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Optimization of the Assisted Bidirectional Glenn Procedure for First Stage Single Ventricle Repair

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Abstract

Background: First-stage single-ventricle palliation is challenging to manage, and significant interstage morbidity and mortality remain. Prior computational and in vitro studies of the assisted bidirectional Glenn (ABG), a novel first-stage procedure that has shown potential for early conversion to a more stable augmented Glenn physiology, demonstrated increased pulmonary flow and oxygen delivery while decreasing cardiac work, as compared to conventional stage-1 alternatives. This study aims to identify optimal shunt designs for the ABG to improve pulmonary flow while maintaining or decreasing superior vena caval (SVC) pressure.

Methods: A representative three-dimensional model of a neonatal bidirectional Glenn (BDG) was created, with a shunt connecting the innominate artery to the SVC. The shunt design was studied as a six-parameter constrained shape optimization problem. We simulated hemodynamics for each candidate designs using a multiscale finite element flow solver and compared performance against designs with taper-less shunts, the standalone BDG, and a simplified control volume model. Three values of pulmonary vascular resistance (PVR) of 2.3, 4.3, and 7.1 WUm² were studied.

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Supplemental Material

Supplementary material for this article is available online.

Results: Increases in pulmonary flow were generally accompanied by increases in SVC pressure, except at low PVR (2.3 WUm²), where the optimal shunt geometry achieved a 13% increase in pulmonary flow without incurring any increase in SVC pressure. Shunt outlet area was the most influential design parameter, while others had minimal effect.

Conclusion: Assisted bidirectional Glenn performance is sensitive to PVR and shunt outlet diameter. An increase in pulmonary flow without a corresponding increase in SVC pressure is possible only when PVR is low.

Keywords

congenital heart surgery; computer applications; pulmonary vascular resistance; cavopulmonary anastomosis

Introduction

Single-ventricle physiology comprises a family of complex congenital cardiac malformations, where the circulation is driven by only one functional ventricle. Palliative care for this physiology typically involves a three-stage procedure, culminating in the Fontan circulation.^{1,2} First-stage palliation is typically performed within the first few days after birth. This stage establishes a source of pulmonary blood flow via a systemic-to-pulmonary connection, commonly achieved via placement of a modified Blalock-Taussig shunt (mBTs).^{3,4} Subsequently, the second and third stages remove the systemic-to-pulmonary shunt, and reroute the venous return from the upper and lower body directly to the pulmonary arteries (PAs). Despite improvements in surgical methods and clinical management, outcomes of the mBTs placement remain unsatisfactory,⁵ with stage-1 mortality rates approaching 30% at some centers. Since the systemic-to-pulmonary shunt is in parallel to the systemic circulation in stage-1 physiology, cardiac output (CO) is nearly doubled, increasing the volume load on the single ventricle. The conduit used for the shunt is typically synthetic, making it susceptible to thrombosis, which can lead to sudden death.⁶

Recently, the assisted bidirectional Glenn (ABG) was proposed as a novel alternative for using a systemic-to-pulmonary shunt as the source for pulmonary blood flow in stage-1 palliation.⁷ The ABG procedure would adopt a superior cavopulmonary connection (SCPC), as in a conventional bidirectional Glenn (BDG) anastomosis, as the primary source of pulmonary blood flow, with an additional systemic-to-SCPC shunt implanted by connecting the innominate artery and superior vena cava (SVC). This shunt serves two purposes: one, it acts as a source of accessory pulmonary blood flow and two, as an *ejector-pump* by supplying a low-volume but high-energy jet to drive the low-energy pulmonary flow. The kinetic energy from the high-energy jet is converted to potential energy, with the potential of increasing flow downstream *without* elevating pressure upstream. The notion of a systemic ejector pump that accelerates the venous return was also explored previously⁸ in the context of accessory blood flow supplied directly to the PAs but was hampered by unacceptably high SVC pressures. By adopting a highly restricted flow from the innominate artery to the SVC, instead of to the branch PAs, we have previously demonstrated acceptable SVC pressures concomitant with improved systemic oxygen saturation.^{7,9}

The potential benefits of the ABG procedure, compared to the traditional mBTs, include a reduced load on the functioning ventricle (the flow through the shunt would be highly restrictive to produce the high-energy jet), the potential to reduce the three-stage palliation to two stages, a more stable pulmonary blood flow with an SCPC circulation, and removal of the morbidities associated with mBTs shunt thrombosis following traditional stage-1 palliation. However, despite the benefits in achieving better pulmonary blood flow and oxygen saturations as compared to the BDG, the ABG also resulted in increased SVC pressure under high pulmonary vascular resistance (PVR) conditions.

The objective of this study is to examine the potential for improving the flow and hemodynamics of the ABG through engineering design optimization. Our ABG design objectives are to increase the pulmonary flow, and decrease or maintain SVC pressure, within clinically acceptable levels. We consider two broad parameter families that affect these objectives: (1) physiological conditions such as the distal vascular resistance and (2) geometric parameters that define the shunt shape. We also discuss the effect of the ABG on other factors of clinical importance, such as oxygen delivery and systemic and mixed venous oxygen saturations. Finally, we infer design parameters with the highest hemodynamic impact and discuss design considerations and challenges that need to be addressed in order to allow clinical translation of the ABG.

Methods

Model Construction and Shunt Parametrization

The three-dimensional (3D) geometric models are comprised of cylindrical idealizations of the aortopulmonary geometry, based on our former ABG study,⁷ and a parametrized converging systemic-to-SCPC shunt (Figure 1). Each model includes a circular aortic arch, four upper branches, two coronary arteries, the SVC, the two PAs, and the systemic-to-pulmonary shunt. The 3D idealized geometries were created using Sim-Vascular,^{10,11} an open-source pipeline for model construction and hemodynamic simulation. Model construction was automated using scripts so as to expedite the parametric study, following our previous work.^{12,13} The SVC diameter is set to 4.2 mm and the PA diameters are set to 4.8 mm, based on clinically derived geometries from our previous work.⁷ The BDG was constructed as an end-to-side anastomosis between the SVC and right PA. The cardiopulmonary geometry is held fixed, while the shape of the shunt is systematically varied as described in the subsequent paragraphs.

The systemic-to-SCPC shunt is modeled as a converging conduit with a sigmoidal taper between the innominate artery and the SVC, cephalad to the BDG anastomosis. The rationale behind adopting a tapered shunt is based on the ejector pump principle: as a high-pressure stream flows through a tapered conduit, its potential energy is converted into kinetic energy,⁷ which is then delivered into the SCPC. The shunt is parametrized using six geometrical parameters (Figure 2), with simple box constraints (Table 1). For the purpose of optimization, we normalized the parameters based on their bounds, that is, if ξ represents the six-dimensional vector of normalized parameters, then ξ belongs to the set $[0,1]^6$.

The graft centerline is generated using a Hermite spline from the innominate root to the SVC at a distance l_{PA} from the junction. The diameter d of the shunt as a function of the normalized location x along the shunt centerline, starting from innominate ($x = 0$) and ending at the SVC ($x = 1$), is given by the relation:

$$d = \begin{cases} d_i & 0 \leq x \leq l_e - t_w \\ d_i + (d_f - d_i) \alpha(x; t_w, l_e) & l_e - t_w \leq x \leq l_e \\ d_f & l_e \leq x \leq 1 \end{cases}$$

where $\alpha(x; t_w, l_e)$ is given by

$$\alpha(x; t_w, l_e) = \frac{\exp\left(5 - \left(\frac{10(l_e - x)}{t_w}\right)\right)}{1 + \exp\left(5 - \left(\frac{10(l_e - x)}{t_w}\right)\right)}.$$

Based on the above definitions, the shunt has a maximum diameter d_i , a minimum diameter d_f and a sigmoidal taper defined by $\alpha(x; t_w, l_e)$. t_w is the fraction of shunt length devoted to the contraction of the shunt: a small t_w implies a sharper transition from the maximum diameter to the minimum diameter. The length of the nozzle (normalized by total length of the graft) is given by $(1 - l_e)$. The shape of contraction has two key effects on the flow within the shunt. First, the diameters and the rate of contraction directly affect the resistance of the shunt. Second, the contraction offers a mechanism for converting the high systemic pressure energy into kinetic energy. θ_{SVC} is the angle between the SVC centerline and shunt centerline at the SVC-nozzle junction. Hence, θ_{SVC} affects the proportion of jet momentum injected coaxially into the SVC. l_{PA} is the distance from the shunt anastomosis to the BDG junction and thus determines the mixing length of the jet entering the SVC flow stream. In context of a taper-less, “straight” shunt, only three parameters are needed for describing the geometry: the diameter of the shunt ($d = d_f = d_i$), θ_{SVC} , and l_{PA} .

Multiscale Simulations

We adopt a previously well-described multiscale framework¹⁴ that couples local hemodynamics within the ABG to a closed-loop lumped parameter model of the circulatory physiology. The computational domain is decomposed into two components: (1) a 3D anatomic model where the Navier-Stokes equations are solved and (2) a 0-dimensional (0D) lumped parameter network (LPN) representing the physiologic response of the heart and closed-loop circulation. An efficient multidomain approach is used to numerically couple the 3D and 0D domains.¹⁴ We adopt the LPN structure and individual component values from our previous ABG study.⁷ Separate blocks model the upper body, lower body, pulmonary bed, coronary circulation, and heart. These blocks are partitioned into arterial bed, capillary bed, and venous bed sections. The venous return from the upper body is directed to the SVC. In the 3D domain, we assumed an incompressible Newtonian fluid and rigid walls, with a heart rate of 120 bpm and blood density and viscosity of 1,060 kg/m³ and 0.004 Pa-s,

respectively. Simulations were performed using an in-house finite-element solver¹⁵ with a time step size of 0.5 milliseconds. To prevent divergence of simulations caused by physiological reversal of flow at the boundaries, we make use of stabilized formulations.¹⁶ All results for flow rate and pressures were compared against a standard BDG model without the shunt, following standard surgical practice. All simulations were run for six cardiac cycles to allow cyclic convergence and the sixth cardiac cycle was used for this study.

To construct the computational mesh, we used an automated mesh-generation strategy with local refinement at the shunt and the cavopulmonary geometry distal to the shunt. To determine this procedure, we performed mesh convergence studies on a shunt geometry defined by maximum values of d_i and d_f and mean values of the rest of the parameters (given in Table 1). We found that a mesh size of 1.6 million elements was sufficient for obtaining average vena caval pressure and pulmonary flow within 5% for this geometry. Typically, each mesh has 1.2 to 1.6 million elements.

Optimization Setup and Methodology

For a given distal vascular resistance, we aim to identify nozzle designs that maximize pulmonary flow Q_{PA} while avoiding high SVC pressures P_{SVC} . We pose this as a constrained optimization problem in terms of the normalized design parameters ξ and a limiting pressure P_0 :

$$\begin{aligned} & \underset{\xi}{\text{maximize}} \quad Q_{PA}, \\ & \text{subject to } P_{SVC} \leq P_0; \quad 0 \leq \xi \leq 1. \end{aligned}$$

Both P_{SVC} and Q_{PA} were time averaged over a period of one cardiac cycle. Furthermore, P_{SVC} is the area-averaged pressure at the SVC inlet of the 3D model. Optimization was performed for three values of PVR: 2.3, 4.3, and 7.1 WUm², with values of P_0 set to 7.5, 12.5, and 15 mm Hg, respectively. Since the distal vascular resistance has a large impact on vena caval pressure, setting a fixed target pressure across all PVR values is not suitable for finding a sequence of designs, which improve SVC pressure. We set the pressure constraints based on the tenth percentile of a latin hypercube sample (LHS; described below) of our design space at a given PVR.

Due to the high computational cost of each 3D multiscale simulation (approximately 24 hours on a parallel 24 × 2.6 GHz processor machine), we chose a method that is efficient at exploring the six-dimensional design space for the graft. Factors considered in our choice of optimization method included (1) well-distributed coverage of the design space, (2) adaptive resolution in regions of high-performing designs, (3) rigorous convergence properties, and (4) allowing black-box constraints.

We utilize a constrained variant of the surrogate management framework (SMF),^{17,18} a derivative-free optimization method that satisfies all the above properties. The SMF algorithm builds upon a class of pattern search methods which have an established convergence theory.^{17,19} The SMF typically cycles between a search step and a poll step.

The search step utilizes a Kriging surrogate function to globally identify regions that can improve the cost function or the constraint value. On the other hand, the poll step evaluates a set of mesh points neighboring the current best evaluation. The SMF is nonintrusive, which makes it well suited for solvers that are treated as a black box. The SMF is coupled to modules for model generation, meshing, hemodynamics simulation, and postprocessing in an automated loop via a script. To increase efficiency, multiple design candidates were evaluated in parallel for both search and poll. Similar automated frameworks have been applied previously to other cardiovascular optimization problems.^{12,13,20–22}

To initialize the optimization, we used an optimal LHS²³ of 72 design points. Latin hypercube sample is a stratified random sample across the design space, which ensures uniform coverage and provides a good initialization for the optimization procedure. Having a sizable stratified random sample in the candidate set of evaluations also allows for qualitative assessment of global sensitivity to changes in geometry. The same sampled LHS points were simulated for all three PVR values, and resultant flows and pressures were extracted for initializing the optimization and determining the pressure constraints. This uniform LHS sample was also used for assessing variation in P_{SVC} and Q_{PA} for low and high levels of each parameter. To do this, we compared the sample averages of P_{SVC} and Q_{PA} for designs that lie in the lower and upper quartiles for each parameter.

Parametric Studies

Five parametric studies were also performed for taper-less, straight ABG shunts at the three PVR levels to supplement the optimization results. For each parametric study, values of θ_{SVC} and I_{PA} were fixed and seven diameters were tested from 0.6 to 2 mm. A summary of these parametric studies is given in Table 2. Of the five studies considered, (ii) and (iv) were found to perform best and worst, respectively, and these were compared against the optimization results.

Idealized Ejector Model

For comparison, we also developed a descriptive reduced order model that captures the “ejector pump effect” in a quasi-1D control volume model of the BDG junction in isolation from the surrounding physiology. The proximal end of the control volume is the SVC, and the distal end is a lumped representation of the PAs. The extremities of the control volume are attached to lumped, 0D representations of the SVC, aorta, and pulmonary vasculature. The model is described in detail in Appendix A.

The reduced order model assumes (1) steady SVC flow, (2) steady and independent aortic pressure, (3) negligible viscous losses due to the vessel walls, and (4) negligible head losses due to impingement of shunt jet onto the SVC wall and impingement of the augmented SVC flow onto the pulmonary wall. The unknowns in this model are SVC pressure, PA pressure, total pulmonary flow, and shunt flow. This model represents an upper bound on the ejector performance of the shunt, which will most likely be diminished in practice due to mixing losses, viscous friction on the vessel walls, and the head losses at the vascular junctions. It must be noted that this model is designed to understand the ejector pump effect under physiological pressures and flow rates, and hence, it is unsuitable to study other clinically

important quantities such as heart load and oxygen saturations. Finally, this model does not account for interaction of the pulmonary hemodynamics with the full circulatory physiology, or the effect of three-dimensionality of the ABG geometry.

Results

Relation Between SVC Pressure and Pulmonary Flow

Figure 3 shows a compilation of overall performance of all simulated designs from the optimizations, as well as performance of two types of straight shunt and the shunt-less BDG, for the three PVR values. Hemodynamic simulations for over 160 design shapes were run for each PVR value. We first consider the variation in SVC pressure with increase in pulmonary flow at a given PVR value. In general, there is an increase in SVC pressure along with an increase in pulmonary blood flow. The best performing shapes lie on the top-left frontier of the evaluated point clouds, that is, designs that give larger pulmonary flow at a given SVC pressure are deemed “optimal.” Ideally, we aim to increase pulmonary flow while decreasing or maintaining SVC pressure, compared to the BDG. However, only a small number of designs at low PVR show an increase in flow without an increase in pressure relative to the BDG. Indeed, a large majority of ABG shunt designs show “inadequate” performance for all tested PVRs. The frontier of best performing designs is nonlinear: for a small initial rise in SVC pressure, a steep increase in pulmonary flow can be achieved. At $PVR = 2.3 \text{ WUm}^2$, where Q_{PA} can be increased by 13% while incurring negligible changes in P_{SVC} . If we allow a 5% increase in P_{SVC} , pulmonary flow may be increased by nearly 10% for all PVR values. However, designs with greater than 25% increase in pulmonary flow incur greater than 20% increase in SVC pressure at all PVRs.

We now consider the effect of increasing PVR on the SVC pressure and pulmonary flow. Pulmonary vascular resistance strongly affects both quantities, as it elevates pulmonary pressures and restricts flow to the distal pulmonary vasculature. Pulmonary vascular resistance also influences the overall spread in performance due to geometry: ABG designs at lower PVR exhibit a larger scatter of achievable pulmonary flow for a given increase in SVC pressure. On the other hand, simulations at $PVR = 7.1 \text{ WUm}^2$ display a tighter and more linear trade-off between the two quantities. This shows that the coupling between SVC pressure and pulmonary flow increases at higher PVRs.

Comparison Against Idealized ABG Model

Figure 4 compares the variation in SVC pressure with pulmonary flow at the three PVRs obtained from the idealized model against the variation obtained for a straight shunt at $\theta_{SVC} = 20^\circ$ and $l_{PA} = 0.6 \text{ cm}$. There is qualitative agreement in the behavior of SVC pressure with change in pulmonary flow for the two computational models for small increases in pulmonary flow. The difference between the idealized model and the simulation increases rapidly for larger increases in pulmonary flow. The idealized model exhibits reduced SVC pressures for a moderate increase in pulmonary flow. This is typical in the context of an ejector pump, which was the basis for the design of the ABG. However, a comparable decrease in SVC pressure is not reproduced by the ABG at any of the PVRs. This indicates that the role of viscous losses, as well as changes in global hemodynamic parameters, such

as upper-body venous return and aortic pressure, becomes more prominent at larger shunt flow rates.

To find regions of high viscous losses within the ABG, we computed the rate of viscous dissipation within the BDG and two high-flow ABG shunts (Figure 5). The region immediately distal to the shunt-SVC anastomosis and the wall of the PA have high viscous losses. This suggests that modification to the shunt-to-SCPC anastomosis and the cavopulmonary junction may reduce the gap between ideal ejector and actual ABG performance.

Effect of Outlet Diameter on SVC Pressure and Pulmonary flow

Figures 6A and B summarize the effect of the end shunt diameter d_f . Figure 6A shows that SVC pressure follows a tight, near monotonic relationship with the outlet diameter of the graft for all PVR values. This indicates that of the six geometrical parameters tested, d_f dominates as the determining factor for rise in SVC pressure at a given PVR. For a fixed d_f , the other five parameters have the largest effect at low PVR and high d_f , where they account for up to 2 mm Hg (approximately 20%) change in SVC pressure. The contribution of the other five parameters is much smaller for smaller diameters and higher for PVR. A similar but weaker relationship exists between pulmonary flow and d_f as seen in Figure 6B. A larger variability in pulmonary flow, due to variation in the other five parameters, is seen at lower PVR and nearer to the upper bound of the outlet diameter. Conversely, at higher PVRs, the pulmonary flow can be almost completely determined by the outlet diameter of the graft.

Effect of Other Parameters on SVC Pressure and Pulmonary Flow

As established in Figure 6, the other five geometric parameters can have a considerable effect on pulmonary flow. To highlight the effect of these parameters on Q_{PA} , consider the following pairs of maximum-flow tapered shunt designs and straight shunt designs: (b,c), (e,f), and (h,i) in Figure 3. Members of each pair of designs have nearly equal end diameters d_f (within 0.02 mm). However, the tapered shunt exhibited a larger allowable Q_{PA} . Furthermore, for each pair, the tapered designs exhibit equal or slightly lower SVC pressure when compared to their straight counterparts. This provides initial evidence that the other parameters play a significant role in Q_{PA} . The effect of the other design variables is also visible in the two straight shunt configurations considered in Figure 3, as the tested shunt diameters are the same for the two configurations. The (20°, 6 mm) configuration performs better than the (80°, 6 mm) configuration, especially at PVR = 2.3 WUm².

Unlike d_f , assessing the effect of these parameters is more complicated. However, we can infer a general trend for θ_{SVC} using the initial LHS sample drawn before optimization. We extract two groups of samples per parameter: the upper quartile ($x^* = 0.75$) and lower quartile ($x^* = 0.25$) of each parameter from the initial LHS sample. Intergroup differences are tested using a two-way t test for both P_{SVC} and Q_{PA} . This categorical approach allows us to detect any dominant linear effects at the extreme values of each parameter, and hence, we aim to address the differences that arise by altering one parameter from one end of the parameter bounds to the other (eg, testing differences between shapes that have “small”

θ_{SVC} vs shapes with “large” θ_{SVC}). The group averages of P_{SVC} and Q_{PA} are presented in Figure 7. As anticipated, there is a significant difference ($p < 0.05$) in P_{SVC} and Q_{PA} at low and high d_f for all three PVR values. θ_{SVC} also has a significant effect on Q_{PA} ; a large θ_{SVC} is associated with lower values of Q_{PA} . None of the other parameters show a significant difference in sample averages for both P_{SVC} and Q_{PA} , although initial diameter d_i is observed to have a weak positive relationship with Q_{PA} ($p \approx 0.1$) at $PVR = 2.3 \text{ WUm}^2$. The favorability of small θ_{SVC} over larger values is also seen in Figure 8, where three taper-less shunt designs with the same l_{PA} are compared.

Effect on Circulatory Physiology

Figure 9 compares the effect on circulatory physiology for the BDG, straight shunt, and two tapered shapes. These shapes were chosen as representative high-performance shapes for d_f approximately equal to 1.3 and 2 mm, as marked in Figure 3. For comparison, the same quantities are also presented for the BDG and the maximum flow state of the best performing straight shunt ($d_f = 2 \text{ mm}$, $\theta_{SVC} = 20^\circ$, and $l_{PA} = 6 \text{ mm}$). There are three trends that may be observed between the BDG and the optimal ABG models for any PVR: (1) the Q_p/Q_s ratio and pulmonary saturations are enhanced due to the addition of systemic flow. Large pulmonary flow increases are associated with a larger increase in systemic and pulmonary saturations. This is consistent with our previous study⁷ and shows the potential benefit of the ABG; (2) the CO and single-ventricle power (CP) remain within 10% of the values obtained from the BDG. Hence, the overall ventricular loading does not change significantly due to implantation of a systemic-to-SCPC shunt; and (3) the upper body flow (Q_{ub}) and the lower body flow (Q_{lb}) decrease considerably due to implantation of the ABG. A lower systemic flow rate, albeit with a higher oxygen saturation, leads to an overall decrease in oxygen delivery as total pulmonary flow is increased.

Comment

Current stage-1 single-ventricle palliation has a number of undesirable attributes, which contribute to important challenges in clinical management, including high cardiac workload, cyanosis and sudden death due to shunt thrombosis, myocardial ischemia due to coronary steal, and difficult balance between pulmonary over-or undercirculation. Historical clinical studies^{24,25} have reported high mortality when performing the BDG in neonates. This has motivated the development of alternative stage-1 palliation techniques, such as the ABG. The ABG has several theoretical benefits over the mBTS surgery, such as redundancy in pulmonary flow source, reduced ventricular load, supply of hepatic effluents to the pulmonary bed, and increased systemic oxygen saturation. The primary drawbacks to the ABG centers around the potential for elevated SVC pressure, which has been associated with SVC syndrome, peripheral edema, and pleural effusions.

The goal of this study was to identify optimal shunt designs for the ABG that increase pulmonary flow without significantly increasing SVC pressure for stage-1 through an ejector pump effect. We performed optimization to systematically evaluate numerous tapered shunt designs, as well as parametric studies for taper-less straight shunt designs, within a multiscale simulation framework. While we identified a few designs that exhibit a mild

ejector effect, the majority of designs incurred small to moderate increases in SVC pressure. On the other hand, an idealized calculation of ABG hemodynamics indicates it is theoretically possible to reduce SVC pressure within the range of relevant physiological parameters. The discrepancy between the idealized model and 3D simulations, causing a degradation in ejector performance is likely due to viscous effects, losses due to impingement, insufficient mixing length, and three-dimensionality of geometry. These factors contribute to additional viscous dissipation within the cavopulmonary geometry, and further studies are needed to quantify the chief sources of discrepancy between the ideal ejector and the ABG, which may offer avenues for performance improvement.

Pulmonary vascular resistance has a large influence on the efficiency of the ABG. At lower PVR values, the shunt can be optimized to deliver pulmonary flow with a negligible SVC pressure penalty. At higher PVR values, the coupling between SVC pressure and pulmonary flow is stronger, and the corresponding rise in SVC pressure with increased pulmonary flow is larger. This may represent a limitation for implementing the ABG for neonates with high PVR in the early days following birth. However, with the normal physiologic falling of the PVR in the neonatal period, the ABG circulation may be achievable without clinically unacceptable pressures.²⁶ It should be highlighted that the mid to high PVR values adopted in this study were purely speculative, as there is no well-defined clinical PVR range in the neonatal physiology available in literature, as far as we are aware. It is possible that we have exaggerated, or underestimated, the high PVR in the models, which may, respectively, overestimate or underestimate the SVC pressures in the various ABG scenarios. However, with continuing maturation of PAs beyond the neonatal period, the overall ejector effect within the ABG is anticipated to be enhanced with continuing improvements in pulmonary blood flow and drop in SVC pressure. Quantifying these improvements is challenging, due to a lack of predictive models for decrease in PVR, especially with increasing pulmonary flow. Postoperative administration of pulmonary vasodilators, such as Sildenafil and Remodulin, may further increase ABG performance in the early postoperative period. For very large PVR values, pulmonary vasodilators may not provide sufficient decrease in PVR and hence, for such patients, conventional stage-1 palliation such as the modified BT shunt may be more appropriate. As there is large variability in possible values of preoperative neonatal PVR, the decision to perform the ABG as well as shunt design would require patient-specific assessment.

With reference to our previous studies on the ABG physiology,^{7,9} this study aimed to optimize the ABG shunt design. To this end, this study explores a much larger design space for the ABG shunt for three values of PVR, compared to our prior investigations. There is agreement in the hemodynamic trends found between this study and our previous studies across a much larger sample size. This study and our previous computational study related to the ABG⁷ are examples of a “virtual sand-box” for testing next generation surgical improvements, especially when suitable animal models of the physiology are not easily available. This coupled optimization-simulation framework in particular can evaluate a wide range of geometric, anatomical, and physiological parameters and offer insights into local hemodynamics and physiologic response.

Through the use of a multiscale simulation framework coupled to a suitable optimization method, this study demonstrates that the systemic-to-SCPC shunt geometry has a significant effect on the pulmonary circulation, primarily due to varying the shunt outlet diameter d_f . This is in agreement with the study by Zhou et al,⁹ which established a strong connection between the Glenn hemodynamics and shunt diameter ratio (varied by changing the outlet diameter of the shunt). As shunt resistance is extremely sensitive to changes in diameter, d_f directly affects the shunt flow rate. The increase in SVC pressure is predominantly dictated by d_f indicating that the shunt shape and position could be designed according to the patient's tolerance to rise in SVC pressure.

Besides directly affecting the ABG performance, d_f also interacts with the other geometric parameters. Low values of d_f are associated with a lower variance in the performance metrics, which indicate low sensitivity of other design parameters when the end diameter is small. θ_{SVC} affects total pulmonary flow, which is composed of upper body venous return (Q_{ub}) and systemic-to-SCPC shunt flow. Implanting the shunt nearly coaxially to the SVC enhances pulmonary blood flow. A larger angle may be associated with local flow dynamics that effectively obstruct the SVC or the shunt flow. A larger θ_{SVC} decreases momentum transfer from the jet to the upper body venous return. The other geometric variables appear to only weakly affect the simulated physiology. Hence, for any PVR, controlling for d_f and θ_{SVC} reduces most of the geometric variability introduced by the shunt. Since the parameters affecting shunt taper only weakly affect the circulation, especially under small increases of pulmonary flow, a straight shunt may perform as well as a tapered shunt. These findings provide surgical guidelines to aid in choosing shunt size and performing the shunt anastomosis, which may lead to "favorable" ABG performance without executing an optimization study for each patient.

This study finds that, within the parameter bounds considered, the systemic-to-SCPC shunt has a negligible effect on the total CO and single-ventricle work. As noted in our previous study,⁷ the mBTs physiology adds a significant burden on the single ventricle, compared to the BDG and ABG. This arises from a much larger flow rate within the mBTs, compared to the systemic-to-SCPC shunt for the ABG. Elevated systemic saturations are also observed in this study, which is an advantage of the ABG over the BDG. However, there is a slight decrease in the systemic oxygen delivery relative to the BDG, due to a decrease in blood supply to the upper and lower parts of the body. This is similar to the competing flow introduced by the mBTs in stage-1 physiology, although a smaller amount of flow is "stolen," by the shunt in the ABG physiology. This study does not consider autoregulation of aortic pressure after the implantation of the ABG shunt, which may lead to an increase in CO, overall systemic flow, and, hence, systemic oxygen delivery.

Although the ABG theoretically reduces the number of palliative surgeries from three to two, it opens the patient to the risk of subsequent interventional catheterizations, in the event of acute obstruction of the systemic-to-SCPC shunt. We believe that these events may be avoided by the administration of antiplatelet therapy, until the time when typical stage-2 physiology is tolerated by the patient. There may be benefits to allowing the shunt to remain patent until the time of the Fontan surgery, as one may also consider the ABG physiology as an alternative to the stage-2 BDG with accessory flow.²⁷ Delivery of augmented flow to the

SVC instead of either of the PAs, theoretically, would promote an even distribution of hepatic flow. While such studies provide some clinical intuition, the longitudinal benefits or pitfalls of a patent systemic-to-SCPC shunt beyond the neonatal age remain unexplored.

We recognize several limitations in our study. The cardiopulmonary physiology considered here is simplified, and hence, patient-specific physiological differences or abnormalities may affect the performance of the ABG. Patient-specific changes would also need to be reflected within the LPN model applied. This study does not model the risk of hemolysis within the shunt or thrombosis due to the addition of the systemic-to-SCPC shunt. This study also doesn't address vascular growth, adaptation, or vascular remodeling due to the addition of pulmonary flow or changes in hemodynamic demand with age. The LPN used doesn't model the autoregulatory response from the body in order to maintain aortic pressure after implantation. We believe that this response may maintain lower body systemic blood flow and, hence, increase CO after implantation of the shunt. To estimate an upper bound on how large these deviations may be, we resimulated the ABG design that had the largest pulmonary flow, with an increased blood volume that maintains mean aortic pressure. We found that the SVC pressure increased by 16%, while pulmonary flow increased by 12%. Although these differences are not small, these changes would likely be smaller in the other shunt designs, owing to a smaller deviation from baseline lower body systemic flow. Nevertheless, optimal ABG designs with an LPN tuned to account for this autoregulatory response remain unexplored. Blood is assumed to behave as a Newtonian fluid in all the multiscale simulations at a fixed viscosity, though this is generally accepted to be a reasonable assumption in larger vessels. Other computational simplifications include the assumption of rigid vessel walls and idealized (yet standard) models for the calculation of oxygen delivery and saturation. We note that despite the choice of rigid walls, the necessary compliances were accommodated in a "lumped" sense through the multiscale network. This allowed the multiscale model to replicate realistic pressure waveforms. Localized differences occurring due to a compliant vasculature may be relevant, especially when studying the stresses acting on these vessels, and, hence, vessel compliance may be considered in future studies. This study utilizes *in silico* models of the cavopulmonary connection. In order to validate these results, *in vivo* testing in animals could be performed, though nontrivial challenges arise in reproducing single-ventricle physiology in animal models.²⁸

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Appendix

By further simplifying the vena caval geometry for the ABG, we can model the momentum transfer from the systemic-to-SCPC shunt to the venous return using a control volume analysis (Figure A1). The hemodynamic assumptions underlying this model are (1) aortic pressure is steady and constant, that is, the aortic pressure is not altered by the addition of the shunt, (2) the SVC flow rate is also steady and constant, (3) vena caval resistance is negligible; (4) there are negligible head losses junction at SVC-PA junction, and (5) there is no loss in momentum due to jet impingement, viscous dissipation, and so on. We tune the baseline incoming time-averaged SVC flow to match that in the BDG at the same PVR. We also adjust the left atrial pressure P_{LA} to match the values obtained from the LPN of the multiscale BDG simulations. Simple resistors are used to model the pressure–flow relationship within the shunt and the distal vasculature. The resistance of the shunt is altered based on a Poiseuille flow assumption within the shunt.

Figure A1.

Simplified control volume overview for the assisted bidirectional Glenn.

The equation for flow into the systemic-to-SCPC shunt can be expressed as follows:

$$P_{SVC} = P_{ao} - A_s U_s R_s,$$

where U_s is the area-averaged shunt velocity. The equation for P_{PA} using the distal vascular resistance:

$$P_{PA} = P_{LA} + Q_{LA} R_{PVR}.$$

Within the control volume, the equation for conservation of mass:

$$Q_{PA} = Q_s + Q_{SVC},$$

which in terms of velocity yields:

$$U_{PA} A_{SVC} = U_s A_s + U_{SVC} (A_{SVC} - A_s).$$

For conservation of momentum, we assume the sidewall pressure exerted at the expansion shown is P_{SVC} . The equation for conservation of momentum along the downward direction can be written as follows:

$$P_{SVC} = P_{PA} + \frac{\rho}{A_{SVC}} (U_{PA}^2 A_{SVC} - U_{SVC}^2 (A_{SVC} - A_s) - U_s^2 A_s).$$

There is a “jump” in pressure between the SVC and PA due to the conservation of momentum. This pressure difference varies nonlinearly with the incoming SVC flow and shunt flow and, hence, depends on the values of aortic pressure and PVR. We use fixed point iteration for solving the set of equations. Typically, for moderate or large amounts of shunt flow and physiological time-averaged aortic pressures, this idealized model predicts a negative pressure jump, that is, $P_{SVC} < P_{PA}$.

These equations represent an idealistic lower bound on the SVC pressure for fixed values of venous return, aortic pressure, PVR, and shunt resistance. Since this model was tuned to match the SVC pressure and pulmonary flow for the BDG at zero shunt flow, the losses ignored by this idealized model stem exclusively from the effect of injection of additional pulmonary flow. Hence, more realistic geometry-specific bounds can be obtained by estimating additional viscous losses at vessel wall, head losses due to impingement of flow onto vessel walls, nonlinear resistance due to shunt taper, entry angle of the shunt into the SVC, and net area expansion in the PAs.

Abbreviations and Acronyms

ABG	assisted bidirectional Glenn
BDG	bidirectional Glenn
CO	cardiac output
mBTs	modified Blalock-Taussig shunt

P_{SVC}	time-averaged, area-averaged SVC pressure
PA	pulmonary artery
PVR	pulmonary vascular resistance
Q_{PA}	time-averaged pulmonary flow
SCPC	superior cavopulmonary connection
SVC	superior vena cava

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Figure 1.

Outline of the closed loop, multiscale network. The three-dimensional component of the network comprises the aortopulmonary geometry and the systemic-to-SCPC shunt. The remaining components of the closed-loop circulatory system are modeled by a lumped-parameter network. SCPC indicates superior cavopulmonary connection.

Figure 2.

The ABG graft parametrized by the six design variables, as defined in Table 2. ABG indicates assisted bidirectional Glenn.

Figure 3.

Summary of variation in pulmonary flow with SVC pressure for all candidates evaluated from the full six-dimensional optimizations as well as two taper-less shunt studies. A circle represents the SVC pressure and pulmonary flow obtained from a candidate point evaluated during optimization. A square represents the shunt-less (BDG) vena caval pressure and pulmonary flow. The solid lines represent the variation in flow and pressure for a taper-less shunt for variation in diameter at two configurations of (θ_{SVC}, l_{PA}) : $(20^\circ, 6 \text{ mm})$ and $(80^\circ, 6 \text{ mm})$. The former configuration is marked by $\blacktriangle$ and the latter is marked by $\star$. The labeled points are compared in further detail in Figure 7. BDG indicates bidirectional Glenn; SVC, superior vena cava.

Figure 4.

Comparison of SVC pressure and pulmonary flow rate between the ideal ejector model and straight shunt simulations (case (ii) from Table 2) at PVR = 2.3, 4.3, and 7.1 WUm². PVR indicates pulmonary vascular resistance; SVC, superior vena cava.

Figure 5.

Rate of viscous dissipation contours for (A) bidirectional Glenn; B, taper-less shunt (design (c) in Figure 3); and C, a high-performance ABG shunt (design (b) in Figure 3), at PVR = 2.3 WUm², visualized at the cavopulmonary symmetry plane. ABG indicates assisted bidirectional Glenn; PVR, pulmonary vascular resistance.

Figure 6. Effect of d_f on (A) SVC pressure (left) and B, pulmonary flow (right) for PVR = 2.3, 4.3, and 7.1 WUm². PVR indicates pulmonary vascular resistance; SVC, superior vena cava.

Figure 7.

Group averages of (A) SVC pressure and (B) pulmonary flow for the lower and upper quartiles for each parameter. The quartiles were drawn from the initial LHS sample of the domain. The error bars indicate maximum and minimum values for each group. Of the six design parameters, d_f and θ_{SVC} exhibit a significant influence (marked by *) on group means ($p < 0.05$) for Q_{PA} , while only d_f shows a significant impact on P_{SVC} . LHS indicates latin hypercube sample; SVC, superior vena cava.

Figure 8.

Variation in pulmonary flow with SVC pressure for taperless shunt designs with $l_{PA} = 6$ mm for $\theta_{SVC} = 20^\circ, 50^\circ, 80^\circ$, at $PVR = 2.3$ WUm². PVR indicates pulmonary vascular resistance; SVC, superior vena cava.

$PVR = 2.3WUm^2$													
Ref.	Shape	d_f	CO	P_{SVC}	Q_{PA}	Q_{ub}	Q_{lb}	Q_{cor}	OD_s	CP	Q_p/Q_s	Sat_S	Sat_P
-	BDG	-	1.43	6.9	0.56	0.56	0.74	0.11	0.25	0.24	0.39	0.77	0.56
a		1.36	1.47	8.1	0.67	0.46	0.67	0.11	0.23	0.22	0.53	0.80	0.63
b		2.0	1.52	9.0	0.80	0.38	0.61	0.10	0.21	0.21	0.72	0.83	0.69
c		2.0	1.46	9.1	0.73	0.42	0.62	0.10	0.21	0.20	0.64	0.82	0.66
$PVR = 4.3WUm^2$													
Ref.	Shape	d_f	CO	P_{SVC}	Q_{PA}	Q_{ub}	Q_{lb}	Q_{cor}	OD_s	CP	Q_p/Q_s	Sat_S	Sat_P
-	BDG	-	1.46	11.3	0.53	0.53	0.80	0.12	0.25	0.26	0.36	0.76	0.54
d		1.32	1.46	12.9	0.60	0.42	0.72	0.11	0.23	0.23	0.47	0.79	0.59
e		2.0	1.51	14.0	0.73	0.33	0.67	0.10	0.21	0.22	0.64	0.82	0.66
f		2.0	1.46	14.2	0.67	0.37	0.65	0.10	0.21	0.21	0.58	0.81	0.63
$PVR = 7.1WUm^2$													
Ref.	Shape	d_f	CO	P_{SVC}	Q_{PA}	Q_{ub}	Q_{lb}	Q_{cor}	OD_s	CP	Q_p/Q_s	Sat_S	Sat_P
-	BDG	-	1.42	13.6	0.46	0.46	0.83	0.12	0.24	0.26	0.32	0.73	0.47
g		1.20	1.44	14.8	0.53	0.37	0.78	0.11	0.22	0.24	0.41	0.76	0.54
h		1.98	1.42	15.0	0.61	0.25	0.69	0.10	0.20	0.21	0.55	0.79	0.60
i		2.0	1.37	16.9	0.58	0.29	0.59	0.11	0.20	0.22	0.49	0.77	0.57

Figure 9.

Hemodynamic quantities for BDG, straight shunt, and two shapes of ABG shunt (labeled in Figure 3): CO, Cardiac Output (L/min); P_{SVC} , SVC pressure (mm Hg); Q_{PA} , pulmonary flow rate (L/min); Q_{ub} , upper body flow rate (L/min); Q_{lb} , lower body flow rate (L/min); Q_{cor} , flow rate to the coronaries (L/min); OD_s , systemic oxygen delivery (mL_{O_2}/sec); CP, single ventricular power (Watts); Q_p/Q_s , ratio of Q_p to Q_s ; Sat_S , systemic oxygen saturation

(%); Sat_P , oxygen saturation in pulmonary arteries (%). ABG indicates assisted bidirectional Glenn; BDG, bidirectional Glenn.

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Table 1.

Bounds for Geometrical Parameters That Define Systemic-to-SCPC Shunt Geometry.

Parameter	Lower Bound	Upper Bound
Inlet diameter, d_i	2.0 mm	3.8 mm
Outlet diameter, d_f	0.6 mm	2.0 mm
Angle between nozzle and SVC, θ_{SVC}	20°	80°
Distance between shunt outlet and SVC-PA junction, l_{PA}	6.0 mm	12.0 mm
Relative length of entrance section to total shunt length, l_e	0.6	0.9
Relative taper length to total shunt length, t_w	0.1	0.5

Abbreviations: SCPC, superior cavopulmonary connection; SVC, superior vena cava; SVC-PA, SVC pulmonary artery.

Table 2.

Parametric Studies for Taper-Less Shunt.

Study	Diameter ($d_i = d_f$, mm)	θ_{SVC}	l_{PA} (mm)
(i)	0.6, 0.83, 1.06, 1.30, 1.53, 1.76, 2.0	50°	9
(ii)	0.6, 0.83, 1.06, 1.30, 1.53, 1.76, 2.0	20°	6
(iii)	0.6, 0.83, 1.06, 1.30, 1.53, 1.76, 2.0	20°	12
(iv)	0.6, 0.83, 1.06, 1.30, 1.53, 1.76, 2.0	80°	6
(v)	0.6, 0.83, 1.06, 1.30, 1.53, 1.76, 2.0	80°	12