized by the absence of respiratory effort while obstructive sleep apnea is characterized by the presence of respiratory effort. On polysomnogram recording, CSA diagnosis requires the presence of five or more central apneas per hour of sleep and symptoms of insomnia, excessive daytime sleepiness or frequent arousals, and/or hypersomnolence during the day ([2]Badr 2023). A common measure of the severity of CSA is the apnea-hypopnea index (AHI), defined as the mean number of apnea and/or hypopnea episodes that occur during sleep divided by the number of hours of sleep, and is expressed in events per hour. In general, an AHI of < 5 events per hour is categorized as normal, 5 to 14 events per hour as mild, 15 to 30 events per hour as moderate, and > 30 events per hour as severe.

“The goals of therapy in patients with CSA are to normalize sleep-related breathing patterns (i.e., abolish central apneas, decrease or eliminate oxygen desaturations) and thereby improve both the quality of sleep and daytime symptoms and function (Badr 2023).” CSA treatments include positive airway pressure therapies, such as continuous positive airway pressure (CPAP) and bilevel positive airway pressure, adaptive servo ventilation, supplemental oxygen, and medications including acetazolamide, theophylline, and sedative-hypnotic agents. Phrenic nerve stimulation is a potential treatment option for adults with CSA who do not respond to currently available apnea therapies (Badr 2023; Hayes 2023).

Phrenic nerve stimulation is achieved through implantation of the remedē System (Respicardia Inc, Minnetonka, MN). The device causes diaphragmatic contractions similar to normal breathing and can produce improvements in sleep in patients with CSA (Badr 2023). The remedē System was FDA approved on October 6, 2017, for adult patients who

have been diagnosed with moderate-to-severe CSA (FDA 2017). The system is similar to an implantable pacemaker and consists of a battery pack and small, thin wires placed under the skin in the upper chest area that monitor respiratory signals and causes normal breathing to be restored by stimulating the phrenic nerve to communicate with the diaphragm (FDA 2017). The system is programmed using an external System Programmer and Programming Wand. The device monitors the patient's respiratory signals during sleep and stimulates the nerve to move the diaphragm and restore normal breathing. This device is not intended for use in patients with obstructive sleep apnea (Badr 2023).

The remedē System is classified by the Food and Drug Administration (FDA) as an implanted phrenic nerve stimulation device for CSA and is regulated as a Class III device, classified under the Product Code PSR (FDA 2018). A newer model of the remedē System (remedē EL-X System) was approved on July 28, 2021, that is much smaller in size and has approximately 40% improved battery life compared to the previous model (FDA 2021; Hayes 2023). The remedē System received FDA approval for magnetic resonance imaging conditional labeling for 1.5T and 3T scanners on March 28, 2023 (FDA 2023).

COVERAGE POLICY

Implantable neurostimulators for the treatment of central sleep apnea (e.g., remedē System) **are considered experimental and investigational**. There is insufficient evidence in the peer-reviewed medical literature to establish long-term safety, efficacy, and effect on net health outcomes.

DOCUMENTATION REQUIREMENTS. Molina Healthcare reserves the right to require that additional documentation be made available as part of its coverage determination; quality improvement; and fraud; waste and abuse prevention processes. Documentation required may include, but is not limited to, patient records, test results and credentials of the provider ordering or performing a drug or service. Molina Healthcare may deny reimbursement or take additional appropriate action if the documentation provided does not support the initial determination that the drugs or services were medically necessary, not investigational, or experimental, and otherwise within the scope of benefits afforded to the member, and/or the documentation demonstrates a pattern of billing or other practice that is inappropriate or excessive.

SUMMARY OF MEDICAL EVIDENCE

Overall, the quality of evidence evaluating the clinical impact of the phrenic nerve stimulation with the remedē System in adults with CSA is low. Further large, randomized, comparative, controlled studies are needed to determine the safety and efficacy, define optimal patient selection, and assess long-term effect of phrenic nerve stimulation on CSA-related morbidity and mortality.

Wang et al. (2023) completed a meta-analysis to evaluate the efficacy of phrenic nerve stimulation in patients with CSA. The meta-analysis included 10 studies with a total of 580 participants. The outcomes measured included AHI, central apnea index, arousal index, percentage of sleep with oxygen saturation < 90%, Epworth Sleepiness Scale, and sleep efficiency. Researchers noted significant reductions in AHI ($p < 0.00001$), central apnea index ($p < 0.00001$), and arousal index ($p = 0.0002$) following implantation and therapy via phrenic nerve stimulation. Sleep efficiency, Epworth Sleepiness Scale, and percentage of sleep with oxygen saturation < 90% were not significantly different following phrenic nerve stimulator implantation and therapy ($p > 0.05$). Researchers noted that "the use of phrenic nerve stimulation appears to be safe and feasible in patients with CSA; however, larger, independent [randomized controlled trials] are required to investigate the efficacy and long-term effect of phrenic nerve stimulation," particularly regarding Epworth Sleepiness Scale and percentage of sleep with oxygen saturation < 90%.

Potratz et al. (2021) completed a single-center, prospective, open-label study to determine "the effects of phrenic nerve stimulation on functional physical performance capacity and hypoxemic burden in patients with heart failure and CSA." Inclusion criteria included heart failure with preserved or reduced ejection fraction and optimal guideline-driven heart failure therapy for a minimum of 6 months at the time of phrenic nerve stimulator implantation. Participants were excluded if they used any mask-based therapies for CSA, any malignancies requiring treatment, end-stage renal disease or dialysis, and heart valve disease that impacted hemodynamic status. Eligibility for phrenic nerve stimulator implantation included AHI ≥ 20, the proportion of central apneas being ≥ 50% with a central apnea index ≥ 30, and the proportion of obstructive apnea events ≤ 20%. The primary outcome measured was the "change in symptom-limited standardized 6-minute walk test and hypoxemic burden before phrenic nerve stimulator implantation and after six

months of phrenic nerve stimulation." Secondary outcomes measured included left ventricular ejection fraction, central respiratory events in total, central apnea index, AHI, hypopnea index, obstructive apnea index, and hypoxemic burden (time with oxygen saturation < 90% on polysomnogram) prior to implantation and after 6-months of phrenic nerve stimulation treatment. The study enrolled a total of 24 participants with approximately 15 in NYHA class II heart failure and 9 in NYHA class III heart failure. Baseline AHI was 38.1±17.9 and 6-minute walk test distance was 369.5±163.5m. The AHI decreased to 17.3±9.4 and the 6-minute walk test distance improved to 410±169.7m (p = 0.035) at the 6-month follow-up. The total number of central respiratory events at baseline was 109.5±102.5 and improved significantly to 38.6±53.5 (p = 0.027) at the 6-month follow-up. Baseline hypopnea index was 14.5±9.7 compared to 17.9±15.8 (p = 0.39) at 6-month follow-up. Baseline central apnea index was 18±16.8 compared to 7.2±10 (p = 0.02) at 6-month follow-up. Baseline obstructive apnea index was 2±2.4 compared to 5.7±9 (p = 0.2) at 6-month follow-up. The hypoxemic burden at baseline was 81±55.7 minutes and decreased significantly to 27.8±42.7 minutes (p < 0.01) at 6-month follow-up. Left ventricular ejection fraction remained relatively the same with a baseline of 42.4±13.4 compared to 41.9±14.7 at the 6-month follow-up. Researchers were not able to establish heart failure associated benefits with the implantation of phrenic nerve stimulation. However, significant improvements were noted in physical capacity (6-minute walk test distance), hypoxemic burden (time with oxygen saturation < 90%), and AHI. Limitations of this study included a small sample size, lack of a control group, and the results cannot be generalized to smaller treatment centers.

Costanzo et al. (2016) performed an industry-supported, multicenter, prospective, randomized controlled sham study that served as the pivotal trial for FDA approval of the remedē System. The study aimed to determine the safety and assess the effectiveness of the remedē System in reducing AHI in moderate to severe CSA (AHI of least 20 events/hour shown on polysomnogram). Central apneas needed to be > 50% of the apneas, happen at least 30 times per night, and the obstructive apnea index needed to be < 20%.

A total of 151 adult participants (at least 18 years of age, mean age 65 years, 89% male, 95% Caucasian) who were stable for at least 30 days and had appropriate guideline-based therapy underwent device implantation in the pectoral region. Participants were randomly assigned (1:1) by a computer-generated method stratified by site to either stimulation [treatment; 73 of 151 (52%) subjects having the device activated] or no stimulation (78 serving as controls with no activation; n=78) for 6 months. All patients received neurostimulation for the 6 months following the initial 6 months. Treatment assignment was known to both patients and clinicians. The system was activated one month after insertion and activation gradually increased in the treatment group until diaphragmatic capture without disrupting sleep was accomplished.

- 64% had heart failure, but patients with stage D heart failure were excluded.
- Other exclusion criteria included phrenic nerve palsy, cerebrovascular event in previous 12 months, central sleep apnea secondary to opioids, and advanced renal disease.

The primary effectiveness endpoint was the proportion of the treatment patients versus controls who achieved a 50% or greater reduction in AHIs measured in a polysomnogram lab. The primary safety endpoint was freedom from adverse events during the 12 months. Subjects were evaluated regularly until the end of the trial. After 6 months, the remedē System was activated in the sham group. Effectiveness was based on modified intention to treat data at 6 months (n=141). A significantly higher number of subjects in the active remedē System group had a 50% or better reduction in AHI from baseline to 6 months post-procedure (p<0.0001).

- Baseline AHIs were 48.8 + 19.3 in the treatment group and 43.7 + 16.8 in the control group.
- After 6 months, results showed that 51% of the patients with the active implant (35/68 or 51%) had at least a 50% reduction in their AHI from baseline. The control group (subjects with an inactive implant) had an 11% decrease in their AHI (8/73 or 11%). However, the mean AHI still remained elevated at a mean of 25 although 26 of the 35 had an AHI < 20.
- Other statistically significant improvements between the groups were the arousal events per hour, the percent of rapid eye movement sleep, the oxygen desaturation > 4%, and the Epworth Sleepiness Scale. Treatment patients also had higher scores on the patient global assessment.
- A total of 76% of subjects in the remedē System group reported improvement in quality of life. Safety results were based on intention to treat data for 12 months (n=151). There were seven deaths, but none found to be related to the device or treatment.

The most common adverse events reported included concomitant device interaction, implant site infection, and

swelling and local tissue damage or pocket erosion. The number of subjects free from serious adverse events was 91% (95% CI, 86% to 95%); however, 13 subjects had serious adverse events including impending pocket erosion, implant site infection, lead dislodgement, concomitant device interaction, elevated transaminase, extra-respiratory stimulation, implant site hematoma, lad component failure, lead displacement, and non-cardiac chest pain. The remedē System should not be used by patients with an active infection or by patients who are known to require magnetic resonance imaging. Transvenous neurostimulation could provide a treatment option for central sleep apnea. Limitations of the study included a low percentage of female subjects, potential referral bias, and a loss to follow-up.

A summary of the pivotal trial found the following:

- 87% completed trial, 93% included in analyses (exclusions decreased the numbers to 68 in the treatment and 73 in the control group).
- Comparing neurostimulation vs. no stimulation:
  - ≥ 50% improvement in apnea-hypopnea index at 6 months in 51% vs. 11% ($p < 0.0001$, NNT 3)
  - Treatment-related serious adverse events at 12 months in 8% vs. 9% (no p value reported)
- Neurostimulation was associated with improved central apnea index, arousal index, oxygen desaturation, sleepiness score, and health-related quality of life at 6 months ($p < 0.0001$ for each).
- Subjects who experienced non-serious adverse events were 48%. Implants were unsuccessful in 5 subjects, and the rate of explants was 5.3% (8/151).
- 7 patients died (unrelated to implant, system, or treatment), of whom 4 died in first 6 months (2 deaths in each group, treatment group and control group); 3 additional deaths occurred in the second six months but considered not related to the device.

Costanzo et al. (2018) reported on the 12-month outcomes of the Costanzo et al. (2016) pivotal trial. At 12 months post remedē System implantation, the treatment group had an active device for 12 months (n=54), and the control group had an active device for 6 months (n=65). After 12 months, 67% of the treatment group had a ≥ 50% reduction in AHI from baseline (36 of 54; 95% CI, 53% to 78%). Subjects continued to have improvements in sleep metrics, oxygenation, and quality of life. For the control group with an active device for 6 months, 55% of subjects had a ≥ 50% reduction in AHI from baseline (36 of 65; 95% CI, 43% to 67%). At 12 months follow-up, there were 7 reported deaths, but they were not related to the procedure or device. The freedom from serious adverse events at 12 months for the intent-to-treat population was 91% (138 of 151; 95% CI, 86% to 95%). The authors concluded that the remedē System improves sleep metrics and quality of life for at least 12 months without safety concerns. However, the evidence to date remains insufficient to demonstrate the safety and efficacy of this new implantable device, with 12-month safety outcomes data available from a single randomized controlled sham study. Large, randomized, controlled studies are needed to assess long-term outcomes.

Costanzo et al. (2021) reported on the 5-year safety and efficacy outcomes from the pivotal trial conducted by Costanzo et al. (2016). Approximately 53 out of 151 participants were available for follow-up at 5 years with 42 participants being included for analysis. Participants were asked to undergo an overnight polysomnogram at baseline and then at 1-, 2-, and 5-years. A home polysomnography study was used for follow-up at the 3-year follow-up. The analysis of 5-year outcomes showed sustained improvement in all sleep-related disordered breathing events. The most significant improvements were noted in AHI, central apnea index, 4% oxygen desaturation index, minutes of sleep with oxygen saturation < 90%, and percentage of rapid eye movement sleep. The AHI was 46 at baseline, 18 at 1-year, 16 at 2-years, 14 at 3-years, and 17 at 5-years ($p < 0.001$). The central apnea index was 23 at baseline and improved to 1 at each subsequent follow-up period ($p < 0.001$). The 4% oxygen desaturation index improved from 39 at baseline to 15 at 5-years ($p < 0.001$). The minutes of sleep with oxygen saturation < 90% at baseline was 31, 11 at 1-year, 13 at 2-years, and 11 at 5-years ($p=0.134$). The percentage of rapid eye movement sleep at baseline was 10, 14 at 1-year, 17 at 2-years, and 21 at 5-years ($p=0.001$). No serious adverse events were reported between the 2- and 3-year follow-up periods. A total of four serious adverse events were reported between years 3 and 5 and included stimulation lead dislocation “followed by multiple procedures with hospitalization to extract the lead and place a new lead, which subsequently dislodged and required another replacement procedure” (n=1), stimulation lead component failure requiring a hospital stay for lead replacement (n=2), and “implant site infection after device replacement requiring device explant and hospital stay” (n=1). No deaths were reported. Researchers noted that long-term results demonstrated consistent safety and efficacy of phrenic nerve stimulation for CSA.

Abraham et al. (2015) evaluated transvenous unilateral phrenic nerve stimulation to treat CSA in a prospective, multicenter, non-randomized study to determine feasibility, safety, and efficacy. Central apnea was defined as the

absence of airflow and respiratory effort for >10 sec and mixed apnea was considered a minimum of three respiratory efforts with absent inspiratory effort at the beginning of the episode. Eligible patients had an AHI of at least 20 and at least one-half of the events were of central origin. Patients with obstructive events > 20% were excluded. Fifty-seven (n=57) patients with CSA underwent baseline polysomnography followed by transvenous phrenic nerve stimulation system implantation and follow-up. Feasibility was assessed by implantation success rate and therapy delivery. Safety was evaluated by monitoring of device- and procedure-related adverse events. Efficacy was evaluated by changes in the apnea-hypopnea index at 3 months. Quality of life at 6 months was evaluated using a sleepiness questionnaire, patient global assessment, and, in patients with heart failure at baseline, the Minnesota Living with Heart Failure Questionnaire. The study met its primary end point, demonstrating a 55% reduction in apnea-hypopnea index from baseline to 3 months (49.5 ± 14.6 episodes/h vs. 22.4 ± 13.6 episodes/h of sleep; $p < 0.0001$; 95% confidence interval for change: -32.3 to -21.9). Central apnea index, oxygenation, and arousals significantly improved. Favorable effects on quality of life and sleepiness were noted. Epworth Sleepiness Scores (EPS) were improved at six months and 36 patients with heart failure showed improvement by an average of 10 points in the Minnesota Living With Heart Failure Questionnaire ($p=0.0009$). Device- or procedure-related serious adverse events occurred in 26% of patients through 6 months post therapy initiation, predominantly due to lead repositioning early in the study. Therapy was well tolerated. Efficacy was maintained at 6 months. The authors concluded, transvenous unilateral phrenic nerve stimulation appears safe and effective for CSA, however these findings should be confirmed in a large, prospective randomized, controlled trial (NCT01124370). Limitations of the study included small sample size, lack of a control arm, short follow-up duration, and potential for referral bias.

Jagielski et al. (2016) evaluated 12-month outcomes of the study by Abraham et al. (2015). A total of 47 patients with CSA were treated with the remedē System for a minimum of 3 months. Sleep-disordered breathing parameters were evaluated by polysomnography at 3, 6, and 12-month follow-up. Sleep symptoms and quality of life was also evaluated; 41 patients completed all follow-up polysomnograms and were included in the analysis. At 12 months, there was sustained improvement compared with baseline in the (AHI), central apnea index and there was sustained improvement in the oxygen desaturation index, rapid eye movement sleep and sleep efficiency: AHI (49.9 ± 15.1 versus 27.5 ± 18.3 events/hour, $p < 0.001$); central apnea index (28.2 ± 15.0 versus 6.0 ± 9.2 events/hour, $p < 0.001$); oxygen desaturation index (46.1 ± 19.1 versus 26.9 ± 18.0 events/hour, $p < 0.001$), rapid eye movement sleep (11.4 ± 6.1 % versus 17.1 ± 8.0 %, $p < 0.001$), and sleep efficiency (69.3 ± 16.9 % versus 75.6 ± 17.1 %, $p = 0.024$). There were also continued favorable effects on sleepiness and quality of life; 3 deaths unrelated to remade System therapy and 5 serious adverse events occurred over 12 months of follow-up. The authors noted the main limitations of the study were the non-randomized (lack of a control group), open-label nature of the trial, the small sample size and small number of female subjects enrolled in the study, and the loss to follow-up. The researchers recommended larger, randomized, controlled trials to confirm their results.

Ponikowski et al. (2012) conducted a prospective, non-randomized trial to determine the feasibility of using unilateral transvenous phrenic nerve stimulation for the treatment of CSA in patients with heart failure. Thirty-one patients from six centers were selected; 16 were able to undergo two nights of polysomnography and were enrolled in the study. Measurements of AHI, central apnea index, obstructive apnea index, hypopnea index, arousal index, and 4% oxygen index were performed prior to and after phrenic nerve stimulation of 271 + 71 minutes. The AHI, central apnea index, arousal index, and 4% oxygen index significantly improved although there were no significant changes in the hypopnea index or obstructive apnea index. The AHI remained elevated at 23 (12-27) events/hour. There were two adverse events of a lead thrombus and an episode of ventricular tachycardia, neither of which was considered directly related to the phrenic nerve stimulation therapy. The authors stated the study provided a strong proof of concept and that large-scale, long-term randomized controlled trials using an implanted system were needed. The study was supported by Respicardia, Inc. and six of the authors were paid consultants and one an employee of this company.

National and Specialty Organizations

The **American Thoracic Society (ATS)** published a research statement for research priorities for patients with heart failure and CSA (Orr et al. 2021). The research statement mentions the documented improvements in AHI and central apnea index associated with phrenic nerve stimulation for short-term outcomes (≤ 1 year). The statement concludes that additional research is needed with a focus on long-term outcomes.

The **American Academy of Sleep Medicine (AASM)** issued an updated guideline in 2016 on adaptive servo-ventilation in patients with CSA (Aurora et al. 2016). The guideline does not include diaphragmatic/phrenic nerve

stimulation or diaphragm pacing as a recommended treatment for this condition. The AASM also published the *Practice Parameters with an Evidence-Based Literature Review and Meta-Analyses* with the following recommendations (Aurora et al. 2012):

- Optimize treatment of heart failure.
- CPAP indicated as initial treatment to normalize the AHI (< 15).*
- Nocturnal oxygen therapy is indicated*
  - May be given as supplement to CPAP or for patients who are intolerant of CPAP.
  - Consider repeat sleep study with oxygen to evaluate resolution of central sleep apnea events.
- Second-line options to consider, based on limited evidence.
- Bilevel positive airway pressure in a spontaneous timed mode targeted to normalize the apnea hypopnea index, only if no response to CPAP, oxygen therapy, and adaptive servo ventilation**
- Acetazolamide and theophylline** should be considered only if:
  - Standard medical therapy is optimized.
  - Positive airway pressure therapy not tolerated.
  - Close clinical follow-up provided for adverse effects .

* AASM Standard, **AASM Option

A 2017 update to the guidelines published in 2015 was published by the **American College of Cardiology (ACC), American Heart Association (AHA),** and the **Heart Failure Society of America (HFSA).** The update focused on the management of heart failure. The guideline does not include diaphragmatic/phrenic nerve stimulation or diaphragm pacing as a recommended treatment for heart failure management. The guideline includes the following recommendations regarding sleep disordered breathing:

- In patients with NYHA class II-IV heart failure and suspicious of sleep disordered breathing or excessive daytime sleepiness, a formal sleep assessment is reasonable.
- In patients with cardiovascular disease and obstructive sleep apnea, CPAP may be reasonable to improve sleep quality and daytime sleepiness.
- In patients with NYHA class II-IV heart failure with reduced ejection faction and CSA, adaptive servo-ventilation causes harm.

SUPPLEMENTAL INFORMATION

Definitions

Apnea: cessation of airflow for > 10 seconds

Central Apnea Index: the number of central sleep apnea episodes per hour of sleep

Apnea Hypopnea Index (AHI): The number of apneas plus the number of hypopneas during the entire sleeping period, times 60, divided by total sleep time in minutes; unit: event per hour (AASM 2023).

CODING & BILLING INFORMATION

CPT (Current Procedural Terminology) Codes

Code	Description
33276	Insertion of phrenic nerve stimulator system (pulse generator and stimulating lead[s]), including vessel catheterization, all imaging guidance, and pulse generator initial analysis with diagnostic mode activation, when performed
33277	Insertion of phrenic nerve stimulator transvenous sensing lead (List separately in addition to code for primary procedure)
33278	Removal of phrenic nerve stimulator, including vessel catheterization, all imaging guidance, and interrogation and programming, when performed; system, including pulse generator and lead(s)
33279	Removal of phrenic nerve stimulator, including vessel catheterization, all imaging guidance, and

	interrogation and programming, when performed; transvenous stimulation or sensing lead(s) only
33280	Removal of phrenic nerve stimulator, including vessel catheterization, all imaging guidance, and interrogation and programming, when performed; pulse generator only
33281	Repositioning of phrenic nerve stimulator transvenous lead(s)
33287	Removal and replacement of phrenic nerve stimulator, including vessel catheterization, all imaging guidance, and interrogation and programming, when performed; pulse generator
33288	Removal and replacement of phrenic nerve stimulator, including vessel catheterization, all imaging guidance, and interrogation and programming, when performed; transvenous stimulation or sensing lead(s)

HCPCS (Healthcare Common Procedure Coding) Code

Code	Description
C1823	Generator, neurostimulator (implantable), non-rechargeable, with transvenous sensing and stimulation leads

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

12/13/2023	Policy reviewed, no changes to criteria. Updated Overview, Summary of Medical Evidence, Supplemental Information, and References. IRO Peer Review on November 7, 2023, by a practicing, board-certified physician with specialties in Sleep Medicine and Neurology.
12/14/2022	Policy reviewed, no changes to criteria.
02/08/2021	Policy reviewed, no changes to criteria.
04/23/2020	Policy reviewed, no changes.
03/11/2019	New policy. IRO Peer Review on January 24, 2019, by a practicing, board-certified physician with specialties in Internal Medicine and Pulmonary Disease.

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APPENDIX

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