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Patient–specific hemodynamic modeling to optimize LVAD speed and right heart health

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BACKGROUND: Left ventricular assist device (LVAD) speed optimization and right heart failure post device implantation are major clinical challenges. Right heart catheterization (RHC)–guided speed titration studies are often performed to optimize LVAD settings, which are unknown and must be optimized for each patient. A virtual hemodynamic model (VHM) that can be tailored to each patient may provide useful guidance and reduce repeated studies.
METHODS: We conducted a retrospective analysis on 16 patients implanted with HeartMate 3 (HM3) who underwent RHC speed titration study as an outpatient. A custom-designed VHM was built and customized for each patient based on RHC measurements. VHM predictions were obtained for multiple scenarios: (1) population–based pulmonary system parameters, (2) patient-specific systemic and pulmonary resistance and capacitance parameters, (3) clinical optimization–based patient-specific mean arterial pressure (MAP), and (4) several MAP targets ranging from 70 to 90 mm Hg.
RESULTS: All patients who underwent RHC speed titration had a clinician–guided speed increase, with a median increase of 300 revolutions per minute (rpm). Using each patient's customized VHM, virtual speed optimization demonstrated congruence with clinician-guided optimization, with a median predicted speed increase of 321 rpm. After virtual optimization, there was a decrease in the pulmonary artery pressure for 13 patients (81.25%), indicating a predicted improvement in pulmonary parameters.
CONCLUSIONS: For our cohort of 16 patients, there was an overall congruence between clinician–guided and patient–specific VHM-predicted optimal LVAD speeds. The magnitude of speed change varied depending on individual patient targets. This may provide individualized speed titration goals and lessen the need for repeat invasive studies.
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Background

Left ventricular assist device (LVAD) therapy for end-stage heart failure patients remains a strong treatment option; however, its efficacy is hindered by several complications that significantly influence the quality of life, morbidity,

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and mortality. Among these complications, hemodynamically related adverse events continue to remain an active area of research focus due to the potential of optimizing patient treatment options in a customized manner. Specifically, LVAD speed optimization and right heart failure (RHF) post-LVAD implantation are major clinical challenges, particularly as the duration of LVAD support has increased.[1]

Several studies have reported varying incidence of post-LVAD RHF, ranging from 5% to 40%, which is also associated with poor outcomes.[1-5] Indeed, RHF was noted as an important factor in the cause of complications and death according to recent Interagency Registry for Mechanically Assisted Circulatory Support data.[6] Multiple factors contribute to RHF including pre-, post-, and intraoperative procedures.[7] Among these factors, hemodynamic optimization of LVAD speed and associated blood pressure management plays a critical role in the onset and precipitation of RHF due to the pump-patient interplay. A poorly optimized LVAD speed may cause pressure-volume overloading of the right ventricle that, coupled with a septal shift for excessive speeds, can result in RHF.[8-11] Similarly, abnormally high pulmonary vascular resistances can also precipitate RHF if left unchecked. The lack of clear guidelines and patient management strategies makes it difficult for optimizing patients with a view to reducing post-LVAD RHF.[12]

Understanding the interplay between the LVAD and patient's circulatory system is critical for optimizing LVAD therapy and RHF.[9,13] The ideal LVAD set speed is unknown and must be individualized for each patient. Right heart catheterization (RHC)–guided speed titration studies are often performed to optimize LVAD settings. This can predispose patients to the risk of an invasive procedure as well as the uncertainty of how programmed speed changes will affect patients beyond the immediate period of intraprocedural observation. A computational model of ideal speed settings may therefore provide clinicians with an initial approach to speed optimization and potentially reduce the need for repeated invasive studies. In this study, we therefore sought (1) to compare clinician–guided and computational model–predicted optimal LVAD speeds and mean arterial pressure (MAP) and (2) to evaluate what-if scenarios to determine LVAD speeds for different MAP targets.

Methods

Patient data

In this Institutional Review Board-approved retrospective study, 16 consecutive patients implanted with an HeartMate 3 (HM3) LVAD at the University of Florida over the age of 18 who underwent RHC speed titration as an outpatient were reviewed retrospectively. Median age was 63 years, 69% of patients were male and 75% were White. Exactly 69% were implanted as destination therapy. Median baseline speed was 5,100 revolutions per minute (rpm). Baseline hemodynamic and LVAD data were obtained before RHC, as well as after clinician–guided speed optimization during follow-up.

The study is divided into 2 phases: (1) A "customization" phase where patient-specific parameters are incorporated in the virtual hemodynamic model (VHM) to tailor it for each patient, followed by (2) the "optimization" phase where the customized patient-specific model is probed to explore speed optimization and other settings.

Virtual patient–specific hemodynamic model customization

We used a custom–designed computational hemodynamic lumped parameter model of the circulatory system incorporating the systemic and pulmonary resistances, capacitances, along with the 4 chambers of the heart via 30+ coupled differential-algebraic equations validated in previous LVAD studies.[9,14] The HM3 LVAD was incorporated via manufacturer–provided pressure-flow curves allowing for parallel flow through the LVAD and left ventricle. For each patient, the VHM was customized to reflect the measured hemodynamic parameters, namely systemic vascular resistance, left ventricle contractility, LVAD speed, MAP, and cardiac output. A dedicated multivariable optimization algorithm was designed to tailor the VHM parameters to match patient measurements for each patient, as shown in the central schematic in Figure 1.

Two versions of the VHM were created for each patient to investigate the importance of model customization: (1) with population-based pulmonary parameters[15-17] and (2) incorporating pulmonary RHC measurements. The pulmonary parameters such as pulmonary vascular resistance (PVR) and pulmonary capacitance were incorporated as follows: $PVR = \frac{(mPAP - PCWP)}{CO}$ and $Pulmonary\ Capacitance = \frac{CO/HR}{sPAP - dPAP}$. Here, mPAP is the mean pulmonary artery pressure, PCWP is the wedge pressure, sPAP and dPAP are the systolic and diastolic pulmonary artery pressures, respectively, which were obtained from patient-specific RHC measurements. For patients who did not have a heart rate(HR) measurement, a value of 70 bpm was specified in the VHM. The originally measured LVAD speed, MAP, cardiac output, PVR, pulmonary capacitance, and systemic vascular resistance were used to customize the VHM for each patient to obtain 16 different patient–specific hemodynamic models that matched RHC and LVAD measurements preoptimization. Optimization was performed as described below.

Virtual patient optimization

Model optimization was performed with the VHM for multiple scenarios: (1) population–based pulmonary system model, (2) including patient–specific systemic parameters, PVR, and pulmonary capacitance, (3) clinician–guided optimization-based patient-specific MAP, and (4) for several MAP targets ranging from 70 to 90 mm Hg. For each scenario, we analyzed the optimal LVAD speed to match

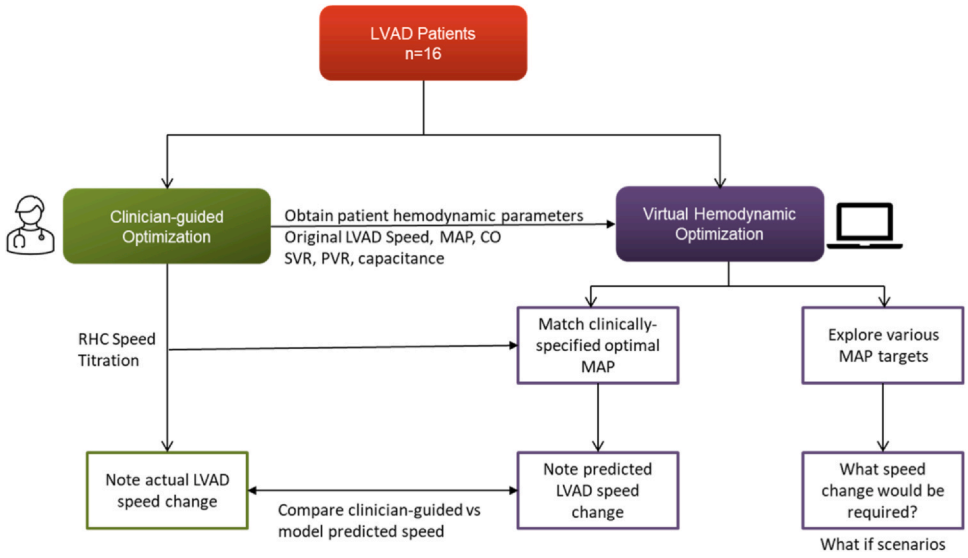

Figure 1 Central figure indicating the approach used for this study to compare clinician-guided optimization to patient-specific virtual hemodynamic model prediction. CO, cardiac output; LVAD, left ventricular assist device; MAP, mean arterial pressure; PVR, pulmonary vascular resistance; RHC, right heart catheterization; SVR, systemic vascular resistance.

the specific scenario and the difference from the clinically obtained optimal speeds to assess congruency. Furthermore, we assessed the reduction of right heart overload after virtual patient optimization for each scenario.

Comparison to patient outcomes

Outcomes for patients were analyzed and correlated to congruency of clinician–guided and model–predicted LVAD speeds. Congruency was defined as a difference of less than 200 rpm between clinician-guided and model-predicted speeds.

VHM optimization and all analyses were performed in MATLAB (MathWorks Inc., Natick, MA). Normality of data was investigated using Shapiro-Wilk assessment, and statistical analysis was performed using paired 2-tailed *t*-tests to compare clinician-guided and model-predicted metrics.

Results

Virtual optimization with population-based pulmonary system model

Using population-based pulmonary parameters indicated a large mismatch between patient measurements and VHM parameters, as seen in Figure 2a for the pulmonary artery pressure (PAP) range. Bland-Altman analysis determined a bias of nearly 22 mm Hg between the patient measurements and the population-based models for the systolic PAP, indicating mismatch for the patient cohort and proportional error, as seen in Figure 2b. Due to the mismatch, all

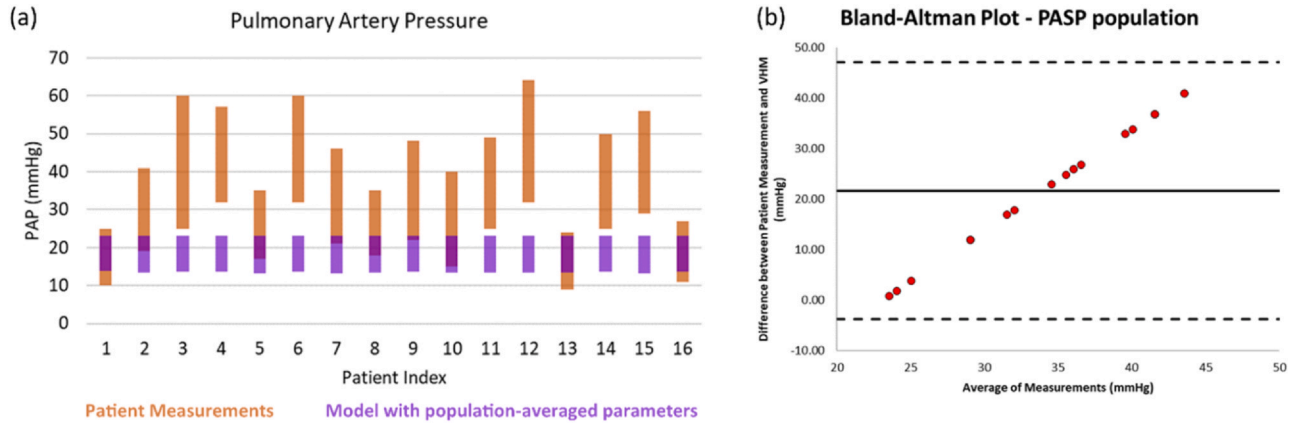

Figure 2 (a) VHM results with population-based parameters (purple) compared to patient measurements (orange) for pulmonary artery pressure variation, indicating the need for patient–specific model customization, (b) Bland-Altman plot for systolic PAP depicting a proportional error and significant bias of nearly 22 mm Hg between population-based model and patient measurements indicating mismatch for patient cohort. PAP, pulmonary artery pressure; PASP, pulmonary artery systolic pressure; VHM, virtual hemodynamic model.

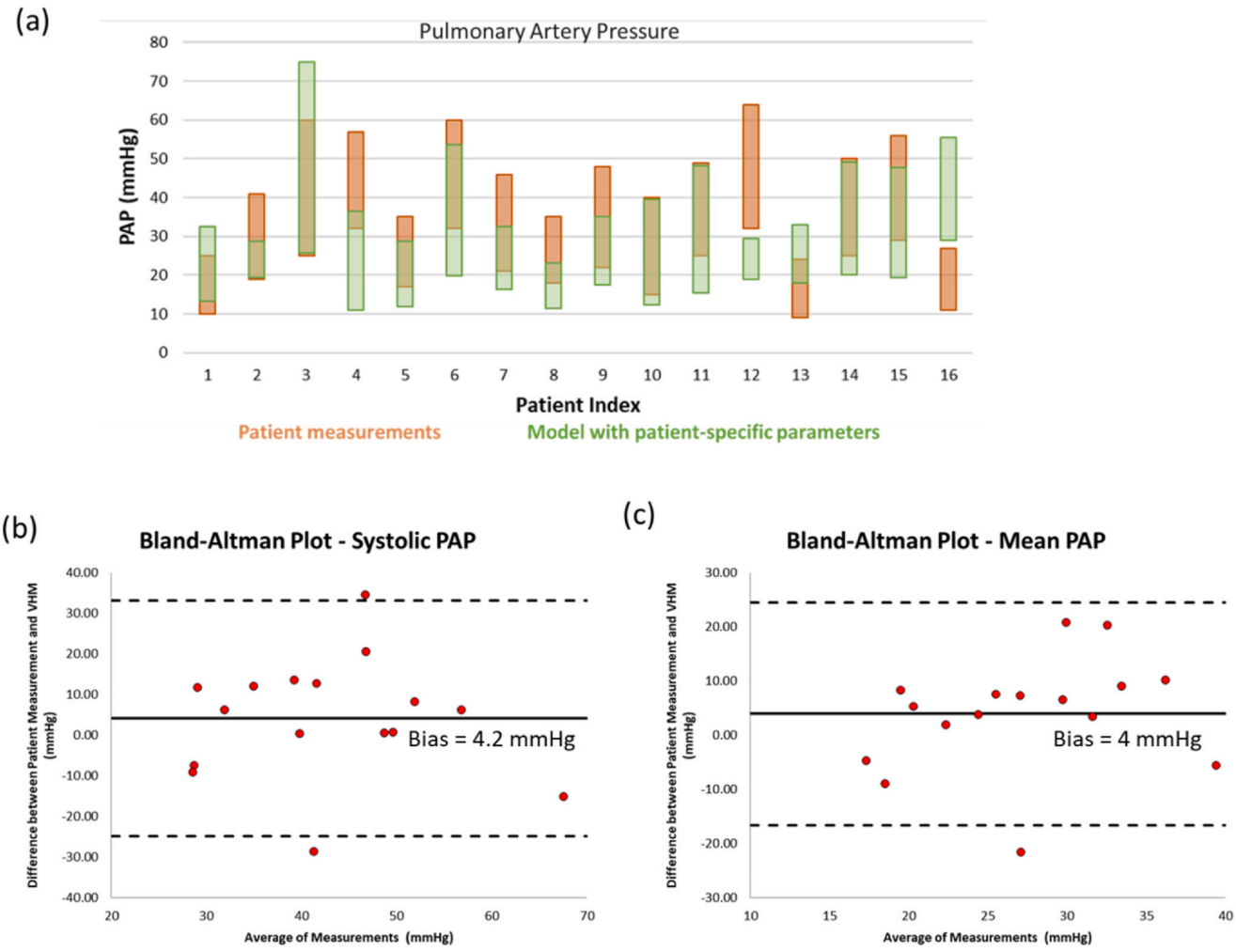

Figure 3 (a) Comparison of customized patient–specific hemodynamic model (green) with patient RHC measurements for pulmonary artery pressure ranges, demonstrating the efficacy of model customization for each patient in our cohort. Exactly 81.25% of patients demonstrated an improvement in matching pulmonary parameters after customization, (b) Bland-Altman plot of systolic PAP and (c) Bland-Altman plot of mean PAP between customized model and patient measurements depicting minimal bias of approximately 4 mm Hg, indicating a more accurate representation of patient cohort investigated in this study. PAP, pulmonary artery pressure; RHC, right heart catheterization.

subsequent analyses were performed with patient-specific parameters for increased accuracy and relevancy.

Virtual optimization with patient-specific systemic and pulmonary parameters

Including patient-specific systemic and pulmonary parameters matched the patient measurements well, as seen in Figure 3a for the PAP range for most patients before virtual optimization. Exactly 81.25% of patients showed improvements in matching RHC measurements and only 2 patients (P12 and P16) had a mismatch in the measured vs VHM range of PAP. Bland-Altman analysis showed a minimal bias of only around 4 mm Hg for both sPAP and mPAP using patient-specific parameters, as seen in Figure 3b and c, indicating that the patient-specific approach is representative of the patient cohort investigated in this study.

Using the patient-specific VHM, we performed LVAD speed optimization to match clinically specified postoptimization MAP values. Virtual hemodynamic optimization predicted a speed increase for each patient, which aligned with the clinician-guided optimization (which also resulted in a speed increase for each patient). Maximal speed change predicted by virtual optimization and after clinical-guided optimization were 755 and 600 rpm, respectively, as seen in Figure 4.

However, the magnitude of speed increase varied between patients, as seen in Figure 5. The VHM predicted a smaller increase for 9 patients compared to clinical-guided optimization. Overall, the median speed increase for clinician-guided optimization was 300 rpm, while the median speed increase predicted by our virtual optimization model was 321 rpm, indicating congruency. Further, statistical analysis using a paired 2-tailed *t*-test resulted in a *p*-value of 0.72 between the clinician–guided and model–predicted median LVAD speeds indicating that the 2 groups are not statistically different. This behavior was also seen for the median speed change for the clinician-guided and the model-predicted groups ($p = 0.72$, paired 2-tailed *t*-test) indicating that both groups are not statistically different.

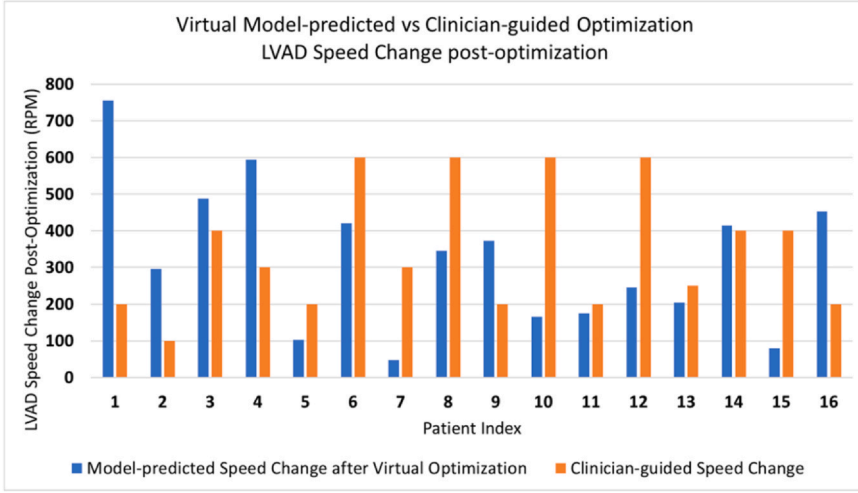

Figure 4 Comparison of LVAD speed change predicted by clinician-guided and virtual model–predicted optimization. LVAD, left ventricular assist device.

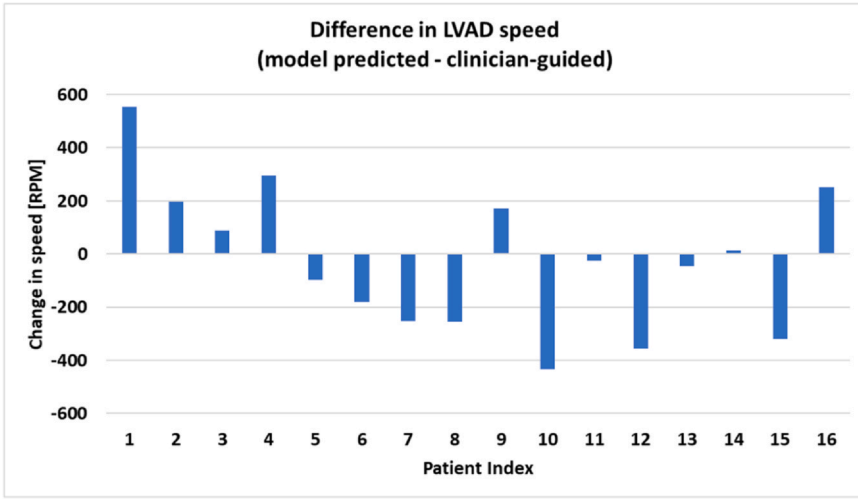

Figure 5 Change in LVAD speed after optimization comparison between virtual model predictions and actual clinician–guided speed optimization. Median speed change for clinician-guided and virtual model–predicted optimization were 300 and 321 rpm, respectively. LVAD, left ventricular assist device.

After virtual optimization, there was a decrease in the PAP range and mPAP for 13 patients (81.25%), indicating a predicted improvement in pulmonary parameters, as seen in Figure 6. Model optimization predicted a decrease in the systolic PAP for 12 patients, with a median decrease of 12 mm Hg. Overall, virtual optimization predicted a median reduction of 6.6 mm Hg in mPAP.

Comparison to patient outcomes

Exactly 75% of patients who had noncongruence (i.e., a difference of over 200 rpm between clinician-guided and model-predicted speeds) had a heart failure or LVAD-related admission within 6 months of the speed titration study compared to 12.5% of the patients who had congruence. Exactly 37.5% of patients who had noncongruence died within 1 year of the speed titration study compared to 0 patients who had congruency.

Virtual optimization for different MAP targets

We also performed virtual optimization for various MAP targets. Using the VHM, there was an overall predicted speed decrease of 250 rpm for a MAP target of 70 mm Hg, with 14 patients (87.5%) requiring a speed decrease from the clinically measured preoptimization speed. For a target MAP of 80 mm Hg, 10 patients (62.5%) required a speed reduction, with an overall required speed decrease of 60 rpm, as shown in Figure 7. As MAP target increased to 90 mm Hg, 10 patients (62.5%) required a speed increase, with an overall median speed increase of 140 rpm necessary to meet the target MAP of 90 mm Hg.

Table 1 indicates the median speed change compared to clinically-measured preoptimization, and the median LVAD speed for several conditions evaluated using our VHM. There was congruency in the median LVAD speed (5,400 for clinician-guided optimization vs 5,407 for patient–specific VHM optimization). Additionally, the median speed change for

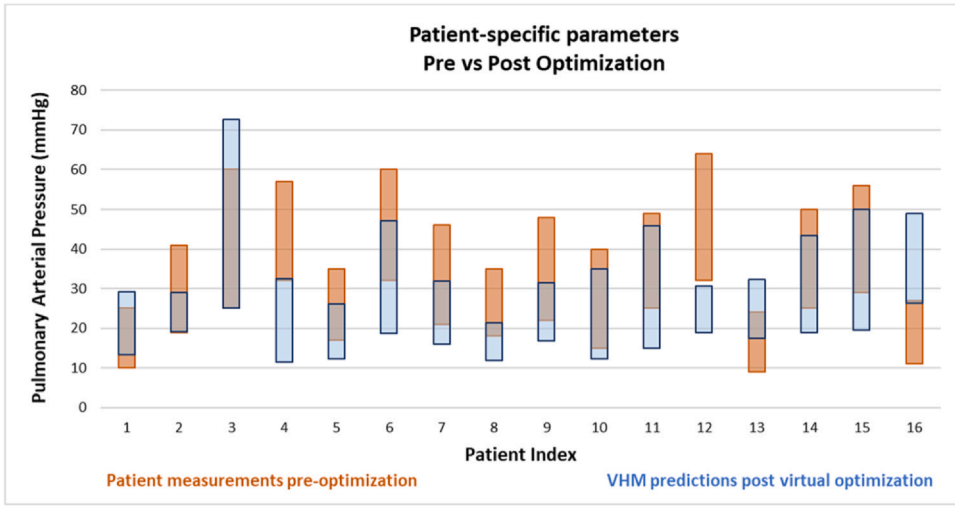

Figure 6 Predicted effect of virtual model–predicted speed optimization on pulmonary artery pressure range, comparing pre-optimization (orange) and after virtual optimization (blue). Virtual optimization resulted in a decrease in pulmonary pressures for 13 patients (81.25%) indicating improvement postoptimization.

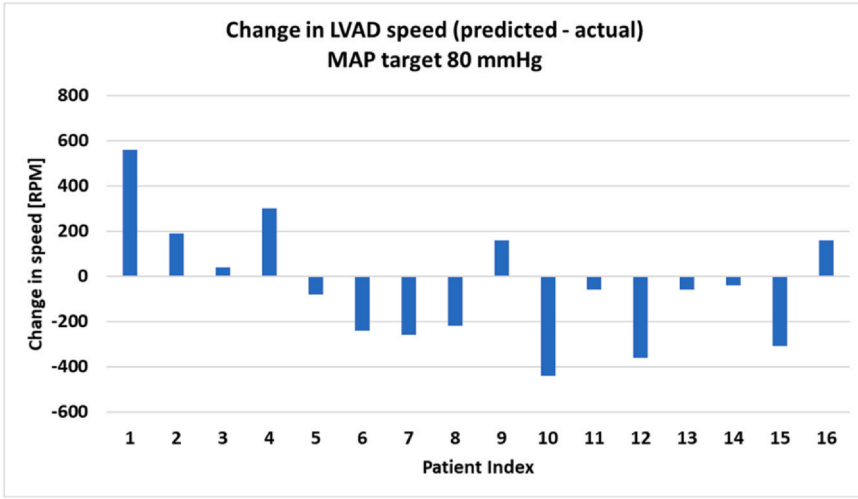

Figure 7 Change in LVAD speed for a hypothetical MAP target of 80 mm Hg. Median speed change after virtual optimization was 325 rpm, compared to median speed change of 300 rpm after clinician-guided optimization. Note the variation in the magnitude of speed change for each patient based on model customization, indicating the necessity of patient-specific optimization assessment. LVAD, left ventricular assist device; MAP, mean arterial pressure.

clinician-guided optimization was 300 rpm, while the patient-specific VHM incorporating both systemic and pulmonary parameters indicated a median speed change of 321 rpm, demonstrating congruency. The median speed change for the aforementioned multiple MAP targets increased with increasing MAP, as shown in Table 1.

Discussion

RHF after LVAD implantation has been an ongoing major complication of LVAD therapy. The main parameters influencing this delicate interplay between pulmonary, systemic and LVAD parameters have been wedge pressure, PVR, MAP, degree of left heart failure, and LVAD settings. Among these, the preload-afterload relationship, coupled with the LVAD parameters, is crucial to investigate and understand the interplay between the patient and device. In this study, we sought to develop and utilize a VHM to (1) better understand the necessity and impact of patient-specific model customization, (2) compare VHM optimization to clinical-guided optimization, and (3) explore virtual optimization options for several clinically relevant scenarios.

Lumped parameter-based hemodynamic models have been utilized by researchers previously, including our group.[9,11,15,18] However, one of the most challenging tasks is determining some of the parameters for the systemic and pulmonary circulations to customize the model for each patient. A recent study analyzed the hemodynamics using a combination of invasive ramp test measurements and a computational hemodynamic model on 4 patients, determined that such models can provide valuable insight into LVAD-

Table 1 Comparison of Median LVAD Speed Change and Median LVAD Speeds Between Clinician-Guided and Virtual Patient–Specific Optimization for Various Targets

MAP target	Clinician-guided optimization	Patient-specific VHM optimization	MAP target 70 mm Hg	MAP target 80 mm Hg	MAP target 90 mm Hg
Median speed change from preoptimization setting (rpm)	300	321	160	325	530
Median LVAD speed postoptimization (rpm)	5,400	5,407	5,160	5,360	5,580

Abbreviations: LVAD, left ventricular assist device; MAP, mean arterial pressure; VHM, virtual hemodynamic model.

patient hemodynamics and stated the need for further research into this domain.[11] In this study, we incorporated several critical systemic and pulmonary parameters including ventricular contractility, systemic vascular resistance, MAP, systemic vascular resistance (SVR), and pulmonary capacitance using clinical measurements obtained via RHC for 16 patients. First, we analyzed the impact of using population–derived pulmonary parameters vs patient-derived parameters while keeping all other parameters consistent. Our study showed that, using population-derived values, there was a significant difference in the VHM-predicted pulmonary behavior, especially PAP, as shown in Figure 2. The population-based model parameters indicated a "healthy" range of PAP that did not reflect the pulmonary circulatory behavior of our patient cohort. Out of the 16 patients, only 3 patients demonstrated an overlap in PAP. Bland-Altman analysis corroborated this mismatch, indicating not only a significant mismatch and a bias of nearly 22 mm Hg, but also proportional error in using population-based parameters as shown in Figure 2b. This provided a benchmark indicating that population-derived parameters are not recommended when performing such hemodynamic optimization analysis, and more detailed patient-based assessment is needed. When patient–derived RHC parameters were incorporated into the VHM, we achieved significantly greater overlap and customized behavior in pulmonary system parameters, as seen in Figure 3. This was also backed up by the Bland-Altman analysis which indicated a very minimal bias of around 4 mm Hg, as shown in Figure 3b and c, resulting in a much better representation of the patient cohort investigated in this study. Specifically, the parameters that contributed the most toward achieving successful customization were PVR and pulmonary capacitance for the pulmonary circulation. Similarly, SVR was the major customization parameter that had the greatest positive impact for the systemic circulation. Thus, our analysis clearly demonstrates that before utilizing hemodynamic models for analysis or patient optimization, the models need to be customized to each patient for which RHC-derived parameters are essential, along with standard measured parameters, for patient-specific hemodynamic analysis.

We performed patient-specific analyses on 16 patients to compare the VHM–predicted optimal LVAD speed with RHC clinician-guided optimization. After model customization for each patient, VHM predicted a median speed increase of 321 rpm (median speed 5,407 rpm) for our patient cohort. This compared very well with clinical-guided optimization, which had a median speed increase of 300 rpm (median speed 5,400 rpm) postoptimization, as shown in Table 1. Statistical analyses using a paired 2-tailed *t*-test gave a *p*-value of 0.72 for the median postoptimization speed for both groups, indicating that the 2 groups are not different from each other (using $p < 0.05$ for significance). This is also true for the median speed changes postoptimization: statistical analyses indicated a *p*-value of 0.72, once again indicating that these 2 groups were not different (using $p < 0.05$ for significance). Normality was evaluated using the Shapiro-Wilk test, which indicated that the optimal LVAD speeds distributions were normal for both clinician-guided and model-predicted data, and also for the speed change distribution for the model-predicted data. The clinician–guided speed change distribution did not show normality as per the Shapiro-Wilk test. However, despite the differences between the groups for each patient (which is expected in a patient-specific analysis), the VHM was congruent with clinician-guided optimization. Using the VHM, we analyzed the effect of optimization on pulmonary parameters, using PAP as an indicator of afterload on the right ventricle. In our study, 13 patients (81.25%) had a decrease in mPAP after VHM optimization, as shown in Figure 5, with a median decrease in mPAP of 6.6 mm Hg postoptimization. Importantly, VHM optimization indicated different optimal speed requirements for each patient. Specifically, 7 patients required a speed increase while the remaining 9 patients needed a speed decrease compared to clinician-guided optimization, demonstrating the patient-specific nature of hemodynamic optimization. Overall, the median difference between VHM optimization and clinician-guided optimization was a decrease of 35 rpm, indicating that the VHM was congruent with clinical optimization. We further analyzed clinically relevant what-if scenarios of different MAP targets ranging from 70 to 90 mm Hg. As expected, higher MAP targets necessitated a larger speed increase, while a lower MAP target of 70 mm Hg required an overall speed reduction in most patients, due to the pressure-flow relationships and pump-patient interdependence. This naturally depended upon the initial preoptimization speed and MAP; however, the magnitude of speed change for each patient was different, as shown in Figures 4 and 5, further emphasizing the patient-specific approach necessary for hemodynamic optimization.

We further analyzed the outcomes of patients that were congruent (defined as being within a difference of 200 rpm between clinician–guided and model–predicted optimized speeds) and those that were not. Patient outcomes indicated

a correlation of noncongruency of model-predicted speed optimization with adverse outcomes, such as hospital readmission due to LVAD-related issues or death. It is possible that the patients who were noncongruent for clinician-guided vs VHM optimization had other factors influencing their cardiovascular system function, specifically pulmonary circulation and pump-patient interaction. Given the small sample size of this study, it is not powered to detect clinical differences or to conduct rigorous statistical analyses, but our analysis is hypothesis generating and suggests further exploration in multicenter trials with appropriate power and adjusted for various factors such as age, gender, patient history, duration of LVAD support and aortic valve opening. This also provides more insight into the use of RHC titration tests in a patient-specific manner keeping in mind other potential contributing factors.

Hemodynamic ramp tests via RHC are routinely performed in many LVAD centers for monitoring and follow-up. Data obtained from such tests can be invaluable for patient-specific hemodynamic optimization, especially with LVAD patients spending increasingly long times on the device, either as destination therapy or due to the long wait times for transplantation. Post-LVAD RHF assessment is crucial for patient health and outcomes. Hemodynamic optimization models such as the VHM presented in this study can aid clinicians in evaluating performance, identifying and analyzing trends in critical parameters, and also explore what-if scenarios in a longitudinal fashion. Recently, a new hemodynamic index was proposed by a group from Italy and demonstrated that the change in index values was correlated to RHF.[13] Moreover, patients with late RHF are associated with worse survival and adverse events, as evidenced in multiple studies.[6,8,12,19] It is being increasingly acknowledged that RHF is not an acute event in many cases, but rather exists on a spectrum and is intrinsically linked to LVAD operation and pump-patient interplay requiring optimization and evaluation. Another recent study further underscores this need for optimization and evolution—the incidence of RHF is high immediately postimplantation and tends to reduce and evolve in the longer term, highlighting the moving target nature of RHF.[12] Exactly 19% of the patients analyzed in their cohort developed RHF at 1-month postimplantation, with all RHF groups (mild, moderate, and severe) having significantly worse outcomes and adverse events. Studies such as ours could help provide more insight into the dynamic and evolving nature of RHF by assessing customized hemodynamic models using patient hemodynamic measurements and exploring the interdependencies between various relevant parameters for improving RHF prediction and patient outcomes.

Limitations

This was a single-center study and would benefit from diverse patient data from multiple centers; future studies are planned to include multiple institutions. The small sample size also precluded rigorous statistical analyses and larger datasets are needed. As this is a computational hemodynamic model, limitations exist in representing the dynamic cardiovascular system response including baroreflex, responsiveness time, preload, afterload, and systemic-pulmonary interaction. Some patients did not have heart rate measurements available at the time of RHC, and metrics of right ventricular function were not recorded. Furthermore, RHC data were only available during ramp studies and clinical optimization and no invasive hemodynamic data were collected postoptimization. Thus, future studies are envisioned to facilitate increased data collection post-ramp or optimization study, including echo-based measurements of right ventricular function, pulmonary parameters, and cardiac output to analyze the change in these parameters after LVAD speed and customized patient adjustment for obtaining patient-specific models. Finally, the RHF status of the above patients is unknown—future studies will include following patients RHF status and identifying customized model parameters for both groups. Future studies will also focus on recording and correlating short-term and long-term patient outcomes, along with other adverse events for RHF and non-RHF cohorts.

Conclusion

In this retrospective study of HM3 patients, we developed customized hemodynamic models for each patient to evaluate clinical-guided vs computational model–predicted LVAD speed optimization. Overall congruence was seen between RHC clinician-guided LVAD speed optimization and customized virtual hemodynamic computational model-predicted speeds. This may provide individualized speed titration goals and lessen the need for repeat invasive studies. Future investigations will include a prospective comparison focusing on clinical outcomes of those individuals with a congruence vs incongruence between clinical-guided and virtual optimization.

Author contributions

Mustafa Ahmed: Conceptualization, Investigation, Data Curation, Writing—review and editing; Holly Grant: Software, Formal analysis, Investigation; Jasmine Martinez: Software, Formal analysis, Methodology; Joshua Thomas: Writing—review and editing; Mohammad Al-Ani: Writing—review and editing; Alex Parker: Writing—review and editing; Juan Vilaro: Writing—review and editing; JuanAranda: Writing—review and editing; Venkat Keshav Chivukula: Conceptualization, Investigation, Supervision, Project administration, Writing—Original draft, reviewing and editing.

Disclosure statement

Mustafa Ahmed reports a relationship with Evaheart Inc. that includes board membership. The other authors declare that they have no known competing financial interests or

personal relationships that could have appeared to influence the work reported in this paper.

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