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Hemodynamic Pump-Patient Interactions and Left Ventricular Assist Device Imaging

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INTRODUCTION

Continuous-flow left ventricular assist devices (cfLVADs) have become durable options for patients with advanced heart failure as a mode to improve both quality of life and survival, either as destination therapy or bridge to transplantation.^{1,2} A patient's optimal level of hemodynamic support from LVADs may vary based on several contributing factors, such as gender, body size, volume status, presence of existing right ventricular dysfunction, and parameters determined by the clinician. Hemodynamic optimization of patients with an LVAD includes implementation of guideline-directed medical therapy with the use of diuretics, beta-blockers, angiotensin-receptor blockers/angiotensin-converting enzyme/neprilysin inhibitors, and mineralocorticoid antagonists; furthermore, the International Society of Heart and Lung Transplantation recommends that cfLVAD speeds should be adjusted to adequately unload the left ventricle, while maintaining a midline intraventricular septum along with minimizing mitral valve regurgitation (MR) plus allowing for intermittent aortic valve (AV) opening.³ The use of echocardiography and invasive hemodynamics is imperative, and complementary clinical tools to improve the hemodynamic profile of a patient with an LVAD. This review describes the following topics: (1) normal hemodynamic characteristics of durable mechanical support, (2) methods to optimize LVAD speed, and (3) morphologic changes of both the left and right ventricle following LVAD implantation.

HEMODYNAMICS OF LEFT VENTRICULAR ASSIST DEVICES

Contemporary LVADs are classified as either axial pumps, such as the HeartMate II (Abbott, Abbott Park, IL) or centrifugal pumps, which consist of the HVAD (Medtronic, Minneapolis, Minnesota) and HeartMate 3 (Abbott, Abbott Park, IL). These devices are implanted with different techniques and have variable flow capacities, but are governed by the same principle of continuous pumping of blood out of the left ventricle (LV) throughout

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the cardiac cycle. Pressure-volume loops (PVLs) best demonstrate the physiologic relationship between ventricular filling and contraction in respect to external changes in loading conditions. Under normal conditions, the PVL is characterized by a trapezoidal shape with a rounded top. PVL appearance in the setting of LVAD transforms from a trapezoidal shape to a triangular shape, which is reflective of the loss of normal isovolemic periods, where systemic blood flow is not dependent on ejection through the AV⁴ (Fig. 1). Stepwise increases in LVAD speed will coincide with similar increases in flow under static loading conditions and will cause a progressive leftward shift of the PVL, depicting increasing degrees of LV unloading. Additionally, the higher the achieved LVAD flow correlates with decreasing peak LV pressure generation and higher systemic arterial pressures, with the subsequent ventricular unloading leading to decreased pulmonary capillary wedge and left atrial pressures.

The relationships of blood flow (Q) through a cLVAD and pressure differential (H) between the inflow and outflow cannulas (pump pressure) can best be described by the HQ curve (Fig. 2). The HQ curve appearance in axial versus centrifugal pump varies and ultimately reflects the differing engineered characteristics of the pump and therefore physiologic response to speed changes. At a given pump speed, pump flow decreases as the pressure gradient across the inflow and outflow cannula increases. At a given pressure gradient, pump flow increases as the pump speed increases. However, intrinsic properties of the LV and the aorta in regards to pressure differential during systole and diastole will additionally impact the degree of flow changes in response to increases in pump speed. Centrifugal pumps, specifically the HVAD and HeartMate 3, operate over a wide range of flows for a very small change in pressure differential across the pump (see Fig. 2A).^{5,6} The flat head curve for centrifugal pump is reflective of the high pump flow pulsatility inherent to the device. This results in a large dynamic range of flow of up to approximately 10 L/min for a relatively small change in pressure. From a clinical standpoint, the flat HQ curve translates to little change in the pressure differential in response to low flows, which can occur commonly in hypovolemia or even right heart failure. Therefore, relatively static pressure differentials translate to less susceptibility to increased suction during low flow conditions. In contrast, axial flow pumps (HeartMate II) have much steeper HQ curves, in which there is linear inverse relationship in flow with pump pressure differential (see Fig. 2B). Consequently, similar pressure differentials across the pump will produce less flow pulsatility (3–7 L/min for example) compared with the centrifugal pump design. Furthermore, the axial LVADs are more likely to generate a greater pressure differential in the setting of low flows, which may in turn trigger suction events within the LV cavity. The combined intrinsic properties of differential pump pulsatility demonstrated on the HQ curves along with varying ventricular and aortic loading conditions govern the complex pump-patient interactions.

LEFT VENTRICULAR ASSIST DEVICE SPEED OPTIMIZATION

A unique pump-patient interaction is present for all subtypes of patients who receive durable mechanical circulatory support. This is due to the several contributing factors that ultimately affect LVAD output, which include patient body size, gender, mean arterial pressure, volume status, intrinsic myocardial contractility, pre-LVAD implantation right ventricular (RV) function, vasoactive medications, and the presence or absence of neuro-hormonal blockade.

Other than these factors, the clinician has the ability to alter the performance of LVAD by changing the device speed, with the goal of optimizing hemodynamics. Device speed optimization, defined as the speed in which the LV is adequately unloaded with a midline intraventricular septum with minimal MR and intermittent AV opening, can vary dramatically from patient to patient.⁷

Following LVAD implantation in the immediate postoperative period, noninvasive imaging is routinely deferred until perioperative inotropes and vasoactive drugs are weaned off to best assess the need for pump speed optimization. LVAD speed optimization, known as ramp testing, can be achieved through the use of echocardiography and invasive hemodynamics.

TWO-DIMENSIONAL AND 3-DIMENSIONAL ECHOCARDIOGRAPHIC OPTIMIZATION

Our group was the first to describe a formalized protocol for LVAD speed testing for the purpose of speed optimization and diagnosing device thrombosis.³ In brief, before performing a ramp test, appropriate anticoagulation must be confirmed with international normalized ratio greater than 1.8 or partial thromboplastin time greater than 60 seconds. Opening arterial pressure by Doppler during the study should be >65 mm Hg at baseline to proceed. The parasternal long-axis view is then primarily used to assess LV end-diastolic dimension (LVEDD), LV end-systolic dimension (LVESD), frequency of AV opening, degree of aortic insufficiency (AI), MR, and heart rate. Continuous-flow LVAD parameters, including power, PI (pulsatility index), and flow, are recorded at each stage. Baseline evaluation of these variables are also recorded at the given speed at presentation. For the HeartMate II devices, the device speed is then lowered to 8000 rpm and 2300 rpm for the HVAD devices. After 2 minutes of washout time, LVEDD, LVESD, degree of MR, AI, assessment of AV opening, Doppler blood pressure, and heart rate are all recorded. Pump parameters (power, PI, and flow) are also recorded during each stage. Stepwise increase in speed at intervals of 400 rpm for Heart-Mate II and 100 rpm for HVAD are then made; other protocols have shown 130-rpm intervals in the HVAD physiologically correlates with 400-rpm intervals in the HeartMate II.⁸ The testing speed range for HeartMate II is 8000 to 12,000 rpm and 2200 to 3200 rpm for the HVAD. The protocol is complete once the upper limit speed is reached, LVEDD reaches less than 3.0 cm, suction event, or ventricular ectopic beats occur. The clinician must also pay attention to development of premature ventricular contractions, which may indicate contact of the inflow cannula with the septum. From a medical optimization standpoint, the speed is adjusted to clinically achieve a midline intraventricular septum, minimal MR, and intermittent AV opening to prevent development of AI.

Ramp testing can be used for device malfunction assessment in general and specifically for pump thrombosis. Patients with obstruction to flow, mainly pump thrombosis, had minimal change in their LVEDD size in response to speed change leading to an attenuated LVEDD slope when calculated by a linear equation (device speed on x-axis vs LVEDD on y-axis).⁹ LVEDD slope of greater than -0.16 was suggestive of device thrombosis when performing echocardiographic ramp testing. Performing a ramp test in patients with HVADs was not

associated with linear LVEDD reduction in response to speed changes as seen with the HeartMate II pumps.^{10,11}

Recently, our group investigated ventricular structural changes in 31 patients (19 with HeartMate II and 12 with HVAD) who underwent hemodynamic ramp studies during which 3D transthoracic echocardiographic (TTE) imaging was performed to determine how device speed changes influence global LV and RV geometry.¹² End-diastolic and systolic volumes using 3D TTE were calculated at each stage during prespecified ramp protocol. In addition, 3D endocardial surface analysis was performed to define LV conicity and sphericity. It has been previously described that adverse remodeling leads to more spherical LV shapes from chronic volume overload in patients with severe mitral regurgitation due to mitral valve prolapse, with postsurgical function improvements occurring as represented by change to more normal, conical shape.¹³ Three-dimensional TTE RV shape analysis was described in the form of free-wall and septal curvature in response to speed changes. Zero curvature indicated a flat surface, whereas the more positive or negative values signify more convexity or concavity of the surface, respectively, from the perspective of a reference point outside of the right ventricle. For the HeartMate II cohort, LV volumes decreased by (127 ± 78 mL; $P < .01$) and became more conical with increasing speeds. RV volumes increased significantly only at highest speeds (stage IV) with an average difference of 60 ± 68 mL between highest and lowest stage ($P < .01$); RV septal shape on average also became more convex (bulging into the LV) at the highest speed setting when compared with the lowest speed setting. For the HVAD cohort, LV volumes similarly decreased when comparing the lowest and highest speeds (51 ± 38 mL, $P < .01$); however, the changes in shape were more global than the longitudinal changes seen with HeartMate II. With regard to the RV, there was a non-statistically significant increase in RV volumes in response to the increase in speed. In addition, there was no change in RV septal or free-wall curvature. Schematics depicting 3D ventricular geometry with respect to pump type are shown in Fig. 3.

It can be postulated that differential changes in 3D TTE-derived ventricular shape may be attributed to pump position. The HeartMate II device is located in the subdiaphragmatic space and results in inferior displacement of the LV apex, whereas the intrathoracic-placed HVAD resides within the LV apex and results in less apical deformation. This is likely the same with the newer HeartMate 3 device, which is located in the intrathoracic position, although studies are lacking in changes in 3D ventricular geometry for this more contemporary pump.

INVASIVE HEMODYNAMIC OPTIMIZATION

An additional aspect to medical optimization of the patient with an LVAD is combining echocardiography and invasive hemodynamics to better improve a patient's hemodynamic profile given the complex interactions between native heart physiology and pump characteristics. We followed the original study of noninvasive ramp testing with a second analysis of LVAD speed adjustment combined with invasive hemodynamics obtained during a right heart catheterization (RHC).¹⁴

Patients with an LVAD were evaluated in the outpatient setting for a simultaneous echocardiographic and hemodynamic ramp test. Baseline central venous pressure (CVP), pulmonary artery pressure, pulmonary capillary wedge pressure (PCWP), Fick cardiac output (CO), and cardiac index are measured. The same steps for speed up-titration are made as previously described, with 2-minute washout periods following speed changes. At each speed interval, right heart pressures, heart rate, Doppler blood pressure, and echocardiographic images are obtained. At the conclusion of the assessment, the clinician chooses the speed that best achieves hemodynamic optimization, defined as a PCWP less than 18 mm Hg and CVP less than 12 mm Hg, with the secondary goals of intermittent AV opening and minimal MR.

In ambulatory outpatients, baseline hemodynamics revealed that only 43% of patients with an LVAD had a CVP and PCWP within the specified normal range.¹⁴ Following the conclusion of invasive hemodynamic ramp testing, 56% of patients achieved normalization of both CVP and PCWP. The hemodynamic profiles of the overall cohort represented by CVP and PCWP are best characterized in Fig. 4. The dashed line in the baseline panel in Fig. 4 equates to a CVP-to-PCWP ratio of 0.63, for which ratios greater than this cut point may suggest right heart failure. There were no significant differences in baseline hemodynamics between pump types. Furthermore, reduction in PCWP was determined to be speed-dependent and flow-dependent instead of device-dependent, and was similar as well between pumps. Small sample regression analysis from other centers following ramp testing demonstrated that speed adjustments of 400 rpm in the HeartMate II were equivalent to a 130-rpm adjustment in the HVAD, corresponding to a relative increase in CO of 0.3 L/min.⁸ In this study, improvement in 6-minute walk distance following hemodynamic ramp optimization was observed, although long-term outcomes and data on other performance indices following ramp studies remains to be seen.

A similar hemodynamic ramp protocol was tested by our group in a contemporary HeartMate 3 cohort.¹⁵ Consistent with our prior findings, speed optimization was able to normalize CVP and PCWP in 50% of patients with abnormal hemodynamics at baseline. All of these hemodynamic studies demonstrate a wide range of baseline hemodynamics with differing responses to speed changes, suggesting a significant benefit in performing speed optimization with the aid of simultaneous invasive hemodynamics.

Given that invasive hemodynamic assessment is not always readily available and inherent procedural risks are present due to chronic anticoagulation, Doppler echocardiographic assessment also may be used when evaluating filling pressures for the LVAD patient. Estep and colleagues¹⁶ performed simultaneous RHC and TTEs on 50 consecutive patients with HeartMate II devices at a baseline speed of 9000 rpm. They derived a multifaceted algorithm based on this cohort incorporating TTE-derived mitral inflow velocities, right atrial and systolic pulmonary pressure estimation, and indexed left atrial volume, which demonstrated considerable diagnostic accuracy in identifying patients with PCWP greater than 15 mm Hg with an area under the curve of 0.89 ($P<.0001$). These data support using TTE in beyond measurement of ventricular dimensions, assessment of MR, and AV closure when optimizing LVAD patients.

Current studies are under way to assess speed optimization on clinical outcome. The HVAD RAMP IT UP trial,¹⁷ which is a randomized study of invasive hemodynamic speed optimization versus standard of care, and other single-center experiences. The American Society of Echocardiography has recently published guidelines on appropriate use of LVAD ramp testing to better ascertain presence of device thrombosis, difficult-to-manage RV failure, or when a patient requires speed optimization in addition to medical therapy titration for signs and symptoms of clinical heart failure.¹⁸

NOVEL ASSESSMENT OF AORTIC INSUFFICIENCY

De novo AI has a prevalence of approximately 25% in the first year following cfLVAD implantation and if left untreated, can lead to progressive heart failure symptoms.^{19,20} The pathophysiologic mechanism is thought to arise from valve commissure and leaflet degradation due to the lack of regular AV opening following cfLVAD implantation.²¹ Furthermore, continuous LV unloading leads to valvular shear stress and an average increase in transvalvular pressure load by 25% and duration of the pressure load by 20% compared with normal circulatory physiology. Progressive AI may also lead to RV dysfunction and whether it reduces survival is unclear.^{22,23}

Given the lack of a true diastolic phase in LVADs, traditional echocardiographic modalities of AI quantification including vena contracta, proximal isovelocity surface area, deceleration time, and regurgitation fraction (RF) may all not be applicable to durable continuous-flow mechanical circulatory support devices. Our group investigated the accuracy of traditional modalities of AI quantification compared with novel methods, including direct calculation of aortic RF plus 2 Doppler-based indices: outflow cannula diastolic acceleration time and outflow cannula systolic-to-diastolic (S/D) peak velocity ratio.²⁴ Simultaneous RHC and TTEs were performed as part of ramp testing for LVAD optimization. Novel aortic RF was calculated by subtracting the venous return to the right side of the heart obtained by RHC via the Fick method from the total left-sided systemic flow measurement using TTE (full derivation in publication by Grinstein and colleagues²⁴). Based on the continuity equation, total left-sided systemic flow is equal to right-sided venous return unless an intracardiac shunt or AI is present. For the 2 additional new methods of AI quantification, a modified right sided parasternal window was used to acquire the pulse wave Doppler signal of the outflow graft of the LVAD cannula, with the diastolic acceleration measured as the slope from the start to end of diastole (Fig. 5A). The S/D ratio was defined by peak systolic velocity divided by peak diastolic velocity of the pulsed wave Doppler signal of the LVAD outflow cannula.

Traditional qualitative RF assessment underestimated the degree of AI when compared with the novel aortic RF, as mean novel aortic RF for qualitatively moderate or greater AI was $45.8\% \pm 3.6\%$, lying within the higher range of RF for moderate AI (Fig. 5B). In addition, novel aortic RF correlated better with PCWP. Significant AI increases left ventricular end-diastolic volume, as was observed by the moderately strong correlation between PCWP and the novel aortic RF. The S/D ratio and outflow cannula diastolic acceleration correlated strongly with regurgitant fraction ($R = 0.91$ and 0.94 , respectively) and PCWP ($R = 0.82$ and 0.65 , respectively) as seen in Fig. 6. Moderate to severe AI as calculated correlated with a

proposed S/D ratio with estimated S/D cut point of less than or equal to 1.1 associated with novel aortic RF of 50%. Similarly, an estimated diastolic slope acceleration cut point of 82 cm/s² was associated with moderate to severe AI by novel aortic RF. These new Doppler indices demonstrated stronger correlation with PCWP compared with vena contracta and can be acquired in most patients with adequate acoustic windows. Furthermore, these novel parameters were shown to be more sensitive in detecting patients at risk for worse outcomes.

Patients with AI have higher PCWP, CVP, and lower pulmonary artery pulsatility index²⁶ than patients without AI. These data further question the mechanism of AI in the LVAD population. Does RV failure lead to reduced flow from the RV to the LV, and as such reduce the frequency of AV opening, leading aortic cusps fusion and AI? Or, does AI causing fluid overload and increased PCWP and RV afterload result in RV failure? The exact mechanism is still unknown and further studies are needed to elucidate the pathophysiology. Timely assessment of AI may be important to identify those patients who will benefit from AV replacement via surgery, transcatheter AV replacement, or AV closure.

SUMMARY

Durable LVAD support is a viable therapy for patients with end-stage heart failure as either a bridge to transplant or bridge to destination therapy. Our understanding of the complex “pump-patient” relationship has grown tremendously since the introduction of these devices more than 20 years ago. The combination of both imaging and invasive hemodynamics is crucial to understand the individual effect of LVADs on specific patient populations. Using those methods already has demonstrated the importance in device troubleshooting and medical optimization of the patient. Further prospective studies are needed to further elucidate the durable long-term effects of these protocols and true utility as part of daily practice.

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KEY POINTS

- Each type of continuous-flow left ventricular assist device (cfLVAD) has a unique pressure differential to flow relationship as demonstrated by H-Q curves.
- Axial flow pumps have steeper, linear H-Q curves, resulting in less flow pulsatility, compared with centrifugal flow pumps, which have flatter H-Q curves—translating to little change in the pressure differential in response to low flows.
- The echocardiographic ramp study can be used to determine the adequate cfLVAD speed to unload the ventricle (midline intraventricular septum, intermittent aortic valve opening and minimize mitral regurgitation).
- Invasive hemodynamics, combined with echocardiography, is a useful assessment tool to best choose a speed optimize hemodynamics (CVP <12 and PCWP <18 mm Hg) and adequately unload the left ventricle.
- Three-dimensional transthoracic echocardiography during ramp studies can help better discern corresponding shapes of the left ventricle and right ventricle during device speed up titration, and suggests that device position (intrapericardial vs extrathoracic) may significantly affect changes in ventricular structure and shape during speed adjustment.

Fig. 1. Flow-dependent changes of the PVL with leftward shift representing greater LV unloading. EDV, end-diastolic volume; ESV, end-systolic volume.

Fig. 2. Flow (Q) compared with pressure differentials (H) between inflow cannula and outflow graft at various pump speeds for (A) centrifugal pump and (B) axial flow pump.

Fig. 3.

Three-dimensional endocardial surfaces of the LV (*orange*) and RV (*red*) were obtained at the lowest (*gray outline*) and highest revolutions per minute (RPM) for both the axial flow pump (*A*) and centrifugal flow pump (*B*). (From Addetia K, Uriel N, Maffessanti F, et al. 3D morphologic changes in LV and RV during LVAD ramp studies. JACC Cardiovasc Imaging 2017;11(2 Pt 1):159–69; with permission.)

Fig. 4.

Plot of individual patients' CVP versus PCWP at baseline, highest LVAD speed, and final measurement; 5 zones are described including normal, left heart failure (LHF), fluid overload, right heart failure (RHF), and hypovolemia (Hypo). (*From Uriel N, Sayer G, Addetia K, et al. Hemodynamic ramp tests in patients with left ventricular assist devices. JACC Heart Fail 2016;4(3):213; with permission.*)

Fig. 5.

(A) Measurement of LVAD outflow cannula diastolic acceleration and LVAD outflow cannula systolic-to-diastolic peak velocity ratio (S/D) using pulse wave Doppler. (B) AI severity measured by a novel mean RF compared with standard transthoracic echocardiography grading using a combination of vena contracta (VC) and visual estimation. (Adapted from Grinstein J, Kruse E, Sayer G, et al. Accurate quantification methods for aortic insufficiency severity in patients with LVAD: role of diastolic flow acceleration and systolic-to-diastolic peak velocity ratio of outflow cannula. JACC Cardiovasc Imaging 2016;9(6):641–51; with permission.)

Fig. 6.

(A) Correlation of LVAD outflow cannula systolic-to-diastolic peak velocity (S/D) ratio compared with RF for patients with trace or greater AI. (B) Correlation of LVAD outflow cannula diastolic acceleration compared with RF. (C) Correlation of LVAD outflow cannula S/D ratio compared with PCWP for patients with trace or greater AI. (D) Correlation of LVAD outflow cannula diastolic acceleration compared with PCWP. (From Grinstein J, Kruse E, Sayer G, et al. Accurate quantification methods for aortic insufficiency severity in patients with LVAD: role of diastolic flow acceleration and systolic-to-diastolic peak velocity ratio of outflow cannula. *JACC Cardiovasc Imaging* 2016;9(6):647; with permission.)