

## Baroreflex Stimulation Devices

**Effective:** February 1, 2023

**Next Review:** September 2023

**Last Review:** December 2022

### IMPORTANT REMINDER

Medical Policies are developed to provide guidance for members and providers regarding coverage in accordance with contract terms. Benefit determinations are based in all cases on the applicable contract language. To the extent there may be any conflict between the Medical Policy and contract language, the contract language takes precedence.

PLEASE NOTE: Contracts exclude from coverage, among other things, services or procedures that are considered investigational or cosmetic. Providers may bill members for services or procedures that are considered investigational or cosmetic. Providers are encouraged to inform members before rendering such services that the members are likely to be financially responsible for the cost of these services.

### DESCRIPTION

Baroreflex stimulation devices are implantable devices used to provide electrical stimulation of the baroreceptors in the carotid arteries, aiming to control blood pressure.

### MEDICAL POLICY CRITERIA

**Note:** Requests for services related to direct complications or consequences that arise from noncovered services should be reviewed by applicable member contract language. Specific member contract language takes precedence over medical policy.

Use of any baroreflex stimulation implanted device, including but not limited to the Barostim neo® Legacy System, is considered **investigational** for all indications, including but not limited to resistant hypertension and heart failure.

*NOTE: A summary of the supporting rationale for the policy criteria is at the end of the policy.*

### CROSS REFERENCES

None

## BACKGROUND

Baroreflex stimulation devices are implantable devices used to provide electrical stimulation of the baroreceptors in the carotid arteries, aiming to control blood pressure. This treatment may also be referred to as baroreflex activation therapy (BAT). The baroreceptors are the part of the autonomic nervous system that regulates basic physiologic functions such as heart rate and blood pressure. When these receptors are stretched, as occurs with increases in blood pressure, the baroreflex, also called the baroreceptor reflex, is activated. A signal is sent to the brain inhibiting sympathetic nervous system output and increasing parasympathetic nervous system output. The effect of this activation is to slow the heart rate and decrease blood pressure.

The goal of treatment of hypertension is prevention of the long-term complications of uncontrolled hypertension including stroke, heart failure, and renal failure. Use of baroreflex stimulation devices has been proposed as a treatment for hypertension resistant to standard medication and as a treatment for heart failure. This type of hypertension, known as resistant hypertension, is defined as elevated blood pressure despite treatment with at least three antihypertensive agents at optimal doses. Resistant hypertension is a relatively common condition. Preliminary investigation has also begun for BAT as a treatment of heart failure.

Currently available baroreflex stimulation devices consist of three components:

- A pulse generator, implanted subcutaneously in the upper chest wall, which controls and delivers electrical energy;
- Carotid sinus leads which are thin wires with electrical contacts that are placed in contact with the carotid baroreceptors to conduct electrical energy from the pulse generator to the baroreceptors; and
- An external programmer used by clinicians to turn the system on and off and regulate the electrical signal delivered to the baroreceptors.

## REGULATORY STATUS

In December 2014, the Barostim neo<sup>®</sup> Legacy System (CVRx, Inc.) received Humanitarian Device Exemption (HDE) approval from the U.S. Food and Drug Administration (FDA) for use in patients with resistant hypertension who have had bilateral implantation of the Rheos<sup>®</sup> Carotid Sinus Leads Models 1010R, 1010L, 1014L, and 1014R (which have been discontinued and are obsolete) and were determined responders in the Rheos<sup>®</sup> pivotal clinical study.

In 2019, Barostim neo<sup>™</sup> was granted premarket approval (PMA P180050) and is indicated for the improvement of symptoms of heart failure such as quality of life, six-minute hall walk and functional status, for patients who remain symptomatic despite treatment with guideline-directed medical therapy, are NYHA Class III or Class II (with a recent history of Class III), have a left ventricular ejection fraction  $\leq 35\%$ , and a NT-proBNP  $< 1600$  pg/ml, excluding patients indicated for Cardiac Resynchronization Therapy (CRT) according to AHA/ACC/ESC guidelines.

No other devices or indications have received FDA approval.

## EVIDENCE SUMMARY

In order to determine the safety and effectiveness of baroreflex stimulation therapy for

treatment of resistant hypertension and heart failure, large, well-designed randomized controlled trials (RCTs) that compare this therapy to both sham and standard medical treatment (intensified drug therapy) are needed. Further, for these chronic conditions, studies with long-term follow-up are necessary to determine the durability of any beneficial treatment effects.

## TREATMENT RESISTANT HYPERTENSION

### Systematic Reviews

A 2018 systematic review by Chunbin included 12 studies (one RCT, 11 prospective) in a qualitative analysis and five prospective studies in a meta-analysis<sup>[1]</sup>. Although the authors concluded that office blood pressure was significantly reduced by BAT, the evidence summarized had a high risk of bias and very limited randomized trial data. There is insufficient evidence to fully evaluate the safety and efficacy of BAT for patients with resistant hypertension.

### Randomized Controlled Trials

In 2010, Bisognano reported the results of a 12 month-long double-blind RCT, the Rheos® pivotal trial.<sup>[2]</sup> Patients with resistant hypertension (n=265) had the Rheos system implanted and after 30 days were randomized (2:1 allocation) to the device turned on or off for a six month period. For the next six months (12-month total follow-up), all patients had the device turned on. Overall safety and efficacy were defined as:

- A greater proportion of treatment patients (versus control patients) achieving at least a 10 mm Hg decrease in systolic blood pressure (SBP) at six months (acute efficacy);
- More than 65% of treatment patients maintaining their SBP response (at least 50% of an initial decrease of  $\geq 20$  mm Hg at six months) over the 6-12 month time period (sustained efficacy);
- At least 82% of patients free from procedural adverse events after 30 days (procedural safety);
- No more than 15% more treatment-related adverse events in the treatment group compared with the control group at six months (therapy safety); and
- At least 72% of all patients free from device-related adverse events at 12 months (device safety).

Findings for the primary efficacy outcomes from this study were mixed. There was insufficient evidence for establishing short term efficacy: at six months, there was no difference in the proportion of treatment vs. control group patients who experienced a SBP decrease (54% vs. 46%,  $p=0.97$ ). Support for long-term efficacy, as defined by the researchers, was found: 88% of patients in the treatment group who had a SBP decrease at six months maintained their decrease to 12 months. The average amount of SBP decrease was not reported, nor was diastolic blood pressure (DBP) reported.

Outcomes for the primary safety endpoints were also mixed. At 30 days, 75% of patients were free of procedural adverse events, a rate which failed to meet the defined criteria for procedural safety (82% rate free from procedural adverse events,  $p=1.00$ ). Major procedural adverse events (n=68) consisted of surgical complications and transient or residual nerve injuries. At six months, rates of adverse effects for therapy safety were similar between the treatment and control groups (91.7% vs. 89.3%,  $p<0.001$  for non-inferiority), indicating the primary endpoint of therapy safety was met. At 12 months, 87.2% of patients were free of

device-related adverse events, exceeding the predefined threshold of 72%, indicating support for device safety.

In summary, this trial attempted to establish efficacy and safety of the Rheos device for the treatment of resistant hypertension. Although some of the results are potentially promising, findings from this study should be interpreted with caution as the primary short-term efficacy outcome included a decrease in SBP of as little as 10 mm Hg, which is unlikely to be clinically significant in this patient population, and long term efficacy was not established. Additionally, the procedural safety of this device is uncertain. A quarter (25.5%) of all patients undergoing implantation of this device had complications arising from the procedure within the first 30 days, a rate which the researchers state is higher than that currently seen among patients with implanted pacemakers.

In 2012, Bakris published longer-term follow-up results from the Rheos pivotal trial.<sup>[3]</sup> Patients implanted with the Rheos device were followed for a range of 22 to 53 months and blood pressure and medication use were evaluated. Although the original report of the trial indicated that 265 patients participated, the follow-up report included 57 additional patients, described as those who, “received therapy immediately in an open-label fashion.” Among the 322 patients implanted with BAT, 276 consented for long-term therapy (12 months beyond the end of the Rheos pivotal trial). Although outcome measures similar to those reported above were also estimated in this group of patients, the lack of a comparative treatment group, without which it is not possible to establish relative treatment benefit compared with standard medical therapy, limits interpretation of these findings.

In 2012, Hoppe published the results of a series of patients treated with the Barostim *neo*<sup>®</sup>.<sup>[4]</sup> Thirty patients from seven centers with resistant hypertension were treated with this device and followed for a six-month period. The mean baseline BP was 172/100. At six months, there was a decrease in BP of 26.0 mmHg systolic and 12.4 mmHg diastolic. The percent of patients achieving adequate BP control, defined as a systolic BP or 140 or less, was 43%. There were three perioperative complications, one device pocket hematoma, one wound complication, and one intermittent pain at the insertion site. One additional patient had longer term intermittent pain at the device site. This clinical trial did not include a comparative treatment group, which limits the interpretation of the study results and follow-up was limited in the treatment group.

## **Nonrandomized Studies**

de Leeuw (2017) published longer-term follow-up results from 383 patients of which 143 had completed five years of follow up and 48 had completed six years of follow-up<sup>[5]</sup>. Over the entire cohort, systolic blood pressure fell from  $179 \pm 24$  mm Hg to  $144 \pm 28$  mm Hg ( $p < 0.0001$ ), whereas office diastolic pressure dropped from  $103 \pm 16$  mm Hg to  $85 \pm 18$  mm Hg ( $p < 0.0001$ ). The authors noted that the effect of BAT was greater than average in patients with signs of heart failure and less than average in patients with isolated systolic hypertension.

Other evidence on the efficacy of baroreflex stimulation therapy is limited to a study on the short-term effects of this treatment on ambulatory blood pressure in 44 subjects, and several small case series that reported on baroreflex stimulation therapy in the treatment of resistant hypertension, which found small decreases in blood pressure up to a year after device implantation<sup>[6-9]</sup> and acute effects on blood pressure after device deactivation and reactivation.<sup>[10]</sup> However, evidence from case series is considered unreliable due to inherent methodological limitations, including non-random allocation of treatment, lack of adequate comparison group, small sample sizes, and short follow-up.

## HEART FAILURE

### Systematic Reviews

An inpatient data (IPD) meta analysis was conducted to evaluate the effect of baroreflex activation therapy (BAT) on heart failure symptoms, Quality of Life (QoL) and N-terminal pro-brain natriuretic peptide (NT-proBNP) in Heart failure with reduced ejection fraction (HFrEF).<sup>[11]</sup> Two studies were selected (a total of 554 patients) from a comprehensive list of all trials recruiting HFrEF patients and utilizing the Barostim™ device. Patients were randomized 1:1 to either Guideline Directed Medical Therapy (Control) or BAT + GDMT (BAT) (open label). BAT showed significant improvement in 6MHW distance of 49 m (95% confidence interval [CI] 33, 64), MLWHF QoL of -13 points (95% CI -17, -10), and 3.4 higher odds of improving at least one NYHA class (95% CI 2.3, 4.9) when comparing from baseline to 6 months. This meta-analysis only includes two open label trials with a limited number of patients.

In 2020, Cai published a systematic review with meta-analysis evaluating the efficacy of baroreflex activation therapy for heart failure.<sup>[12]</sup> The meta-analysis included four RCTs and concluded that baroreflex activation therapy significantly improves quality of life score, 6-minute hall walk distance, New York Heart Association (NYHA) class, N-terminal pro-B-type natriuretic peptide (NT-proBNP), and duration of hospitalization compared to control. However, the four RCTs included in the analysis were from the same patient population, the Hope for Heart Failure (HOPE4HF) study (NCT01471860 and NCT01720160) and the analysis did not account for the overlapping population between studies. Therefore, this meta-analysis likely overestimated the true effect of baroreflex activation therapy. The HOPE4HF RCT and post hoc/subgroup analyses are summarized below.

Schmidt (2020) published the results of a rapid systematic review of data on baroreflex activation therapy in patients with heart failure with reduced ejection fraction.<sup>[13]</sup> This rapid review included data from three studies, one of which was the RCT summarized below. The homogeneity across studies precluded meta-analysis. The authors concluded that additional studies are needed before conclusive statements can be made regarding the efficacy of baroreflex activation for patients with heart failure with reduced ejection fraction.

### Randomized Controlled Trials

The safety and effectiveness data reviewed by the FDA for the 2019 premarket approval of the Barostim Neo System was from the Barostim Neo-Baroreflex Activation Therapy for Heart Failure (BeAT-HF) trial. <sup>[14, 15]</sup> BeAT-HF examined the safety and effectiveness of baroreflex activation therapy in heart failure patients with reduced ejection fraction using an Expedited and Extended Phase design. In the Expedited Phase, baroreflex activation therapy plus guideline-directed medical therapy was compared at six months post-implant to guideline-directed medical therapy alone in three intermediate end points: 6-minute hall walk distance, Minnesota Living with Heart Failure Questionnaire, and N-terminal pro-B-type natriuretic peptide (NT-proBNP). The rate of heart failure morbidity and cardiovascular mortality was compared between the arms to evaluate early trending using predictive probability modeling.

In the Expedited Phase, investigators randomized 264 intended use patients. The primary safety endpoint was the major adverse neurological and cardiovascular event (MANCE) -free rate (which was only measured in the baroreflex group); the lower bound of the one-sided 95% CI of the event-free rate had to be > 85%. Results analysts were blinded to arm assignment. At

six months, the MANCE-free rate was 96.8% (121 of 125 patients), and the one-sided 95% lower bound was 92.8% ( $p < 0.001$ ). Effective endpoint results are summarized in Table 1. The FDA concluded from these results that the system was safe for the intended use population, and all effectiveness endpoints showed a statistically significant benefit for baroreflex activation therapy plus guideline-directed medical therapy compared to guideline-directed medical therapy alone. BeAT-HF includes an Extended Phase in which the heart failure morbidity and cardiovascular mortality end point is based on an expected event rate of 0.4 events/ patient/ year in the guideline-directed medical therapy arm. This trial is ongoing.

**Table 1. Six-Month Change from Baseline for Effectiveness Endpoints in the BeAT-HF Expedited Phase Trial**

	6MHWD		QOL <sup>a</sup>		NT-proBNP	
	BAT + GDMT	GDMT	BAT + GDMT	GDMT	BAT + GDMT	GDMT
n	118	120	120	125	120	123
Mean (SD)	48.6 (66.3)	-7.9 (88.4)	-20.7 (25.4)	-6.2 (20.1)	-21.1% (0.4)	3.3% (0.3)
95% CI	36.5 to 60.7	-23.9 to 8.1	-25.3 to -16.1	-9.8 to -2.7	-32.3% to -8.2%	-8.9% to 17.2%
Difference	60.1		-14.1		-24.6%	
95% CI	40.3 to 79.9		-19.2 to -8.9		-37.6% to -8.7%	
p-value	<0.001		<0.001		0.004	

Notes: 6MHWD: 6-minute hall walk distance; BAT: Barostim therapy; BeAT-HF: Barostim Neo-Baroreflex Activation Therapy for Heart Failure; CI: confidence interval; GDMT: guideline based medical treatment; NA: not applicable; QOL: quality of life; SD: standard deviation. <sup>a</sup> Measured by the Minnesota Living With Heart Failure Quality of Life questionnaire

In 2015, Abraham published a non-blinded trial that included 146 patients with New York Heart Association (NYHA) class III heart failure and ejection fraction  $\leq 35\%$  despite guideline-directed medical therapy.<sup>[16]</sup> Patients were randomized to receive baroreflex stimulation (CVRx Barostim Neo system) in addition to medical therapy ( $n=76$ ) or continued medical therapy alone ( $n=70$ ) for 6 months. The primary safety outcome was the proportion of patients free from MANCE. The authors specified three primary efficacy endpoints, changes in NYHA functional class, quality of life score and 6-minute walk distance.

The overall MANCE-free rate was 97.2%, as rates were not reported separately for the baroreflex stimulation and control groups. In terms of the efficacy outcomes, there was significant improvement in the baroreflex stimulation group versus the control group on each of the three outcomes. Significantly more patients in the treatment group (55%) had at least a 1-class improvement in NYHA functional class compared to the control group (24%),  $p < 0.002$ . The mean quality of life score, as assessed by the Minnesota Living with Heart Failure Questionnaire, was significantly improved in the treatment versus control group (-17.4 points versus 2.1 points, respectively,  $p < 0.001$ ). Similarly, the 6MWD was significantly improved in the treatment compared with the control group (mean: 59.6 meters and 1.5 meters, respectively,  $p = 0.004$ ).

A subsequent publication from this study compared the effects of baroreflex stimulation in those with and without cardiac resynchronization therapy (CRT).<sup>[17]</sup> The authors reported that quality of life score, six-minute hall walk distance, left ventricular ejection fraction, NT-proBNP, and heart failure hospitalizations were significantly improved in the baroreflex stimulation group without CRT, while those in the stimulation group without CRT were not significantly different than non-stimulation controls.

Limitations of this study included a relatively small sample size for common condition, relatively short intervention period and lack of blinding; some of the positive findings on the subjective patient-reported outcomes may be due at least in part to a placebo effect. Also, the source of study funding in the Abraham study was not reported, and nearly all of the authors had financial links to the manufacturer. Additional RCTs with larger sample sizes and longer follow-up are needed to confirm these positive findings.

### Nonrandomized Studies

The remaining evidence for baroreflex stimulation therapy in the treatment of heart failure (HF) is limited to a nonrandomized proof-of-concept study that included 11 patients with NYHA class III HF with ejection fraction (EF) <40%, optimized medication therapy, and ineligible for cardiac resynchronization.<sup>[18]</sup> Efficacy was assessed with serial measurement of muscle sympathetic nerve activity (MSNA) and clinical measures of quality of life and functional capacity. During the six month therapy period there was a significant decrease in MSNA along with significant improvements in baroreflex sensitivity, EF, NYHA class, quality of life, and 6 minute hall walk distance). A decrease in hospitalization and emergency department visits for worsening HF were also reduced. Longer-term follow up of this group found that these improvements were maintained at 12 and 21.5±4.2 months after therapy activation.<sup>[19]</sup> A 43-month follow-up to this study showed that seven of the original 11 patients had survived and the improvements from BAT had been maintained at 43.5±2.1 months<sup>[20]</sup>. Limitations of this study are the small number of subjects, and lack of a control group. This preliminary study was intended for proof-of-concept and hypothesis development for future large, well-designed studies.

## PRACTICE GUIDELINE SUMMARY

There are no evidence-based clinical practice guidelines that recommend the use of baroreflex stimulation devices for the treatment of any condition, including but not limited to resistant hypertension and heart failure.

## SUMMARY

There is not enough evidence to determine the overall impact of baroreflex devices on health outcomes for any condition. There are no evidence-based clinical practice guidelines that recommend the use of baroreflex stimulation devices for the treatment of any condition. Therefore, use of baroreflex stimulation is considered investigational for all indications, including but not limited to resistant hypertension and heart failure.

## REFERENCES

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## CODES

Codes	Number	Description
CPT	0266T	Implantation or replacement of carotid sinus baroreflex activation device; total system (includes generator placement, unilateral or bilateral lead placement, intra-operative interrogation, programming, and repositioning, when performed)
	0267T	Implantation or replacement of carotid sinus baroreflex activation device; lead only, unilateral (includes intra-operative interrogation, programming, and repositioning when performed)
	0268T	Implantation or replacement of carotid sinus baroreflex activation device; pulse generator only (includes intra-operative interrogation, programming, and repositioning when performed)
	0269T	Revision or removal of carotid sinus baroreflex activation device; total system (includes generator placement, unilateral or bilateral lead placement, intra-operative interrogation, programming, and repositioning, when performed)
	0270T	Revision or removal of carotid sinus baroreflex activation device; lead only, unilateral (includes intra-operative interrogation, programming, and repositioning when performed)
	0271T	Revision or removal of carotid sinus baroreflex activation device; pulse generator only (includes intra-operative interrogation, programming, and repositioning when performed)
	0272T	Interrogation device evaluation (in person), carotid sinus baroreflex activation system, including telemetric iterative communication with the implantable device to monitor system diagnostics and programmed therapy values, with interpretation and report (e.g., battery status, lead impedance, pulse amplitude, pulse width, therapy frequency, pathway mode, burst mode, therapy start/stop times each day)
	0273T	Interrogation device evaluation (in person), carotid sinus baroreflex activation system, including telemetric iterative communication with the implantable device to monitor system diagnostics and programmed therapy values, with interpretation and report (e.g., battery status, lead impedance, pulse amplitude, pulse width, therapy frequency, pathway mode, burst mode, therapy start/stop times each day); with programming
HCPCS	C1825	Generator, neurostimulator (implantable), non-rechargeable with carotid sinus baroreceptor stimulation lead(s)

**Date of Origin:** December 2011