A 72-year-old man with a history of rheumatic heart disease, prior bioprosthetic mitral and aortic valve replacement, severe nonischemic dilated cardiomyopathy (left ventricular [LV] ejection fraction 5%), and a history of ventricular tachycardia (VT) underwent implantation of a continuous flow LV assist device (LVAD; HeartMate II; Thoratec Corporation) as destination therapy for severe heart failure. The early postoperative period was unremarkable and he had substantial functional improvement with minimal cardiovascular limiting symptoms after postoperative recovery.

After ≈3 years on LVAD support, he experienced a series of appropriate implantable cardioverter-defibrillator shocks for sustained VT refractory to antiarrhythmic therapy. He underwent VT ablation targeting the basal LV inferoseptum, but he continued to have recurrent VT. Because VT seemed to originate from a deep septal substrate, he underwent an alcohol septal ablation that was also unsuccessful. A week later, VT was terminated during a second endocardial ablation and was noninducible thereafter. He has had no further sustained VT or implantable cardioverter-defibrillator shocks for 10 months of follow-up.

Approximately 4 years after the LVAD implantation, and 6 months after the last VT ablation, he presented with new symptoms of intermittent dizziness, near syncope, and exertional dyspnea. Transesophageal echocardiography demonstrated a relatively improved LV ejection fraction of 25% with septal asymmetry and dynamic obstruction of the LVAD inflow with increased inflow velocities during each native heart systole. He was admitted and underwent repeat contrast echocardiograms to evaluate the obstruction further in varying positions and LVAD RPM settings (Figure; Movies 1–3 in the Data Supplement). A peak systolic velocity of 3.49 m/s was obtained with the patient upright at 9400 rpm. There was no clinical or laboratory evidence of pump thrombosis, including no changes in VAD parameters of power or pulsatility. The plasma lactate dehydrogenase concentration was 325 U/L (normal range 105–333) and the free hemoglobin level was 4.5 mg/dL (normal range 14–17). The material obstructing the LVAD inflow did not have thrombus-like quality on echocardiographic imaging.

Surgical interventions, including repositioning the inflow cannula with or without pump exchange and apical LV myomectomy, were considered but felt to carry excessively high risk. Because the LVAD inflow obstruction occurred mainly during native heart systole, we hypothesized that the physiology was similar to dynamic LV outflow obstruction as observed in hypertrophic obstructive cardiomyopathy. Moderate hydration and adjustment of the LVAD rotor speed (RPM decrease from 9400 to 8800) to increase LV volumes and reduce inflow obstruction yielded limited benefit. However, subsequent addition of β-blockers (Metoprolol Succinate ER 50 mg BID) resolved his dizziness and dyspnea. The supine peak systolic inflow velocity was 2.01 m/s. He has now been free from limiting symptoms for 5 months.

The mechanism of the dynamic inflow obstruction >3 years after the LVAD implantation is not certain. However, we speculate that LVAD-induced LV remodeling and improved systolic function along with VT ablation-induced changes contributed to septal geometry changes that favored dynamic obstruction of LVAD inflow. Regardless of the cause, this is the first reported case of dynamic LVAD inflow obstruction, with clinical and hemodynamic characteristics similar to hypertrophic obstructive cardiomyopathy. We found that conservative management with interventions to assure adequate LV volume and limit hyperdynamic regional contraction may allow relief of dynamic LVAD obstruction and associated symptoms just as with hypertrophic obstructive cardiomyopathy.
Figure. A. Continuous wave Doppler of the left ventricular assist device (LVAD) inflow cannula imaged from a modified apical view. The patient was supine with Heartmate II LVAD set at 9400 rpm. Doppler profile demonstrates continuous inflow velocity of 0.6 m/s (arrow) and pulsatile peak velocities during late systole reaching 2.32 m/s (arrow). B. The patient moved to an upright position with the LVAD set at 9400 rpm. The continuous wave Doppler profile revealed an increase in late systolic pulsatile velocity to 3.49 m/s.
Dynamic Left Ventricular Assist Device Inflow Obstruction
Edo Y. Birati, James N. Kirkpatrick, J. Eduardo Rame, Rupa Bala and Kenneth B. Margulies

*Circ Heart Fail.* 2014;7:225-226
doi: 10.1161/CIRCHEARTFAILURE.113.000941
*Circulation: Heart Failure* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1941-3289. Online ISSN: 1941-3297

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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