Numerical Simulation of a Biventricular Assist Device with Fixed Right Outflow Cannula Banding During Pulmonary Hypertension

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Abstract—As a left ventricular assist device is designed to pump against the systemic vascular resistance (SVR), pulmonary congestion may occur when using such device for right ventricular support. The present study evaluates the efficacy of using a fixed right outflow banding in patients receiving biventricular assist device support under various circulatory conditions, including variations in the SVR, pulmonary vascular resistance (PVR), total blood volume (BV), as well as ventricular contractility. Effect of speed variation on the hemodynamics was also evaluated at varying degrees of PVR. Pulmonary congestion was observed at high SVR and BV. A reduction in right ventricular assist device (RVAD) speed was required to restore pulmonary pressures. Meanwhile, at a high PVR, the risk of ventricular suction was prevalent during systemic hypotension due to low SVR and BV. This could be compensated by increasing RVAD speed. Isolated right heart recovery may aggravate pulmonary congestion, as the failing left ventricle cannot accommodate the resultant increase in the right-sided flow. Compared to partial assistance, the sensitivity of the hemodynamics to changes in VAD speed increased during full assistance. In conclusion, our results demonstrated that the introduction of a banding graft with a 5 mm diameter guaranteed sufficient reserve of the pump speed spectrum for the regulation of acceptable hemodynamics over different clinical scenarios, except under critical conditions where drug administration or volume management is required.

Keywords—Ventricular assist device, Heart failure, Pulmonary vascular resistance, Pulmonary artery banding, Systemic vascular resistance.

INTRODUCTION

Left ventricular failure is recognized as the most common cause of pulmonary hypertension (PH). It is caused by two mechanisms, including a passive effect from a rise in the left atrial pressure (LAP) translated through an increase in the pulmonary venous pressure, and an active constriction of the pulmonary arterioles in severe PH which serves to protect the lung from pulmonary edema. Right ventricular dysfunction may develop as a result of these high pulmonary pressures. PH is a relative contraindication to heart transplantation because the transplanted right ventricle (RV) is prone to failure in the early pre-operative period unless it has been conditioned by previous exposure to elevated pulmonary arterial pressure.

The implantation of a left ventricular assist device (LVAD) has become a common treatment for terminal heart failure patients. Although these devices help to reduce pulmonary arterial pressures in the long-term, their immediate effect is to increase the stress on the RV which subsequently leads to right ventricular dysfunction. This may occur because the LVAD reduces the stability of the interventricular septum and increases the right atrial pressure (RAP), thus increasing right ventricular loading. Right ventricular dysfunction has been noted in up to 48% of patients receiving an LVAD in their post-operative period—eventually requiring a second VAD for right ventricular assistance in about 5–50% of LVAD supported patients.
Due to the lack of commercially available right ventricular assist devices (RVADs), clinicians have implanted LVADs as the RVADs to support the RV in various clinical trials.\(^8,^{13}\) Since the LVAD is afterload sensitive, and as there is a large difference between the systemic vascular resistance (SVR) and the pulmonary vascular resistance (PVR), using an LVAD as an RVAD at the same design speed would result in markedly different flows.\(^8,^{28}\) Consequently, RVAD overpumping may occur, causing pulmonary congestion or RV suction. In order to maintain balanced systemic and pulmonary flows, the RVAD has to be operated at a much lower speed or with an artificially increased resistance in the outflow cannula.\(^8,^{28}\) Running an RVAD at a very low speed, however, produces low RVAD flows and impeller instability in pumps which employ a hydrodynamic bearing, thus increasing the potential for impeller-induced thrombosis.\(^1,^{30}\) On the other hand, restriction of the RVAD outflow graft allows the RVAD to be used at its design speed with an optimized pressure-flow relation. Operating both VADs at the design speed provides a greater flexibility for varying speeds in the face of varying physiological conditions, such as in pulmonary hypertension.\(^5,^8\)

Previous \textit{in vitro} and \textit{in vivo} studies\(^{5,8,12,13,23,28}\) have suggested that the safe banding diameter range lies between 5 and 8 mm, dependent upon the PVR. Although these studies have provided important insights into the use of two VADs in a biventricular assist device (BiVAD) configuration under different pathological states, few have included the effects of PH on overall hemodynamics while using a banded RVAD outflow cannula.\(^{5,28}\) Stevens \textit{et al.}\(^{28}\) suggested the use of different banding diameters for various PVR levels in order to maintain RVAD flows above 5 L/min. On the other hand, Gregory \textit{et al.}\(^{12}\) designed a compliant outflow cannula which passively deforms with afterload. Their experimental results demonstrated that high RVAD speeds (>2600 rpm) were required to maintain pulmonary flows even at a normal PVR condition. Although varying banding diameter could accommodate most physiological scenarios, further surgery is required.

The aim of the present study is to assess the effect of PVR on the hemodynamics of BiVAD-assisted patients under various circulatory conditions, including significant variations in the SVR, total blood volume (BV), as well as left and right ventricular contractility, using a fixed right outflow banding. In addition, the effect of LVAD and RVAD speed variation on the hemodynamics at varying degrees of PVR was also evaluated to investigate the required changes in LVAD or RVAD speeds to restore the pressures and flows using a fixed outflow graft. This study is intended to provide a guide to clinicians on the operation of dual rotary pumps under varying physiological conditions, in particular with patients demonstrating substantial changes in PVR during the early postoperative period.

\section*{Methodology}

\textbf{Model Description}

The numerical model used in the present study is adopted from a heart-pump interaction model developed and validated previously by our group based on experimental measurements obtained from five canines implanted with an LVAD over a wide range of operating conditions.\(^{14,15}\) An RVAD model, which is identical to the validated LVAD model, was then incorporated into the heart-pump interaction model, to investigate the interaction between the cardiovascular system (CVS) and a BiVAD.

In brief, the lumped parameter model consists of three main components: the CVS, the LVAD and the RVAD, as illustrated in Fig. 1. The contractilities of the atria and ventricles were modeled as time-varying elastance functions. The blood inertia, systemic and pulmonary vascular beds were represented by a series of inductances and resistances. Both LVAD and RVAD models include the description of the implantable rotary blood pump (IRBP) based on the VentrAssist\textsuperscript{TM} centrifugal pump, as well as the inlet and outlet cannulae, previously validated against both \textit{in vitro} pulsatile mock circulatory data and \textit{in vivo} animal experiments.\(^{15}\) The inflow of the LVAD was connected to the left ventricle (LV) and the outflow to the aorta, while the inflow of the RVAD was connected to the RV and the outflow to the pulmonary artery. The cannula resistances for both VADs were each modeled in terms of flow dependent resistances, and a variable suction resistance which is a function of the respective ventricular volumes, as described by Lim \textit{et al.}\(^{14}\)

In addition to the existing cannula (\(R_{\text{rout}}\)) and suction resistances (\(R_{\text{suc}}\)), the RVAD model consists of an additional flow-dependent banding resistance (\(R_{\text{banding}}\)) included at the outflow cannula to mimic the right outflow banding graft downstream of the RVAD. Hence, the total outflow cannula resistance for the RVAD, \(R_{\text{outRVAD}}\), is given by:

\begin{equation}
R_{\text{outRVAD}} = R_{\text{rout}} + R_{\text{suc}} + R_{\text{banding}}
\end{equation}

A banding diameter of 5 mm, as recommended by Krabatsch \textit{et al.}\(^{15}\) for a normal PVR condition, was chosen in the present study. The calculated Reynolds number (Re) of the fluid flow in the narrowed graft region falls in the transition region between laminar...
(\(<2300\)) and turbulent (\(>4000\)) flow, depending on the value of the RVAD outflow. As a result, we adopted the mock circulatory results obtained by Krabatsch et al.\(^{12}\) which describe the relationship between banding resistance, flow rate and banding diameter for a restriction length of 35 mm. At a banding diameter of 5 mm, the flow-dependent \(R\)\textsubscript{banding} is defined as:

\[
R\textsubscript{banding} (\text{dynes s cm}^{-5}) = \begin{cases} 636.1 & Q_{PR} < 3.44 \text{ L/min} \\ 241.5 \times Q_{PR} - 194.7 & Q_{PR} \geq 3.44 \text{ L/min} \end{cases}
\]

where \(Q_{PR}\) represents the RVAD flow.

### Simulation Protocol

The left \(E_{maxLV}\) and right \(E_{maxRV}\) ventricular elastances, as well as BV in the CVS model was fitted to reproduce previously published literature data obtained from biventricular failure patients before the insertion of BiVADs.\(^{3,10,11,13,19,31}\) The CVS model was then coupled to the BiVAD model, with both VADs operated at the mid range speed of 2400 rpm, as the recommended speed range for the VentrAssist\textsuperscript{TM} lies between 1800 and 3000 rpm.

In order to assess the robustness of a fixed RVAD outflow banding under varying physiological conditions, significant variations in the SVR, BV, as well as left and right ventricular contractility were simulated, in the presence of elevated PVRs as illustrated in Table 1. In each test scenario, PVR was increased from 100 to 600 dyne s/cm\(^5\) in 100 dyne s/cm\(^5\) increment. Since mean pulmonary artery pressure (MPAP) and PVR are used to define the degree of PH,\(^7\)\(^{,21}\) we defined PH as a condition when MPAP exceeds 40 mmHg together with a PVR of 200–300 dyne s/cm\(^5\) for mild, 301–400 dyne s/cm\(^5\) for moderate and over 400 dyne s/cm\(^5\) for severe hypertension.\(^7,21\)

The effect of LVAD and RVAD speed variation on the hemodynamics at mild (200 dyne s/cm\(^5\)), moderate (400 dyne s/cm\(^5\)) and severe PH (600 dyne s/cm\(^5\)) using a fixed outflow banding was assessed. Two sets of simulations were completed; one in which LVAD speed varied from 1800 to 3000 rpm in 100 rpm increments while RVAD speed was fixed at 2400 rpm and the other in which RVAD speed was increased from 1800 to 3000 rpm in 100 rpm increments with the LVAD speed fixed at 2400 rpm.

Based on personal communication from physicians through Dr. Robert Salamonsen, The Alfred Hospital, Melbourne, the acceptable safety limits for LAP, mean arterial pressure (MAP), MPAP, RAP and cardiac output (CO) were set as 3–20 mmHg, 65–105 mmHg, \(<40\text{ mmHg}\), 3–20 mmHg, and \(>4\text{ L/min}\) respectively. For each of the three test scenarios illustrated in

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**FIGURE 1.** Electrical equivalent circuit of the CVS–BIVAD interaction model. P, pressure; R, resistances; E, elastances; L, inertances; D, diodes. The model consists of three main components: (1) CVS, which includes ten compartments (la, left atrium; lv, left ventricle; ao, aorta; sa, systemic peripheral vessels including the arteries and capillaries; sv, systemic vein, including small and large veins; vc, vena cava; ra, right atrium; rv, right ventricle; pa, pulmonary arteries and capillaries; pu, pulmonary veins, (2) LVAD, which includes the IRBP and the cannula (\(R\textsubscript{lin}\) and \(R\textsubscript{lout}\), inlet and outlet cannula resistances; \(L\textsubscript{lin}\) and \(L\textsubscript{lout}\), inlet and outlet cannula inertances; \(R\textsubscript{lsuc}\), suction resistance) and (3) RVAD, which includes the IRBP and the cannula (\(R\textsubscript{rin}\) and \(R\textsubscript{rout}\), inlet and outlet cannula resistances; \(L\textsubscript{rin}\) and \(L\textsubscript{rout}\), inlet and outlet cannula inertances; \(R\textsubscript{rsuc}\), suction resistance). The intra thoracic pressure, \(P\textsubscript{thor}\), was assigned with –6 mmHg during closed chest simulated conditions.
Table 1, additional simulations were performed to identify the minimum speed changes required to bring the LAP, MPAP, RAP and CO back to their safety levels when any of these variables exceed their acceptable ranges.

RESULTS

As illustrated in Table 2, the simulated hemodynamics agreed reasonably well with that reported by previously published literature data obtained from biventricular failure patients before the insertion of BiVADs.\textsuperscript{3,10,11,19,31} In a non-restricted setting, both the LAP and MPAP were substantially increased under the same speed mode for both VADs, leading to pulmonary congestion. On the other hand, the RAP dropped significantly to the verge of RV suction. This agreed with the experimental results reported by previously published studies,\textsuperscript{25,29} which demonstrated the increased risk of pulmonary congestion and RV suction when both VADs were operated at the same speed using a non-restricted setting. With the banded outflow graft, our hemodynamic results fell within the recommended range, i.e., LAP between 8 and 13 mmHg, and CO > 5 L/min.\textsuperscript{13}

**Table 1. Illustration of variations in the intrinsic parameters for the CVS-BiVAD model to evaluate the robustness of a fixed outflow banding for the RVAD under varying physiological conditions.**

<table>
<thead>
<tr>
<th>Test scenario 1: variation in SVR (dynes s/cm(^5))</th>
<th>Test scenario 2: variation in BV (mL)</th>
<th>Test scenario 3: variation in (E_{\text{maxLV}}/E_{\text{maxRV}}) (mmHg s/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low 800</td>
<td>Low 5300</td>
<td>Improved LVC (E_{\text{maxLV}} = 1.5) (E_{\text{maxRV}} = 0.4)</td>
</tr>
<tr>
<td>Med (baseline) 1200</td>
<td>Med (baseline) 6100</td>
<td>Severe BVF (Baseline) (E_{\text{maxLV}} = 1.0) (E_{\text{maxRV}} = 0.4)</td>
</tr>
<tr>
<td>High 2000</td>
<td>High 6900</td>
<td>Improved RVC (E_{\text{maxLV}} = 1.0) (E_{\text{maxRV}} = 0.6)</td>
</tr>
</tbody>
</table>

In each test scenario, pulmonary vascular resistance (PVR) was increased from 100 to 600 dyne s/cm\(^5\) in 100 dyne s/cm\(^5\) increment. SVR systemic vascular resistance, BV blood volume, LVC left ventricular contractility, RVC right ventricular contractility, \(E_{\text{maxLV}}\) left ventricular elastance, \(E_{\text{maxRV}}\) right ventricular elastance.

**Table 2. Hemodynamics for severe biventricular failure pre- and post-BiVAD insertion, using the same speed mode (2400 rpm) with and without banding of the RVAD outflow cannula.**

<table>
<thead>
<tr>
<th>Hemodynamic variables</th>
<th>Pre-BiVAD insertion</th>
<th>Post-BiVAD insertion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Simulation</td>
<td>Literature</td>
</tr>
<tr>
<td>LAP (mmHg)</td>
<td>21.9</td>
<td>19–29.5\textsuperscript{3,10,19,31}</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>72.0</td>
<td>71–78\textsuperscript{3,19}</td>
</tr>
<tr>
<td>MPAP (mmHg)</td>
<td>31.1</td>
<td>30–37.9\textsuperscript{3,10,19,31}</td>
</tr>
<tr>
<td>RAP (mmHg)</td>
<td>15.7</td>
<td>11.6–20\textsuperscript{3,10,19,31}</td>
</tr>
<tr>
<td>CO (L/min)</td>
<td>3.6</td>
<td>2.6–4.5\textsuperscript{3,10,19,31}</td>
</tr>
<tr>
<td>SVR (dynes s/cm(^5))</td>
<td>1237.5</td>
<td>800–3500\textsuperscript{12}</td>
</tr>
<tr>
<td>PVR (dynes s/cm(^5))</td>
<td>200</td>
<td>100–200\textsuperscript{3,10,12,13,31}</td>
</tr>
<tr>
<td>QpL (L/min)</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>QpR (L/min)</td>
<td>0.0</td>
<td>0.0</td>
</tr>
</tbody>
</table>

A banding diameter of 5 mm, as recommended by Krabatsch et al.\textsuperscript{13} for a normal PVR condition was chosen. LAP left atrial pressure, MAP mean arterial pressure, MPAP mean pulmonary arterial pressure, RAP right atrial pressure, CO cardiac output, SVR systemic vascular resistance, PVR pulmonary vascular resistance, QpL LVAD flow, QpR RVAD flow.
limit of 20 mmHg during high SVR state, and this could be resolved by decreasing the RVAD speed to 1800 rpm. Decreasing SVR showed an opposite trend, with a reduction in LAP, MAP and MPAP, and an increase in RAP and CO. It is worth noting that decreasing SVR increased the magnitude of change in all variables with PVR. At a PVR of 400 dynes s/cm$^5$ and above, the LAP fell below its lower limit, thus increasing the risk of LV suction. Increasing RVAD speed could increase LAP to its acceptable range with a PVR between 400 and 500 dynes s/cm$^5$. However, any further increase in RVAD speed to restore LAP given at PVR of 600 dynes s/cm$^5$ led to an increase in MPAP above its upper threshold limit of 40 mmHg.

Variation in BV, on the other hand, did not significantly affect the magnitude of change in LAP, MAP, MPAP, RAP and CO with varying PVR levels. As indicated in Fig. 3, increasing BV produced a substantial increase in all variables. Consequently, the upper limit for LAP was exceeded at a normal PVR of 100 dynes s/cm$^5$, while MPAP rose above 40 mmHg at PVR above 500 dynes s/cm$^5$. A reduction in RVAD speed to 1800 rpm was required to reduce LAP below 20 mmHg at a PVR of 100 dynes s/cm$^5$, while a decrease to 2000 rpm was required to reduce MPAP below 40 mmHg at a PVR of 600 dynes s/cm$^5$. Low BV condition, on the other hand, experienced a greater risk of LV suction, where our simulation results showed that LAP dropped below its lower limit at a PVR of 400 dynes s/cm$^5$ and above. On the other hand, the risk of RV suction was prevalent at a PVR of 400 dynes s/cm$^5$ and below. Increasing RVAD speed or decreasing LVAD speed could help minimize the risk of LV suction, while the risk of RV suction can be lowered with an increase in LVAD speed or a reduction in RVAD speed. However, since both LAP and RAP values were substantially decreased at a low BV condition, speed alteration aimed to minimize the risk of suction in one ventricle would increase the chance for suction in the other.

Figure 4 illustrates the effect of improved LV and RV contractility on LAP, MAP, MPAP, RAP and CO at varying PVR. Improved RV contractility caused a substantial increase in LAP and MPAP, with a lesser increase in MAP and CO. In addition, RAP was significantly reduced. With an improvement in RV contractility, increasing PVR levels have a lesser effect on the reduction of LAP and CO as compared to a severe BVF condition. On the contrary, MPAP rose substantially with increasing PVR, leading to pulmonary congestion at a PVR of 500 dynes s/cm$^5$ and above. Further simulations showed that a reduction in RVAD speed to 1800 rpm was required to reduce MPAP below 40 mmHg at a PVR of 600 dynes s/cm$^5$. 

![Figure 2. Effect of systemic vascular resistance (SVR low: 800 dynes s/cm$^5$, SVR med: 1200 dynes s/cm$^5$ and SVR high: 2000 dynes s/cm$^5$) on pressures and flow at varying degrees of PVR levels. LAP: left atrial pressure; MPAP: mean pulmonary arterial pressure; RAP: right atrial pressure; CO: cardiac output.](image-url)
FIGURE 3. Effect of blood volume (BV low: 5300 mL, BV med: 6100 mL and BV high: 6900 mL) on pressures and flow at varying degrees of PVR levels.

FIGURE 4. Effect of cardiac contractility, i.e., increased left ventricular contractility (LVC), severe biventricular failure (BVF) and increased right ventricular contractility (RVC) on pressures and flow at varying degrees of PVR.
Improvement in LV contractility caused a significant increase in CO and MAP at normal PVR, however with increasing PVR they dropped substantially.

**Sensitivity Analysis of VAD Speeds Upon Hemodynamics at Varying Levels of PVRs**

Figures 5 and 6 show the pressures and flows with varying RVAD and LVAD speeds at baseline PVR, moderate and severe PH using a fixed outflow banding. LAP and MPAP increased, while RAP decreased with increasing RVAD speed. The opposite trend was observed with increasing LVAD speed. Compared to RVAD speed, increasing LVAD speed has a more substantial effect on CO during baseline condition and moderate PH. On the contrary, RVAD speed affected CO more substantially at a severe PH (high PVR).

All variables showed a change in trend (slope) at “knee points” corresponding to the transition between partial assistance and full assistance. This occurred earlier as PVR increased (i.e., RVAD speed: 2600 rpm for severe PH; 2800 rpm for baseline PVR and moderate PH; LVAD speed: 2000 rpm for severe PH; 2200 rpm for baseline PVR and moderate PH). Compared to partial assistance, the degree of change in all variables with an increase in VAD speed increased during full assistance. Comparing between the varying PVR levels, the rate of MPAP and RAP change with RVAD speed increased as PVR elevates, leading to pulmonary congestion at 2600 rpm during severe PH. Similarly, LAP fell more rapidly with increasing LVAD speed during severe PH, leading to suction at 2600 rpm.

A comparison between LVAD and RVAD flows with changing VAD speeds at baseline PVR, moderate and severe PH was illustrated in Fig. 7. LVAD flows remained relatively constant with RVAD speed during partial RVAD assistance, and showed a minor increase with increasing RVAD speed during full RVAD assistance. Similar observations were found with regards to the effect of LVAD speed on RVAD flows. On the contrary, RVAD flow increased considerably with increasing RVAD speed, with a reduced rate of increase during full RVAD assistance. Similarly, LVAD flow showed a steep increase with increasing LVAD speed at partial assistance, with a lesser degree of increase during full assistance. The severity of PH had a negligible effect on the dependency between RVAD and LVAD flows with their respective speeds during partial assistance. During full assistance, reduced VAD flows especially the LVAD flow was observed during severe PH.

![Figure 5](image-url)  
**FIGURE 5.** Effect of RVAD speed variation on pressures and flows at baseline PVR (200 dynes s/cm$^5$), moderate PH (400 dynes s/cm$^5$) and severe PH (600 dynes s/cm$^5$) using a fixed outflow banding.
FIGURE 6. Effect of LVAD speed variation on pressures and flows at baseline PVR, moderate PH and severe PH using a fixed outflow banding.

FIGURE 7. Effect of LVAD and RVAD speed variation on left (LVAD flow) and right pump outflows (RVAD flow) during baseline PVR, moderate PH and severe PH using a fixed outflow banding.
DISCUSSION

In the present study, we have performed parameter variations on a CVS-BiVAD model using a fixed right outflow banding to extend our understanding of the interaction between the CVS and the BiVAD while undergoing mild to severe PH. Different perturbations, in terms of cardiac contractility, SVR, PVR and BV, were performed to span the wide physiological ranges possible in heart failure patients. Pump speed changes required to accommodate these physiological variations were investigated. These are not easily reproducible in acute animal heart failure models due to the difficulties in controlling and altering certain CVS parameters in such preparations. As most published clinical cases recommended the use of 5 mm RVAD outflow banding diameter for patients, this was the diameter chosen for the present study.

Elevated PVR is common in patients with end stage biventricular heart failure. During the post-operative period, a steady decline of PVR (improved PH) is frequently observed in these patients, however the rate of this decline is unpredictable. Moreover, a sudden drop in PVR has been reported following extubation, leading to an increase in RVAD flow and the onset of pulmonary edema when both VADs were operated at the same speed, as observed in our simulation results without using a RVAD banding graft (Table 2). With the banded outflow graft under baseline SVR and BV conditions, we found that all hemodynamic results fell within the recommended range up to a PVR of 500 dynes s/cm² (Fig. 2). At excessive PVR (i.e., 600 dynes s/cm²), however, LV filling was substantially reduced, and a slight increase in RVAD speed or a minor decrease in LVAD speed was required to increase LAP above its lower threshold limit.

The onset of ventricular suction and pulmonary congestion varies substantially with changing SVR and BV. As reported by Saed et al., and demonstrated in our simulation studies (Fig. 2), systemic hypertension, frequently occurring in severe heart failure patients, caused a reduction in LVAD flow and subsequently resulted in pulmonary congestion. Since both VADs were operated at the midrange speed initially, restoration of LAP to its safety range was possible by reducing the RVAD speed further within the recommended operating speed region. However, precaution has to be taken to ensure that CO stays above the lower threshold limit. On the other hand, during severe PH state, risk of LV suction was prevalent during systemic hypotension due to low SVR, which might occur following cardiac surgery. The low LAP and MAP could be compensated by increasing RVAD speed, while keeping in mind that the concomitant increase in MPAP should not exceed 40 mmHg to guard against hypertension as indicated by previous findings. Furthermore, post-operative bleeding (low BV) during biventricular support further exacerbated this complication by exposing both ventricles to suction events (Fig. 3). Although adjustments of VAD speeds could help to minimize the risk of suction, our simulations demonstrate that blood transfusion is required during severe blood loss, as a speed alteration aimed to minimize the risk of suction in one ventricle would increase the chance of suction in another.

It has been reported clinically that gradual normalization of the ventricular contractility could happen following the placement of a VAD. Our simulation results demonstrated that isolated right heart recovery may aggravate pulmonary congestion, as the failing LV could not accommodate the resultant increase in the right-sided flow (Fig. 4). In this case, the RV can be partially supported by the RVAD with reduced pump speed. Weaning from the RVAD can be performed after the RV function has recovered sufficiently during the post-operative period, allowing treatment with an isolated LVAD therapy.

A major finding of this study highlighted the importance of the transition point from partial to full LVAD/RVAD assistance on the sensitivity of the hemodynamic variables to changes in LVAD/RVAD speed. Our simulation results (Figs. 5 and 6) suggested that total CO was comparatively less sensitive to changes in VAD speeds during partial assistance. Increasing VAD speeds elevated their corresponding pump flow and thus lowered the ventricular preload, leading to a reduction in the native heart contribution through the Frank-Starling mechanism. As a result, total CO showed a minor increment during partial assistance, subsequently affecting the change in other hemodynamic variables with VAD speed. Although increasing RVAD speed from 1800 to 3000 rpm produced a substantial increase in RVAD flow (Fig. 7), only small changes were observed in CO (i.e., 8% at baseline, 10.6% at moderate PH and 17.4% at severe PH, as shown in Fig. 5). Based on previously published experimental findings obtained under a non-restricted setting (i.e., without banding), the percentage of CO increase due to the RVAD speed ranged between 13 and 30%. Our further investigation (not shown) revealed that the usage of a banded outflow graft significantly reduced the percentage of CO increase with an increase in RVAD speed. In comparison to a non-restricted setting, restricting the diameter of the right outflow graft reduced the contribution of the RVAD as the blood was more inclined to flow through the less resistant pulmonary valve, thereby delayed the transition from partial assistance to full assistance. Apart from the restriction, the percentage increase in CO
with RVAD speed was also affected by the relative contractility between the left and the RVs. For instance, depressed LV contractility with a limited Starling response further reduced the percentage increase in CO with RVAD speed, as increased RVAD speed may cause RV suction and pulmonary congestion, which prevents further increase in the total CO.

One limitation of the present study is that simulations of various physiological conditions were performed in the absence of auto regulatory and reflex mechanisms. In addition, while SVR and PVR are known to be highly non-linear and depend on various factors,9,24,28 we have chosen to use linear representations for both resistances at this stage. Since the effect of SVR and PVR on overall hemodynamics formed an important part of analysis in the present paper, inclusion of non-linearities in the resistances would substantially complicate the interpretation of the results. Further experimental work which perturbs the CVS to a wide range of operating points, e.g., by performing afterload, preload and pump speed changes, are required for more accurate representation of the resistances.

CONCLUSION

In this study, we have investigated the applicability of a fixed right outflow banding under various circulatory conditions. Our simulation results showed that the introduction of a banding graft with a 5 mm diameter guaranteed sufficient reserve of the pump speed spectrum for the regulation of acceptable hemodynamics over different clinical scenarios, except at extreme conditions where drug administration or volume management is required. With a greater flexibility in pump speed variations, further optimization of the biventricular support through implementation of advanced control systems could be carried out in accordance with the post-operative conditions of the patients.

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