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Advancing Heart Failure Treatment With Barostim: An Electrophysiologist's Real-World Success Story

Dr. Verma:
Hi. My name is Dr. Sumit Verma. I'm a cardiac electrophysiologist at Baptist Healthcare in Pensacola, Florida. Today, I will share my experience with the Barostim device and discuss best practices to ensure success with the device.

The Barostim implant is FDA-approved for improvement in symptoms in patients with NYHA Class II and Class III heart failure, who have an ejection fraction of less than or equal to 35%, despite optimal GDMT and an NT-proBNP of less than 1600. The main guiding factor is to discuss their quality of life, and if they have a poor quality of life, limited exercise tolerance, etc., then I think those are the best candidates for this intervention.

The device works by stimulating the carotid baroreceptors. In heart failure, the baroreceptors have reduced signaling, which leads to reduced parasympathetic tone, increased sympathetic tone, and activation of the renin angiotensin system. We have found that direct high-frequency stimulation of these carotid receptors helps rebalance the autonomic nervous system.

In the BEAT-HF trial, patients were randomized to guideline-directed medical therapy alone or GDMT plus Barostim implantation. In the device arm, patients had improvement in the 6-minute walk test by about 60 meters and improvement in quality of life scores. The data signaled toward reduction in hard endpoints such as LVAD transplant and death but was not statistically significant in those particular endpoints.

As you may know, the device is implanted surgically by a cardiac or vascular surgeon or someone familiar with a carotid dissection. However, selection and referral for the implant is usually from a cardiologist or heart failure specialist.

I feel that electrophysiologists have a very important role in the selection of this device, and I will explain why. There are several reasons for this. Number one, often EPs are the first point of contact to discuss device therapy for heart failure patients, whether it is an ICD, a CRTD, or conduction system pacing device. We can anticipate potential device-device interactions, as may occur with implantation of subcutaneous ICDs or extravascular ICDs with Barostim, and possibly avoid those devices if we think Barostim may be required later.

Selecting the correct ICD and its location in the chest is important for planning. Let's call it managing the real estate. In a hypothetical example, imagine a patient with a prior right carotid endarterectomy, consideration may be given to implanting an ICD on the right side and saving the left side for Barostim since the left carotid has not been intervened upon.

About 70% of heart failure patients are not candidates for CRTD due to lack of a left bundle branch block. And in these patients, I consider offering Barostim prior to or after ICD therapy. Even CRTD nonresponders can potentially benefit from Barostim implantation.

Recommending a primary prevention ICD alone is not helpful in improving symptoms. It is not unusual for me to recommend 2 devices —a primary prevention ICD and Barostim and depending upon the severity of the symptoms. The acceptance of this dual-device strategy has been very high amongst patients, with only a few declining.

Next, the EP device clinic is well suited to monitor and track the Barostim device. We can enroll and follow these patients in our device clinic where they are scheduled for titrations and monitoring of their battery status. And as an electrophysiologist, it is fascinating to see the improved control of AFib, PVCs, and other ventricular arrhythmias such as ventricular tachycardia and ventricular fibrillation even, in some cases. Although improved arrhythmia control with Barostim is anecdotal at this point.

At our center, we have studied the changes in the LV ejection fraction and found this statistically significant increase in the EF in about 25 patients, both ischemic and non-ischemic, with an improvement from a baseline of 22% to 33%, with a statistically significant *P* value

of 0.004. This data is consistent with studies from other centers as well. Although the series is small of about 25 patients, the initial results are very encouraging in regards to LVEF improvement. And finally, data from other different health systems suggests a very remarkable 80% decrease in heart failure hospitalizations.

Before I end, I'd like to discuss 2 cases. The first patient is a 78-year-old male with non-ischemic cardiomyopathy. As is common in advanced heart failure patients, he underwent several arrhythmia-related interventions, such as ICD generator change, SVT ablation, a CRTD and an AV node ablation, and eventually two complex PVC ablations, including an alcohol ablation of the septum during which he suffered a pulseless electrical activity arrest and a subsequently prolonged recovery.

Plotted in this graph is the gradual drop in the ejection fraction from a baseline of 35% to about 14%, despite multiple interventions. So this is by no means a typical Barostim patient, as we would usually like to intervene at an earlier stage. Nevertheless, he did undergo Barostim implantation and had improvement in the ejection fraction to back above 30, and he maintains it 6 months out.

The timing is highly suggestive of a beneficial effect of Barostim. This is an interesting example of how this intervention can reverse the downward trend in heart failure patients.

The second case is a 58-year-old female with non-ischemic cardiomyopathy who was referred for primary prevention AICD implantation. The patient had severe symptoms with NYHA Class III, and she was more interested in therapies that would help improve her quality of life, and so I referred her for Barostim implantation first.

Remarkably, after 3 months, her LVEF also improved to 50%, and her symptoms improved dramatically, and now she is being followed in our clinic without ICD implantation, as she no longer meets the criteria for primary prevention ICD.

Above all, the most rewarding part of this experience has been the follow-ups where patients report that the majority of their symptoms related to heart failure, such as fatigue, poor exercise tolerance, dyspnea, anxiety, depression, and brain fog, etc., are improved and they are back to feeling normal and enjoying their hobbies and life in general with their families.

I hope you will find this information useful in the management of heart failure patients in your practice. Thank you for your attention.