From the Eastern Vascular Society

Early experience with baroreflex activation therapy from a vascular surgery perspective

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ABSTRACT

Baroreflex activation therapy (BAT) is an emerging device-based treatment for patients with heart failure with a reduced ejection fraction refractory to maximally tolerated goal-directed medical therapy. Currently, there is sparse literature on the critical role that vascular surgeons serve in the delivery of this novel therapy. This single-institution series describes the creation of a BAT program and elaborates on the function of vascular surgeons in the multidisciplinary heart failure team. The preoperative evaluation, perioperative care, and postoperative course of patients receiving BAT from March 2022 to August 2023 were retrospectively analyzed. Eleven patients were evaluated by a dedicated heart failure cardiologist for medical eligibility and assessed by a vascular surgeon for technical feasibility. Of the 11 patients, 7 were men (63.6%). The median age was 60.5 years (range, 44-73 years). No patient (0.00%) had existing carotid artery disease, and one patient (9.1%) had undergone previous neck radiation therapy. All 11 patients (100%) had an existing cardiac implantable electronic device, and BAT implantation was performed on the same side as the cardiac implantable electronic device in two patients (18.1%). Four patients (36.4%) required preoperative hospital admission for medical optimization before surgery. The median length of surgery was 82 minutes (range, 58-113 minutes), and the median length of stay in the hospital after surgery was 1 day (range, 0-6 days). No major adverse neurologic or cardiovascular events, cranial nerve injuries, device complications requiring reintervention, or heart failure-related mortality at 6 months occurred. Three patients (27.3%) experienced extraneous stimulations, which affected BAT tolerability. Within 6 months after BAT implantation, no significant improvements were observed for several heart failure disease burden markers compared with 6 months before BAT implantation. Our early results demonstrate that BAT implantation is a safe procedure with rare complications. Vascular surgeons play an important role in the multidisciplinary delivery of this novel device-based therapy. More data are needed to understand whether BAT is beneficial in the treatment of heart failure with a reduced ejection fraction refractory to maximally tolerated goal-directed medical therapy. (J Vasc Surg Cases Innov Tech 2024;10:101464.)

Keywords: Baroreflex activation therapy; Barostim; Carotid; Extraneous stimulation; Heart failure

Chronic overactivation of the sympathetic autonomic nervous system is well established as one of the major drivers in the pathophysiology of heart failure with a reduced ejection fraction (HFrEF), regardless of the etiology.[1,2] Logically, many heart failure therapies aim to counteract this maladaptive response. To reduce the burden of polypharmacy and to augment existing treatment options, device-based therapies are now being considered for these challenging patients.[3,4]

Baroreflex activation therapy (BAT) is predicated on the concept of direct electrical stimulation of carotid baroreceptors, resulting in centrally mediated afferent sympathetic inhibiting fibers and efferent activating vagal fibers and, ultimately, leading to improvement of heart failure symptoms.[5] In August 2019, the Barostim NEO system (CVRx) was granted premarket approval (approval no. P180050) for the treatment of HFrEF refractory to maximally tolerated goal-directed medical therapy (GDMT). At the time of this study, the Barostim NEO system was the only BAT device currently available to patients in the United States.[6]

Since its introduction, several studies have analyzed the efficacy of Barostim and its impact on patients' quality of life, exercise capacity, N-terminal pro-brain natriuretic peptide (NT-proBNP) levels, and other markers of HFrEF disease burden.[7,8] Nonetheless, these devices remain investigational, and robust evidence to inform best clinical practice is currently unavailable.

The aim of this study is to describe the implementation of a BAT program at a large tertiary academic institution, elaborate on the role of vascular surgeons in the

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multidisciplinary heart failure team, and retrospectively analyze the outcomes of patients who received the BAT device since the inception of the program.

METHODS

This retrospective review entails the first 18 months after initiation of the BAT program, starting from the beginning of March 2022 to the end of August 2023. Details of the first 11 patients receiving implantation of the BAT device are included. The institutional review board of the University of Miami approved this study and waived the requirement for patient written informed consent. No external sources of funding were provided for this study.

Study population. The University of Miami advanced heart failure team identified a subset of patients with advanced heart failure suitable for BAT, as guided by established criteria.[7-9] Additional consideration was given to patients challenged by the burden of regular maximal GDMT adherence. These patients did not meet the criteria for durable mechanical circulatory support[10] or cardiac transplantation and often had already received other advanced heart failure interventions.

Multidisciplinary BAT team. Next, vascular surgeons and advanced heart failure team stakeholders jointly established a protocolized workflow for BAT delivery (Fig 1). The team includes a dedicated heart failure cardiologist responsible for all patients receiving BAT and device maintenance, a single vascular surgeon responsible for managing device-related complications, cardiovascular anesthesiologists familiar with BAT, the preoperative considerations, and the expected intraoperative changes, an advanced practice provider assisting with medical management of heart failure, and a CVRx device representative who supports all stakeholders with education and assists with BAT titrations intraoperatively and postoperatively. Additional buy-in from the hospital administration is required to guarantee reimbursement. Patients continue working with ancillary staff—physical therapists, occupational therapists, dieticians, mental health professionals, and pharmacists—to achieve lifestyle optimization.

Device specifications and implantation. The Barostim NEO2 system consists of an implantable pulse generator (IPG), a carotid sinus lead (CSL) with a 2-mm carotid electrode, and an external programmer (ie, a laptop or tablet with software allowing for noninvasive adjustment of BAT parameters and tracking relevant data). An electrical pulse train of programmed frequency, pulse width, and constant current amplitude is delivered according to different schedules, programmed in accordance with the expected fluctuations in hemodynamics throughout the day.

Procedure planning and selection. On confirmation of medical suitability, vascular surgeons evaluate the technical feasibility. First, a comprehensive history and physical examination are performed, much like before carotid revascularization. High-risk features are assessed, such as previous neck surgery or radiation, tracheostomy, contralateral vocal cord paralysis, and neck or spine immobility.

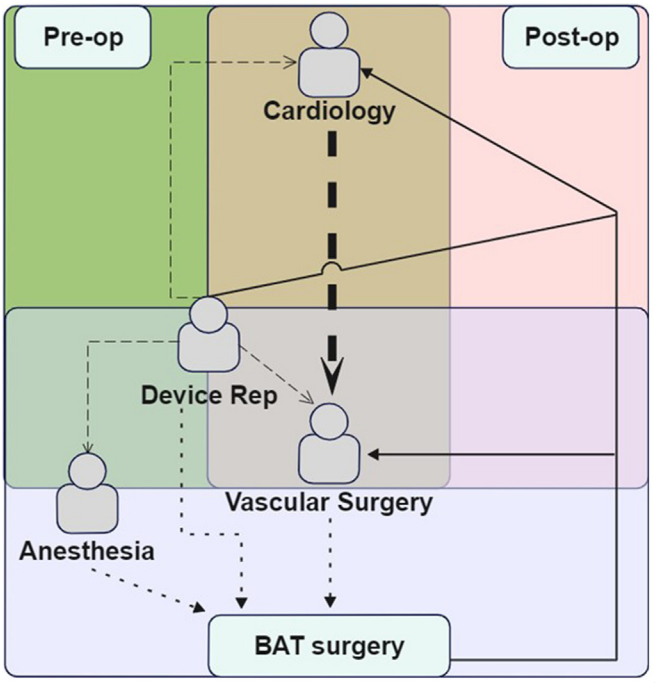

Fig 1. Baroreceptor activation therapy (BAT) workflow protocol. *Thick dashed line* indicates referrals from cardiology to vascular surgery in the preoperative (*Pre-op*) and postoperative (*Post-op*) periods; *thin dashed line*, education and support of all stakeholders by the manufacturer; *dotted line*, parties involved in device implantation and reintervention surgery; and *solid line*, parties involved in postoperative BAT maintenance and surveillance. *Rep*, Representative.

The chest wall anatomy is evaluated, including recording the presence of any existing cardiac implantable electronic devices (CIEDs), chemotherapy ports, subglandular breast implants, and any other interfering anatomy. Previous device migration could suggest weak fascial tissue and could complicate IPG anchoring. Per the device manufacturer, right-sided implantation is preferred based on the results from previous trials.

A carotid duplex ultrasound scan is obtained to evaluate the carotid bifurcation location, abnormalities, and the presence of atherosclerosis. Strict adherence to the instructions for use are observed by the vascular surgeon. Contraindications include bilateral carotid bifurcations above the level of the mandible, carotid atherosclerosis >50% or ulcerative plaques, and a known allergy to silicone or titanium. Additional contraindications include baroreflex failure or autonomic neuropathy, uncontrolled symptomatic cardiac bradyarrhythmia, and an active indication for cardiac resynchronization therapy.[11]

The vascular surgeons discuss the findings with the cardiology and anesthesia team to determine whether and when antiplatelet or anticoagulation medications can be

stopped, whether and when certain antihypertensive drugs can be stopped, and whether the patient is able to undergo general anesthesia.

Anesthesia considerations. The underlying principle of anesthesia during BAT implantation is judicious monitoring of hemodynamic parameters throughout surgery, such that the intraoperative baroreflex response is not obfuscated. General anesthesia with an arterial line is standard. A superficial block or conscious sedation regimen can blunt the baroreflex and are not performed. During baroreceptor mapping, temporary blood pressure and heart rate fluctuations and electrocardiogram artifacts occur. Preoperatively, brief withholding of antihypertensive agents can help preserve an accurate hemodynamic response. Likewise, adjustments to pacemakers could decrease transient bradycardia.

Intraoperatively, the target systolic blood pressure (SBP) is >90% of conscious values and the target heart rate (HR) is >65 beats per minute. Anesthesia should be administered smoothly, avoiding sudden, dramatic hemodynamic shifts. The use of common agents such as propofol, ketamine, dexmedetomidine, and inhaled anesthetics are discouraged.

Surgical steps. The key steps for BAT implantation include carotid artery exposure, creation of the chest wall pocket, BAT mapping, affixing the electrode to the carotid bulb, tunneling of the CSL, securing the IPG, and closing the incisions.[12] Ultrasound is always used to guide the incision location and orientation. First, minimal dissection of periadventitial tissues is performed, exposing only the anterior surface of the carotid sinus. The chest wall pocket is then created in a subcutaneous plane inferior to the clavicle, either ipsilaterally or contralaterally to the carotid electrode. Next, the CSL is plugged into the IPG, and intraoperative BAT mapping is performed, in conjunction with the anesthesia team, which maintains stable and responsive hemodynamics, and the device representative who operates the external programmer. The electrode is sequentially placed on different locations throughout the carotid sinus until the desired hemodynamic response is elicited. The goal is to obtain a 5% to 10% reduction of the SBP and HR from baseline. Of all patients, 90% will respond at the most common location, the anterior aspect of the internal carotid artery, adjacent to the bifurcation.[12]

The electrode is sewn to the identified target, ensuring full contact and conformance to the carotid bulb. The CSL is detached from the IPG and tunneled from the neck incision to the chest wall pocket, avoiding tension or traction on the electrode. Within the neck, a short length of CSL is looped to create redundancy, allowing for slack on the CSL when the patient's neck is rotated. A small tab attached to the CSL is sewn onto the common carotid artery adventitia, preserving the strain relief loop.

The CSL is reconnected to the IPG, and, together, they are positioned in the pocket with the remaining lead neatly coiled and placed medially. The IPG is fixed to the pectoralis major fascia with retention sutures. Lead mispositioning can cause significant discomfort; thus, excess lead must not be bent, kinked, or lie directly in front of or behind the IPG. Hemostasis is achieved, local anesthetic is applied, and the incisions are closed.[11]

Follow-up. Patients can be discharged home the same day and are seen in the vascular surgery clinic within 2 weeks for a wound check. If reassuring, the protocol allows for a return to vascular surgery care as the need arises. Patients are instructed on the symptoms and signs of device-related complications that might require vascular surgery reevaluation. Moreover, heart failure cardiologists are experienced with monitoring for these symptoms and signs and will refer the patient to vascular surgery care for device-related complications or IPG replacement, which has an average battery life of 5 years.

The patient then attends one BAT titration appointment per month at which BAT is incrementally increased until a maximally tolerated level is reached. Excess BAT manifests as problematic hemodynamics (ie, SBP <90 mm Hg, diastolic blood pressure <50 mm Hg, HR <50 beats per minutes) or feelings of extraneous stimulations from the BAT device.

Outcomes. Patient demographics, characteristics, medical history, and medications and perioperative data, including operative time, estimated blood loss, and length of hospital admissions, were obtained. The primary outcomes were BAT-related morbidities, including cranial nerve injuries, wound complications requiring intervention, device-related infections, extraneous stimulations (ie, symptoms such as jaw pain, abnormal sensations attributed to device activity), and postprocedural major adverse neurologic and cardiovascular (MANCE) events such as cerebrovascular accidents and acute coronary syndromes (ACSs). The secondary outcomes were cardiovascular-related mortality and selected markers of heart failure disease burden, including NT-proBNP levels, left ventricular ejection fraction on transthoracic echocardiography, New York Heart Association (NYHA) class, number of GDMT agents, and frequency of heart failure hospitalizations. Additional qualitative analysis was provided regarding the clinical course of notable patients.

Statistical analysis. The patients' medical records were reviewed, and descriptive statistical analysis was performed. Frequencies and percentages are used to

Table I. Patient demographics, characteristics, and medical history

Characteristic	Median (range) or No. (%)
Age, years	60.5 (44-73)
Body mass index, kg/m^2	32.6 (24.9-41.1)
Male sex	7 (63.6)
Race	
Non-White	8 (72.7)
Non-Hispanic	7 (63.6)
Comorbidity	
Nonischemic cardiomyopathy	7 (63.6)
Atrial fibrillation	8 (72.7)
Carotid stenosis	0 (0.00)
Cerebrovascular accident	1 (9.09)
Coronary artery disease	5 (45.5)
Diabetes mellitus	4 (35.4)
Chronic kidney disease	3 (27.3)
Peripheral artery disease	1 (9.09)
Hostile neck anatomy	1 (9.09)
Preoperative antiplatelet therapy	6 (54.5)
Preoperative anticoagulant therapy	8 (72.7)
Existing cardiac implantable electronic device	11 (100)

present categorical data. Continuous variables are included as the median and minimum to maximum range.

The patients were stratified into two groups: 6 months before BAT and 6 months after BAT. Select secondary outcomes were compared using the Wilcoxon signed rank sum test. Statistical significance was determined using a two-tailed test with $\alpha < .05$. Statistical analysis was performed using Stata SE, version 16.1 (StataCorp).

RESULTS

The median age of the patients was 60.5 years (range, 44-73 years). Of the 11 patients, 3 were White (27.2%), 4 were African American (36.4%), and 4 were Hispanic (36.4%). Most of the patients were men (63.6%). The etiology of heart failure was predominantly nonischemic (72.7%) and included peripartum (18.2%), familial (18.2%), oncologic therapy induced (9.1%), idiopathic (9.1%), and postinfectious (9.1%). All 11 patients had a long-standing diagnosis of advanced heart failure based on existing criteria.[9,13]

One patient (9.1%) had a history of a cerebrovascular accident. No patient (0.00%) had carotid artery atherosclerosis or high-risk carotid artery features based on ultrasound, although one patient (9.1%) had hostile neck anatomy due to previous radiation therapy. The rate at which other cardiovascular risk factors were present varied. Eight patients (72.7%) received long-term anticoagulation therapy, and six (54.5%) were taking at least one antiplatelet medication. All 11 patients (100%) had an existing CIED (Table I). Also, all 11 patients (100%) referred to vascular surgery for technical evaluation of BAT implantation were approved.

Four patients (36.4%) required preoperative hospital admission for medical optimization of acute heart failure exacerbation. The median length of hospital admission before surgery was 0 days (range, 0-7 days). Most received only gentle diuresis. However, one patient (9.1%) with multiple previous revascularizations for severe coronary artery disease was admitted for 7 days due to chest pain and persistent tachycardia, necessitating evaluation to rule out an ACS. The patient improved with intravenous diuretics and resumption of home medications and was deemed fit for same-admission surgery by the advanced heart failure team.

One patient (9.1%) developed an unstable tachyarrhythmia on anesthesia induction, requiring external defibrillation. He converted to normal sinus rhythm, remained hemodynamically stable, was evaluated intraoperatively by the advanced heart failure team, and was deemed fit for surgery. BAT implantation proceeded uneventfully. All 11 patients (100%) had their IPG placed ipsilateral to the carotid electrode. One patient (9.1%) with prior neck radiation received left-sided device implantation. The BAT device was implanted on the same side as an existing CIED in 2 patients (18.1%). One patient (9.1%) underwent concurrent ipsilateral chemotherapy port explanation. All 11 patients (100%) underwent successful baroreflex activation, meeting the goals for an intraoperative decrease in the SBP and HR. The median length of surgery was 82 minutes (range, 58-113 minutes).

Six patients (54.5%) required hospitalization after surgery, with a median length of hospital stay postoperatively of 1 day (range, 0-6 days). The reason for admission included hypotension, nausea, pain, and heart failure optimization. In addition to medical treatment, the patients were provided heart failure education and resources to address barriers to self-care. One patient (9.1%) was evaluated by psychiatrists, prescribed antidepressants, and provided outpatient mental health resources. Nonetheless, most of the admitted patients had minimal symptoms that promptly resolved the morning following surgery and likely would have tolerated same-day discharge (Table II).

The median length of follow-up was 338 days (range, 115-654 days). No cranial nerve injuries, perioperative cerebrovascular accidents, ACSs, wound complications requiring intervention, device-related infections, or device explanations occurred. There were no cardiovascular mortalities at 6 months. Patients were seen for a median of one postoperative visit (range, zero to five) before being discharged from the vascular surgery clinic (Table III).

Ten patients (90.9%) remain closely followed up by the advanced heart failure team for medical management

Table II. Perioperative details

Characteristic	Median (range) or No. (%)
American Society of Anesthesiologists class	4 (3-4)
Surgery time, minutes	82 (58-113)
Left-sided device	1 (9.09)
Ipsilateral implantable pulse generator	11 (100)
Hospital admission, days	
Preoperatively	0 (0-7)
Postoperatively	1 (0-6)
Estimated blood loss, mL	10 (5-25)

Table III. Postoperative outcomes

Characteristic	Median (range) or No. (%)
Follow-up, days	338 (115-654)
Vascular surgery clinic follow-up, No.	1 (0-5)
Cardiovascular-related death at 6 months	0 (0.00)
Cardiovascular-related death	1 (9.09)
Cerebrovascular accident	0 (0.00)
Acute coronary syndrome	0 (0.00)
Cranial nerve injuries	0 (0.00)
Wound complications	0 (0.00)
Device-related infection	0 (0.00)
Device-related reintervention	0 (0.00)
Extraneous stimulation	3 (27.3)

and BAT calibration. Three patients (27.3%) have complained of extraneous stimulations. One patient (9.1%) described feeling a stabbing neck pain ipsilateral to the device, preceded by a vibration sensation occurring at the same time every day. The BAT device was down-titrated, and the pain subsequently resolved. The patient has continued feeling regular vibration sensations, although the symptoms are tolerable. Another patient (9.1%) complained of stuttering speech, jaw pain, and chewing difficulties that started soon after BAT up-titration. The following visit, the BAT was down-titrated to a previously tolerated level, with temporary improvement. Shortly afterward, the side effects returned, requiring another down-titration. A third patient (9.1%) reported pronounced neck pressure, feelings of strangulation, globus sensation, and voice changes that worsened at nighttime when sleeping on the same side as the device. Accordingly, the BAT device was turned off, resolving the symptoms. A trial of device reactivation was intolerable. Described as life limiting, the patient is now being considered for a revision or explantation.

Five markers of heart failure disease burden were compared 6 months before BAT and 6 months after BAT surgery. No significant differences were found in the number of heart failure hospitalizations, left ventricular ejection fraction levels, NYHA class, or number of GDMT agents (Table IV). Nonetheless, most patients either had improved or unchanged results (Fig 2). In contrast, the NT-proBNP levels were significantly higher 6 months after BAT ($P < .05$). Nine patients (81.8%) had an increase of ≥10%, and only one patient (9.1%) had improvement (from 1308 pg/mL to 572 pg/mL).

One patient (9.1%) died of cardiovascular-related reasons at 438 days. Despite multiple attempts to optimize therapy, the patient continued to experience acute decompensated heart failure, requiring nine prolonged hospitalizations after his surgery. During his ultimate episode, he presented with abdominal pain and shortness of breath, with imaging findings suggesting pulmonary metastasis of unknown origin. Hospice care was pursed, and, soon afterward, the patient died.

DISCUSSION

The major findings from this study are as follows:

1. BAT implantation was technically successful in our patients.
2. The incidence of major postoperative complications in our patients was low.
3) More data are needed to understand whether BAT is beneficial for advanced HFrEF.

To date, only one other study focusing on the surgical perspectives and technical aspects of modern BAT in the United States has been previously published. In 2016, using data from the pivotal HOPE4HF (hope for heart failure) trial, Weaver et al[12] noted a 97.2% MANCE-free rate. The system- and procedure-related complication-free rate was 85.9% with no cranial nerve injuries in all 71 patients receiving BAT implantation. After the postoperative period, only one patient (1.8%) complained of persistent numbness in the area of the neck incision, which was attributed to transection of the transverse cervical skin nerve.[8,12]

Similarly, in the BeAT-HF (baroreflex activation therapy for heart failure) study, the other pivotal BAT trial, the MANCE-free rate was 97%, the system- or procedure-related complication-free rate was 94%, and no cranial nerve injuries occurred. Two patients (1.6%) developed a device-related infection requiring explantation, one case of voice hoarseness (0.8%) occurred, and one case of nerve damage/stimulation (0.8%) was reported, although the extent of the symptoms is unclear.[7]

Our institution's experience matches these results, with a 100% MANCE-free rate, no cranial nerve injuries, and no device-related reinterventions within 6 months. However, our series differed in that three patients (27.3%) reported intolerable symptoms attributed to extraneous stimulation of nearby tissues by the device, requiring BAT down-titration in all three cases. It is possible that

Table IV. Markers of heart failure disease burden before and after baroreceptor activation therapy (BAT) surgery

Marker	6 Months before BAT	6 Months after BAT	*P* value
Heart failure hospitalization, No.	1 (0-3)	1 (0-9)	>.05
LVEF, %	20-25 (10-15 to 55-60)	20-25 (10-15 to 55-60)	>.05
NYHA class	3 (2-4)	2 (1-4)	>.05
GDMT drugs, No.	4 (2-4)	3 (2-3)	>.05
NT-proBNP, pg/mL	827 (35-14,697)	1589 (42-31,846)	<.05

GDMT, Goal-directed medical therapy; *LVEF*, left ventricular ejection fraction; *NT-proBNP*, N-terminal pro-brain natriuretic peptide; *NYHA*, New York Heart Association.

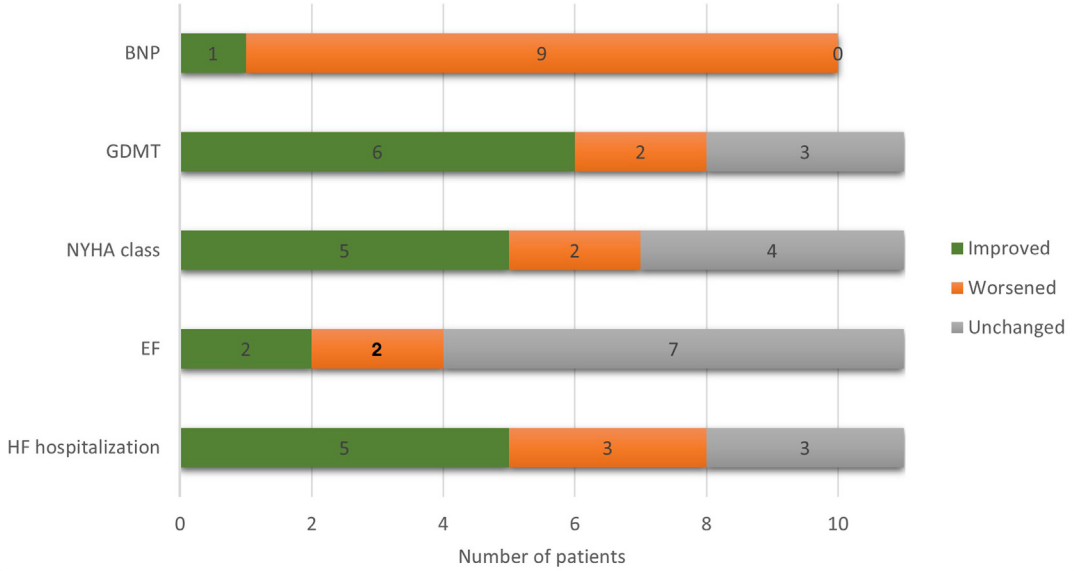

Fig 2. Patient frequency and type of change in select markers of heart failure disease burden from 6 months before to 6 months after baroreceptor activation therapy (BAT) surgery. *BNP*, Brain natriuretic peptide; *EF*, ejection fraction; *GDMT*, goal-directed medical therapy; *HF*, heart failure; *NYHA*, New York Heart Association.

extraneous stimulation from the BAT device could be an underreported, but clinically relevant, entity. Described by the manufacturer as a known complication that can lead to significant symptoms,[11] few studies have analyzed the long-term effects of extraneous stimulation on BAT tolerability. In a single-center retrospective study, Heusser et al[14] reported that 12 patients (66.7%) experienced side effects from extraneous stimulation significant enough that the stimulation intensity had to be reduced. Consequently, lowering of stimulation intensity to chronically tolerable levels was associated with a reduction in the acute efficacy of the BAT.[14] It is hypothesized that these abnormal symptoms occur due to stray currents eliciting off-target electric stimulation as the currents travel from the IPG through the CSL to the electrode. It has been identified as efficacy limiting in other neurostimulation technologies and could be an area of improvement for future device iterations.[15] Surgeon modifiable factors to reduce extraneous stimulations include limiting tissue dissection, mindful tunneling, and proper positioning of each BAT component.[14]

The average time of surgery in our series was comparable to the results from the HOPE4HF trial reported Weaver et al,[12] in which the mean procedure time was 99 ± 35 minutes, of which mapping comprised 36 ± 24 minutes. Notably, the manufacturer is considering simplifying implantation such that intraoperative mapping is not required, with all electrodes placed in the anteromedial position by default. Notwithstanding, at the time of our report, the official instructions for use have not yet been revised.

The efficacy of BAT has been demonstrated by several previous studies, with the two most important studies the HOPE4HF and BeAT-HF trials. These studies demonstrate that BAT improves exercise capacity, NYHA class, NT-proBNP levels, and quality of life for patients with HFrEF refractory to maximally tolerated GDMT 6 months after surgery.[16,17] Long-term data are still lacking. The

present study was not designed to evaluate the efficacy of BAT on improving HFrEF, because it is limited by the small sample size and lack of matched controls. Nonetheless, in our patients, no significant differences were found in several markers of HFrEF disease burden when comparing values 6 months before BAT implantation to 6 months afterward. Moreover, in most studies, receiving BAT led to reduced NT-proBNP levels.[5] However, in our series, there was a significant increase in NT-proBNP levels by ≥10% in most of our patients after BAT, even in the context of improvement in other markers of HFrEF disease burden. It is likely that there are multiple unaccounted confounding factors causing a wide variation in the response to BAT.

The limitations to this study are due to its small and heterogeneous sample. Meaningful extrapolations cannot be drawn from these data. The series could be underpowered to detect low frequency, but clinically significant, procedure- and device-related complications. This study only serves as hypothesis generating and cannot definitively demonstrate that BAT implantation is technically achievable and associated with minimal complications. Long-term data and prospective, randomized, controlled, and blinded studies are required to truly prove that BAT is an efficacious and safe therapy for patients with HFrEF refractory to maximally tolerated GDMT. However, much of the literature describing BAT is from Europe, where the technology has been in use for a longer period and there are a wider range of clinical applications, including resistant hypertension.[18,19] Data germane to the U.S. population is still being collected but could be eclipsed by the rate at which new technologies are being developed, such as endovascular options, which are on the horizon.[20] These novel data represent the real-world experience of the most up-to-date BAT technology from the perspective of vascular surgeons treating patients with HFrEF in the United States.

CONCLUSIONS

In this single-institution retrospective review, BAT implantation for patients with HFrEF refractory to maximally tolerated GDMT was safe in the short term, with few complications. Vascular surgeons play an important role in the multidisciplinary delivery of this novel device-based therapy. More data are needed to understand whether BAT is beneficial in the treatment of HFrEF refractory to maximally tolerated GDMT.

DISCLOSURES

L.G. is a consultant for CVRx and receives financial compensation from the company.

REFERENCES

1. Zannad F. Neuromodulation in heart failure. *Eur Heart J Suppl*. 2022;24(Suppl E):E1–E3.
2. Gronda E, Dusi V, D'Elia E, Iacoviello M, Benvenuto E, Vanoli E. Sympathetic activation in heart failure. *Eur Heart J Suppl*. 2022;24:E4–E11.
3. Dusi V, Angelini F, Zile MR, De Ferrari GM. Neuromodulation devices for heart failure. *Eur Heart J Suppl*. 2022;24:E12–E27.
4. Babar N, Giedrimiene D. Updates on baroreflex activation therapy and vagus nerve stimulation for treatment of heart failure with reduced ejection fraction. *Cardiol Res*. 2022;13:11–17.
5. Fudim M, Abraham WT, Von Bardeleben RS, et al. Device therapy in chronic heart failure. *J Am Coll Cardiol*. 2021;78:931–956.
6. Baroreflex Activation Therapy for Heart Failure (BeAT-HF). ClinicalTrials.gov. National Library of Medicine (US); 2019. Report No.: NCT02627196.
7. Zile MR, Lindenfeld J, Weaver FA, et al. Baroreflex activation therapy in patients with heart failure with reduced ejection fraction. *J Am Coll Cardiol*. 2020;76:1–13.
8. Abraham WT, Zile MR, Weaver FA, et al. Baroreflex activation therapy for the treatment of heart failure with a reduced ejection fraction. *JACC Heart Fail*. 2015;3:487–496.
9. Heidenreich PA, Bozkurt B, Aguilar D, et al. 2022 AHA/ACC/HFSA guideline for the management of heart failure: a report of the American College of Cardiology/American Heart Association Joint Committee on clinical practice guidelines. *Circulation*. 2022;145:e955–e957.
10. Cook JL, Colvin M, Francis GS, et al. Recommendations for the use of mechanical circulatory support: ambulatory and community patient care: a scientific statement from the American Heart Association. *Circulation*. 2017;135:e1145–e1158.
11. 900133-001 Rev. B IFU System Overview. Barostim System Instructions for Use: System Overview [Internet]. CVRx, Inc; 2022. Accessed December 1, 2023. https://www.cvrx.com/wp-content/uploads/2023/03/900133-001B_IFU_System-Overview_UNIPOLAR-2104_2102-IPG-103X-CSL-9010-9020-PGM_US_Commercial.pdf.
12. Weaver FA, Abraham WT, Little WC, et al. Surgical experience and long-term results of baroreflex activation therapy for heart failure with reduced ejection fraction. *Semin Thorac Cardiovasc Surg*. 2016;28:320–328.
13. Crespo-Leiro MG, Metra M, Lund LH, et al. Advanced heart failure: a position statement of the heart failure association of the European society of cardiology. *European J of Heart Fail*. 2018;20:1505–1535.
14. Heusser K, Tank J, Brinkmann J, et al. Acute response to unilateral unipolar electrical carotid sinus stimulation in patients with resistant arterial hypertension. *Hypertension*. 2016;67:585–591.
15. Thompson N, Mastitskaya S, Holder D. Avoiding off-target effects in electrical stimulation of the cervical vagus nerve: neuroanatomical tracing techniques to study fascicular anatomy of the vagus nerve. *J Neurosci Methods*. 2019;325:108325.
16. Molina-Linde JM, Cordero-Pereda D, Baños-Álvarez E, Rosario-Lozano MP, Blasco-Amaro JA. Efficacy and safety of baroreflex activation therapy for heart failure with reduced ejection fraction: systematic review. *ESC Heart Failure*. 2023;10:2760–2772.
17. Coats AJS, Abraham WT, Zile MR, et al. Baroreflex activation therapy with the Barostim™ device in patients with heart failure with reduced ejection fraction: a patient level meta-analysis of randomized controlled trials. *Eur J Heart Fail*. 2022;24:1665–1673.
18. Guckel D, Eitz T, El Hamriti M, et al. Baroreflex activation therapy in advanced heart failure therapy: insights from a real-world scenario. *ESC Heart Failure*. 2023;10:284–294.
19. Bisognano JD, Bakris G, Nadim MK, et al. Baroreflex activation therapy lowers blood pressure in patients with resistant hypertension. *J Am Coll Cardiol*. 2011;58:765–773.
20. Van Kleef MEAM, Devireddy CM, Van Der Heyden J, et al. Treatment of resistant hypertension with endovascular baroreflex amplification. *JACC Cardiovasc Interv*. 2022;15:321–332.

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