Control of Respiration-Driven Retrograde Flow in the Subdiaphragmatic Venous Return of the Fontan Circulation

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Respiration can impose a profound influence on the sub-diaphragmatic venous return in the total cavopulmonary con-nection (TCPC) of the single-ventricle Fontan circulation.[1–7] The TCPC directly connects the superior vena cava (SVC) and inferior vena cava (IVC) to the pulmonary arteries, resulting in the total venous return flowing passively into the pulmonary circulation. Lacking the ventricular power source, the systemic venous pressure and the respiratory mechanics become the dominant forces for moving pulmonary blood flow. Respiration-gated magnetic resonance (MR) imaging[1–3,5–7] has revealed significant differences to the ECG-triggered acquisitions often applied in modeling studies.[5] Venous and pulmonary arterial flows increase during inspiration and decrease in expiration.[1–3] This pattern is particularly accentuated in the inferior venous return[1–7] because of the recoil of the thoracic cage during expiration and the absence of a venous valve.[2] For many patients, as inspiration wanes, both the IVC and hepatic vein (HV) flows can experience a period of retrograde motion away from the heart.

Late complications of the failing Fontan circulation include problems in the liver and gastrointestinal tract.[2,3] The idea of implanting a valve in the IVC originated with Fontan and Baudet.[8] Hsia *et al.*[2] suggested a valve to reduce hepatic congestion. Baslaim[9] and Zureikat *et al.*[10] reported reduced retrograde HV flows after xenograft conduit implantation. Prenger *et al.*[11] reported positive outcomes with porcine-valved Dacron conduits and Corno *et al.*[12] demonstrated using self-expandable valved stents in the IVC. There are only limited clinical or realistic experimental results on which to discern between the hemodynamic advantages and penalties imposed on the circulation when using a valve.

A consequence of the Fontan circulation is increased venous pressure associated with subnormal cardiac output. Systemic venous hypertension is known to lead to cirrhosis, liver failure, and portal hypertension.[13] Pulmonary vascular resistance (PVR) is cited as the single most important factor limiting cardiac output in the Fontan circulation and changes with growth and age,[13,14] but its role in subdiaphragmatic venous return is largely undocumented.[15] The retrograde to antegrade flow volumes can vary markedly between patients,[1–3,5,16] which implicates pulmonary vascular compliance (PVC) and circulation impedance.[17]

Multidomain models couple a lumped parameter (LP) network of the circulation with a higher dimensional model of the anastomosis site. Several numerical models have been developed to predict patient differences in single-ventricle physiologies,[18,19] including models with respiration.[20] Others have used LP flow loops to assess innovative cardiopulmonary assist devices.[16,21–23] The effects of TCPC flow resistance on cardiac output were reported using an LP-coupled numerical model.[24]

Vukicevic *et al.*[25] described a multidomain mock circulatory system (MCS) of the Fontan circulation, which included respiration and aortic pulsatility. We use this *in vitro* model

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to study the impact of valve therapy on subdiaphragmatic venous hemodynamics.[25] Systems-level impedances and respiration pressures are included to properly model the time-dependent response within the thorax and abdomen. We evaluate the impact of a valve on reducing retrograde volume, improving antegrade flow, and reducing venous pressures and flow power losses.

Materials and Methods

The MCS of the Fontan circulation system is a physical realization of four branches of circulation coupling an LP network with an anatomically accurate, three-dimensional TCPC test section, as shown in the schematic of **Figure 1A** and the photograph of **Figure 1B**.[25] The system is tuned to a particular physiological state using generic reference values for each impedance element scaled by body surface area (BSA) and then adjusted with available patient-specific clinical information.[18,19] A ventricular-assist device (80 cc; Excor, Berlin, Germany) develops the pulsatile aortic pressure. Atrial pressure is maintained constant. Time-dependent thoracic (intrapleural) and abdominal respiration pressures (P_{th} and P_{ab}) associated with normal breathing are applied simultaneously to compliant elements within the thoracic cavity (pulmonary, TCPC, and IVC) and the abdomen.

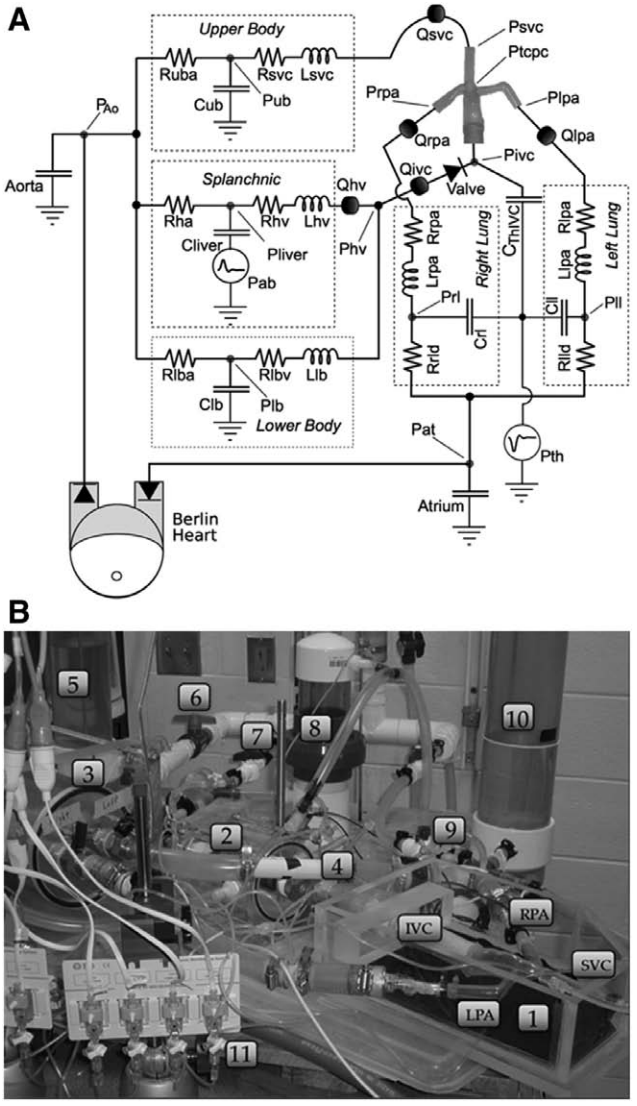

Figure 1. A: Lumped parameter model used for the mock circulatory system. **B**: Photograph of system. 1: total cavopulmonary connection (TCPC) test section, right (RPA) and left (LPA) pulmonary artery, superior vena cava (SVC); 2: inferior vena cava (IVC) compliant element; 3: pulmonary compliance elements; 4: pulmonary resistance element; 5, 10: lower and upper body compliance elements: 6, 7, 9: resistance elements; 8: splanchnic compliant element; 11: pressure transducers.

The TCPC test section is a patient-specific geometry based on MR images and realized using a thin-walled (1 ± 0.2 mm), compliant (C ~ 0.28 ml/mm Hg) silicone phantom (Shelley Medical Imaging, London, Ontario). Residual arterial and venous compliances were distributed between the surrounding compliance elements.[25] The tested valve was an 18 mm pulmonary valved conduit (CVC) (Contegra 200; Medtronic, Inc., Minneapolis, MN). When inserted, it formed part of the IVC downstream of the IVC/HV junction. Valve function was verified by borescope.

Flow rates were measured with electromagnetic probes (Carolina Medical Electronics, King, NC). Pressures were measured using liquid-filled catheters and transducers (DTXplus; BD Medical Systems, Sandy, UT). System operation was controlled using a data acquisition/control board (USB 6211/Labview; National Instruments, Austin, TX). A saline–glycerin blood analog was used (1,060 kg/m^3, 3.3×10^{-6} m^2/s at 22°C).

Flow volumes were calculated by integrating the flow toward (antegrade) or away from (retrograde) the heart during a full respiration cycle. Power loss across the TCPC test section was calculated by integrating instantaneous total pressure, P_t, and flow rate, Q, during the respiration cycle:

$$\dot{W} = (P_t Q)_{IVC} + (P_t Q)_{SVC} - (P_t Q)_{RPA} - (P_t Q)_{LPA}$$

where $P_t = P + \frac{1}{2}\rho\left(\frac{Q}{A}\right)^2$ with Q based on the cycle average.

Setup

Clinical studies report that flow pulsatility and retrograde flow volumes vary markedly between patients.[1–3,6,16] Both were varied here by changing either the pulmonary compliance or the pulmonary resistance. We established one patient-specific baseline condition in the MCS (EXP). Four additional conditions were created by increasing effectively the patient's PVC by 25% (PVC25) and 50% (PVC50) with PVR fixed and by increasing PVR (done by increasing R_{rld} and R_{lld}) by 33% (PVR33) and 90% (PVR90) with PVC fixed. The values used for the five test conditions are representative of patients having a functional Fontan.[15,21,24]

The patient modeled was a 10.8 year-old female, 7 years postlateral tunnel with no fenestration, and with BSA = 1.3 m^2. Clinical cardiac output was 3.3 L/min at a heart rate of 80 bpm, PVR of 2.1 WU, SVR of 18.2 WU, and respiration rate of 17.1 breaths/min (t_R = 3.51 s). The clinical MR velocity maps acquired were gated on respiration during a full respiration cycle.[7]

The patient-specific baseline conditions (EXP) were set by adjusting the LP element values (**Table 1**) to match clinical data. The applied thoracic and abdominal respiration pressure waveforms (**Figure 2A**) were adopted from West[26] under quiet breathing[26–28] during the respiration period t_R. The model consists of an active inspiration period ($0 < t/t_R < t_{insp}/t_R$), followed

Table 1. Values of the Lumped Parameter Experimental Model Parameters

LP Parameters	Patient Specific* BSA = 1.3 m²
Rub	3.27 ± 0.17
Cub	3.12 ± 0.05
Rsvc	0.080 ± 0.002
Rha	7.28 ± 0.23
Cliver	4.41 ± 0.14
Rhv	0.16 ± 0.005
Rlba	2.03 ± 0.12
Clb	3.86 ± 0.12
Rlbv	0.038 ± 0.002
Rrpa	0.034 ± 0.002
Rrld	0.19 ± 0.02
Crl+Crpa	2.15 ± 0.07
Rlpa	0.034 ± 0.002
Rlld	0.19 ± 0.02
Cll+Clpa	2.17 ± 0.07
Civc	2.57 ± 0.08
Ctcpc	0.28 ± 0.05

Resistances (mm Hg-s/ml); compliances (ml/mm Hg).
* 95% level of confidence.
BSA, body surface area; LP, lumped parameter.

by a passive expiration period. The thoracic and abdominal pressures, P_{th} and P_{ab}, were bounded between −1 to −5 mm Hg and 0 to 4 mm Hg, respectively.[28,29] The ascending aortic pressure applied is shown in **Figure 2B**. The mean aortic pressure was fixed at 75 mm Hg and the atrial pressure was fixed at 7 mm Hg. Baseline PVC was tuned to 4.32 ml/mm Hg with $t_{insp}/t_R = 0.4$ to meet clinical flow pulsatility.

The clinical (MR) IVC and SVC flow rates are shown with the experimental flow rates used (EXP) in **Figure 2, C and D**. The resulting antegrade and retrograde IVC flow volumes agree to within 8% and 1% and the SVC mean flow rate to be within 1% of the clinical measurements. The four additional test conditions were developed from this baseline.

Uncertainty Statement

Statistical values reported are ensemble averages during 20 contiguous respiration cycles. The uncertainty in a reported mean flow rate is ±0.7%, in differential pressure is ±0.3 mm Hg, and in retrograde flow volume is ±0.5 ml, each at a 95% confidence and evaluated as reported previously.[25]

Results

PVC Effects

Flow rate and pressure signals are compared in **Figure 3** for each case (EXP, PVC25, and PVC50) both without (baseline) and with the valve. Corresponding results are given in **Table 2**. These three baseline cases provided progressively increasing flow pulsatility, noted by Q_{retro} / Q_{ante}, with increasing IVC and HV antegrade and retrograde flow volumes while cardiac output was kept constant.

Implanting the valve decreased IVC retrograde flow volumes by 54%, 72%, and 67% (**Figure 3, A–C**, **Table 2**) compared with the respective no valve baselines. Inferior vena cava antegrade flow volumes also decreased, whereas overall IVC mean flow rates remained unchanged ($p < 0.05$). Retrograde flow volumes within the HV decreased by 5%, 33%, and 32%, respectively (**Figure 3, D–F** and **Table 2**) during baselines, whereas mean flow rates remained unchanged.

Flow and pressure waveforms were time dependent. Inferior vena cava and HV antegrade flows (**Figure 3, A–f**) were largely unchanged during the inspiration period ($0 < t/t_R < 0.4$) between corresponding baseline and valve cases. After an initial closing leakage, the valve closed blocking retrograde flow ($0.47 < t/t_R < 0.6$). The valve reopened with the resumption of

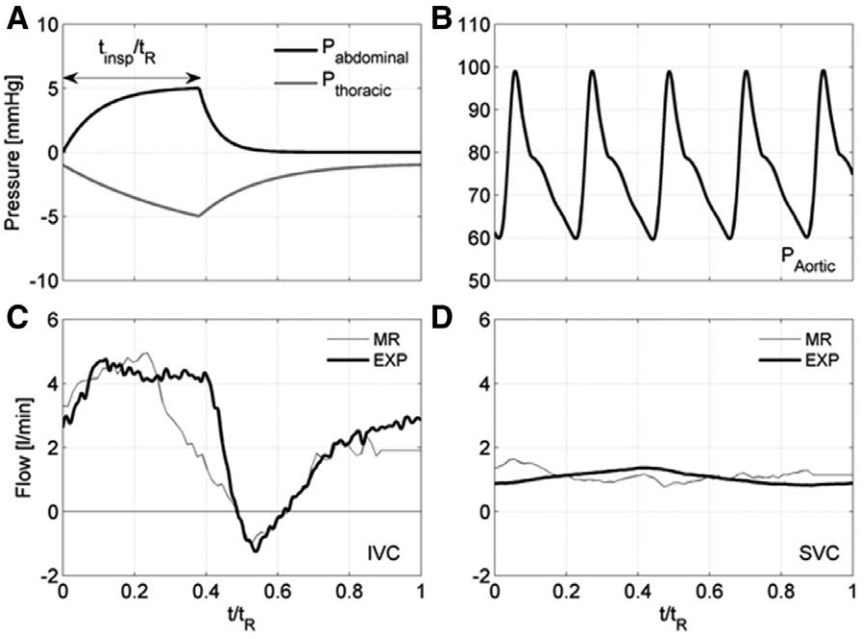

Figure 2. Waveforms used in the experiments: (**A**) respiration pressure and (**B**) aortic pressure. Comparisons of clinical flow waveforms with experimental model: (**C**) inferior vena cava (IVC) and (**D**) superior vena cava (SVC). MR, magnetic resonance.

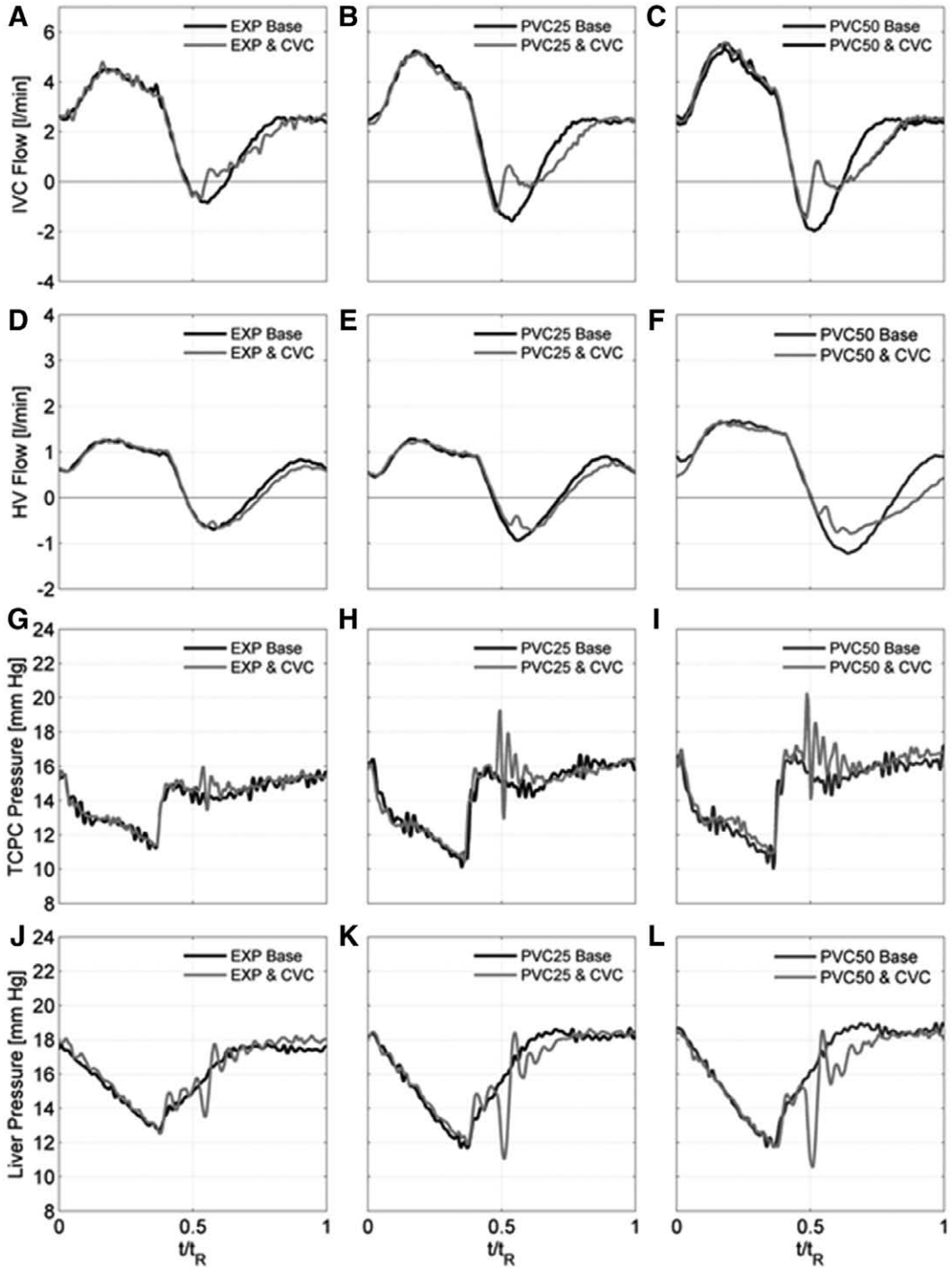

Figure 3. Hemodynamic flow and pressure signals both without (black lines) and with (gray lines) a bovine valved conduit (CVC) with changing conditions (EXP, PVC25, and PVC50): (**A–C**) inferior vena cava (IVC) flows, (**D–F**) hepatic vein (HV) flows, (**G–I**) total cavopulmonary connection (TCPC) pressure, (**J–L**) liver pressure. PVC, pulmonary vascular compliance.

antegrade flow ($0.65 < t/t_R < 1$), but the increases in flow rate during this period were reduced with the valve present. During a full cycle, IVC flow pulsatility was controlled to within 3% in each case.

Total cavopulmonary connection and liver pressures (**Figure 3, G–I**) decreased gradually during the inspiration period ($0< t/t_R < 0.4$) in each case. With the onset of expiration ($t/t_R \sim 0.4$), the IVC pressure increased abruptly, whereas the liver pressure increased more gradually. Without a valve, this developed a pressure gradient necessary for retrograde flow (**Figure 3, A–F**). The valve was closed by the retrograde volume ($0.47 < t/t_R < 0.6$) during which TCPC junction pressures increased and liver pressures decreased. Closure was accompanied by short-duration ($0.47 < t/t_R < 0.7$) pressure oscillations during which TCPC pressures increased by up to 6 mm Hg and liver pressures decreased by 5.5 mm Hg below corresponding

Table 2. Hemodynamic Effects (mean ± SD) Both with and Without a Valve

		Baseline No Valve			Contegra Valved Conduit		
	Mean ± SD	EXP	PVC25	PVC50	EXP	PVC25	PVC50
P_{TCPC}	mm Hg	14.1 ± 1.3	14.2 ± 1.8	14.2 ± 1.9	14.2 ± 1.3	14.5 ± 1.8	14.7 ± 2.0
P_{Liver}		15.6 ± 1.6	15.8 ± 2.3	15.8 ± 2.3	15.3 ± 1.7	15.3 ± 2.2	15.1 ± 2.3
Q_{IVC}	Lpm	2.25 ± 1.6	2.26 ± 2.0	2.29 ± 2.2	2.25 ± 1.5	2.23 ± 1.8	2.26 ± 1.8
Q_{HV}		0.50 ± 1.0	0.51 ± 1.1	0.53 ± 1.2	0.50 ± 0.9	0.50 ± 1.0	0.52 ± 1.0
Q_{svc}		1.14 ± 0.3	1.15 ± 0.5	1.15 ± 0.5	1.15 ± 0.4	1.17 ± 0.5	1.18 ± 0.5
CO		3.39	3.41	3.43	3.40	3.40	3.44
$Q_{ante\ IVC}$	ml/t_R	136.2	143.1	148.2	133.4	133.2	136.6
$Q_{retro\ IVC}$		4.34	10.0	13.3	2.0	2.8	4. 5
$\frac{Q_{retro\ IVC}}{Q_{ante\ IVC}}$		3.2%	7.0%	9.0%	1.5%	2.1%	3.3%
$Q_{ante\ HV}$		36.4	37.3	44.4	35.8	35.4	39.8
$Q_{retro\ HV}$		7.1	9.1	14.2	6.8	6.1	9.7
$\frac{Q_{retro\ HV}}{Q_{ante\ HV}}$		20%	22%	32%	19%	17%	24%
Power loss	mW	24.4	26.0	28.2	21.8	22.3	24.7

CO, cardiac output; HV, hepatic vein; IVC, inferior vena cava; PVC, pulmonary vascular compliance; SVC, superior vena cava; TCPC, total cavopulmonary connection.

baseline values. The valve stayed closed for the 12 to 15% of the respiration cycle consistent with conditions supporting retrograde flow.

Pressure changes across the valve are shown in **Figure 4, A–C**. With expiration, pressures on the downstream side of the valve (IVC) abruptly increased consistent with valve closing. Concurrently, pressures at the HV confluence upstream of the valve showed decreased pressure. A 2 mm Hg (root-mean-square) gradient prevailed across the closed valve. With the valve implanted, power losses (**Table 2**) across the TCPC were reduced by 10% to 12% ($p < 0.05$).

PVR Effects

Pulmonary vascular resistance was increased from the baseline of 2.1 WU (EXP) to 2.8 WU and 4.0 WU (PVR33, PVR90). With constant compliance, the amount of blood volume moved by respiration did not change between cases. Flow and pressure waveforms are compared both with and without the valve in **Figure 5, A–L** with statistical values shown in **Table 3**. Without a valve, increasing PVR augmented flow pulsatility, raised all system pressures, and cardiac output decreased. Retrograde flow volumes increased by 49% and 120% and mean system pressures increased by 10% and 29%, respective to baseline. Duration of retrograde flow lengthened from 10% (EXP) to 16% (PVR90) of a respiration cycle.

The implanted valve decreased and stabilized IVC retrograde flow volumes to within 3% of antegrade flow (**Figure 5, B and C** and **Table 3**). With increased PVR, overall IVC antegrade flow remained nearly constant (PVR33, PVR90) so that the net mean IVC flow increased. For these cases, antegrade flow during late expiration remained about

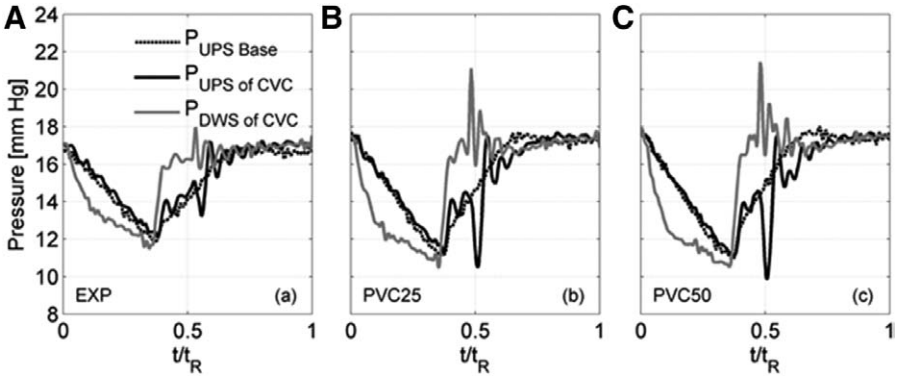

Figure 4. A–C: Measured pressures upstream (UPS; hepatic vein confluence) and downstream (DWS; inferior vena cava) of a bovine valved conduit (CVC) during one respiration cycle for three testing conditions (A, EXP; B, PVC25; C, PVC50). Pressure without valve (baseline) also shown for each case. PVC, pulmonary vascular compliance.

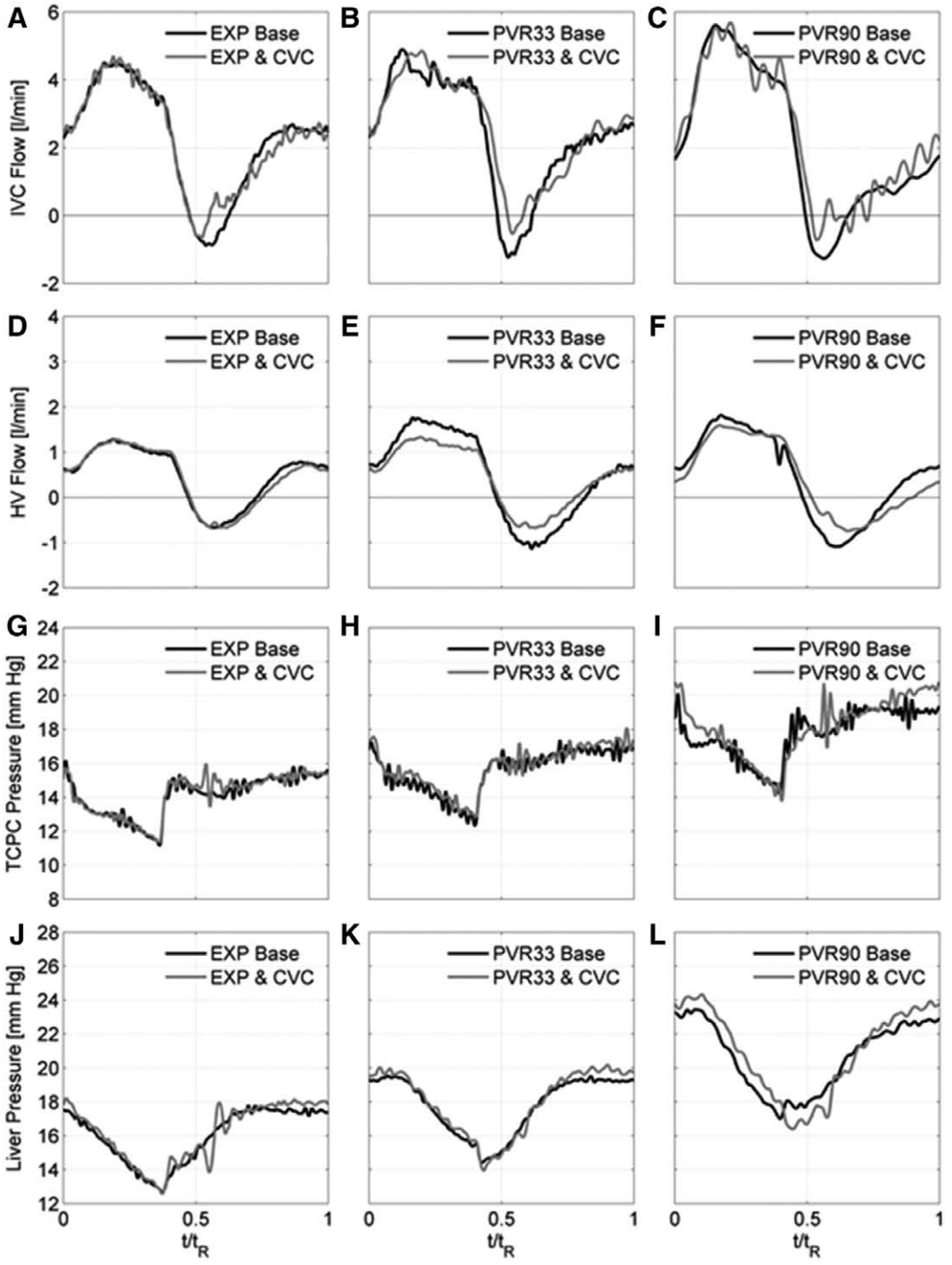

Figure 5. Hemodynamic flow and pressure signals both between without (black lines) and with (gray lines) a bovine valved conduit (CVC) with increasing pulmonary vascular resistance (PVR): (**A–C**) inferior vena cava (IVC) flows, (**D–F**) hepatic vein (HV) flows, (**G–I**) total cavopulmonary connection (TCPC) pressure, (**J–L**) liver pressure.

the same with or without the valve. Retrograde flow volumes within the HV decreased to within 20–26% of antegrade flow (**Figure 5, D–F** and **Table 3**), whereas mean flow rates remained unchanged. Cardiac output remained constant between cases with a valve ($p < 0.05$).

Cycle-averaged mean TCPC and liver pressures were essentially unchanged between valve and no valve cases ($p < 0.05$). Total cavopulmonary connection and liver pressures (**Figure 5, G–L**) decreased gradually during the inspiration period ($0 < t/t_R < 0.4$) in all cases. With the onset of expiration ($t/t_R \sim 0.4$), the IVC pressure increased abruptly, whereas the liver pressure increased more gradually.

Discussion

This study provides a system-level approach for understanding the potential outcomes of introducing IVC valve therapy into the Fontan circulation. The novel design of our

Table 3. Hemodynamic Effects (Mean ± SD) Both with and Without a Valve by Increasing PVR

		Baseline No Valve			Contegra Valved Conduit		
	Mean ± SD	EXP	PVR33	PVR90	EXP	PVR33	PVR90
P_{TCPC}	mm Hg	14.1 ± 1.3	15.6 ± 1.3	18.2 ± 1.2	14.2 ± 1.3	15.7 ± 1.2	18.6 ± 1.2
P_{Liver}		15.6 ± 1.6	17.2 ± 1.5	19.7 ± 1.1	15.3 ± 1.7	17.3 ± 1.9	20.0 ± 2.5
Q_{IVC}	Lpm	2.25 ± 1.6	2.23 ± 1.8	2.16 ± 2.1	2.25 ± 1.5	2.33 ± 1.8	2.38 ± 1.9
Q_{HV}		0.50 ± 1.0	0.48 ± 0.9	0.47 ± 0.9	0.50 ± 0.9	0.47 ± 0.7	0.46 ± 0.8
Q_{svc}		1.14 ± 0.3	1.08 ± 0.4	1.03 ± 0.4	1.15 ± 0.4	1.09 ± 0.5	1.04 ± 0.5
CO		3.39	3.31	3.19	3.40	3.41	3.42
$Q_{ante\ IVC}$	ml/t_R	136.2	136.1	134.8	133.4	138.1	141.7
$Q_{retro\ IVC}$		4.34	5.8	8.6	2.0	1.8	2.7
$\frac{Q_{retro\ IVC}}{Q_{ante\ IVC}}$		3.2%	4.3%	6.4%	1.5%	1.3%	1.9%
$Q_{ante\ HV}$		36.4	41.3	40.2	35.8	34.8	36.4
$Q_{retro\ HV}$		7.1	13.2	12.7	6.8	7.3	9.5
$\frac{Q_{retro\ HV}}{Q_{ante\ HV}}$		20%	32%	32%	19%	21%	26%

CO, cardiac output; HV, hepatic vein; IVC, inferior vena cava; PVR, pulmonary vascular resistance; Svc, superior vena cava; TCPC, total cavopulmonary connection.

system treats respiration and allows for interaction between abdomen and thorax. The system model recapitulates the respiratory-dependent flow reversal in the inferior venous return in the TCPC observed clinically.[1–7] By changing impedance values, we achieved different levels of pulsatility and retrograde blood volume with which to compare the hemodynamic benefits of using a valve. As many of the late Fontan attrition relate to hepatic dysfunction and gastrointestinal protein losing entropathy (PLE), the model not only allows for mechanistic insight into the abnormal flow circulation in Fontan patients but also provides a platform to examine the benefit of controlling or limiting the flow reversal into both the systemic inferior venous and the splanchnic circulation. The study demonstrates that there is a flow benefit with minimal pressure penalty to limiting the respiration-dependent flow reversal using clinically available valve prostheses. As future valve materials develop, our findings identify mechanisms involved and needed toward advancing valve therapy. The study establishes a clinical rationale to further examine surgical or interventional methods that can limit the observed flow reversal in Fontan patients, in particular, those who suffer from liver cirrhosis or PLE. For example, in patients with high PVR, and thus not transplantation candidates, and failing Fontan cause by PLE, the implantation of a valve in the inferior pathway may lead to a more efficient splanchnic venous return and resolution of PLE. We did not test the valve materials for thrombogenic potential and our findings are not intended as clinical recommendations.

Interactions between thoracic and abdominal respiration pressures with the various associated compliance elements were shown to influence the IVC flow direction and result in retrograde flows. The implanted valve reduced IVC retrograde flow significantly while decreasing liver pressures during the 10–16% of the respiration cycle that the valve remained closed. With the valve closed, TCPC pressures increased and liver pressures decreased. Valve closure was assisted by the inertia of retrograde flow volume. Valve closure time corresponded with the period supporting retrograde motion within the IVC, so exact times would change with breathing rhythm. The valve reduced power losses across the TCPC by a modest 10–12% during a respiration cycle. These results are favorable and the improvements more apparent in cases with higher retrograde volume.

Compliance changes served to increase pulsatility by increasing the blood volume moved by respiration pressures between successive cases. The reduction in retrograde flow associated with using a valve was found to be offset by physiological responses that also decreased antegrade flow during late expiration. This limited net gains in antegrade flow from using a valve. By incorporating the splanchnic circulation in our model, we found that the combinations of reduced retrograde flow toward the liver coupled with higher pressures in the TCPC and lower pressures in the HV, each served to reduce the driving force for antegrade flow during late expiration. This demonstrated reduced liver loading during expiration as a consequence of valve therapy. In changing PVR, the compliance and respiration pressures were fixed so that the amount of blood volume moved by respiration did not change between cases. Accordingly, late expiration behavior remained essentially the same between cases. The overall IVC antegrade flow stabilized with a valve and retrograde flow reduced. Progressive increases in PVR demonstrated increased venous pressures. Liver disease is an unfortunate consequence of the Fontan circulation due to increased venous pressures and reduced cardiac output. Unfortunately, the valve did little to reduce mean liver pressures offering only short-duration decreases during valve closure. Notably, valve therapy served to stabilize cardiac output with increasing PVR.

Corno *et al.*[12] also found that a valve implanted in the IVC of healthy adult pigs functioned better with higher retrograde

flow volumes. Santhanakrishnan *et al.*[31] used a flow loop with aortic pulsatility applied directly to the vena cava to study valve function. In their study, respiration and circulation impedances were neglected with the only compliance being a passive TCPC model. They reported that the valve functioned on the cardiac cycle with remarkable improvements in overall hemodynamics. Clinical studies report differing outcomes after implantation of a valved conduit. Baslaim[9] reported that 15 of 18 patients at 48 months showed reduction in retrograde flows, with good oxygen saturation, without thromboembolic episodes, and with good ventricular function. Zureikat *et al.*[10] reported positive results for five patients. However, Schoof *et al.*[32] reported localized thrombosis in three patients.

For a functional Fontan circulation, the retrograde flow pulsatility represented by our experimental models (Tables 2 and 3) fits within the range of published clinical data. For example, Hsia *et al.*[2,3] reported measured values of Q_{retro} / Q_{ante} of $6 \pm 11\%$ and $27 \pm 17\%$ for IVC and HV, respectively, in their respiration study of functional Fontan TCPC patients (N = 31, 14 ± 5 years). Hjortdal *et al.*[1] reported measured values (N = 11, 12.4 ± 4.6 years) within the IVC of $10.5 \pm 12.4\%$. Extending the study to the failing Fontan circulation must apply different physiological conditions than modeled here.

We found no substantial differences to these results when using different heart rates or modest changes in caval flow splits, which is not presented here because of space limitations. Although we have noted improved response to valve therapy under forced heavy breathing, a proper model of the physiological changes associated with increased metabolic activity requires substantially changing the LP values and awaits separate study.

Conclusions

The hemodynamic behavior of the Fontan circulation in single-ventricle physiology is dependent on respiration. Subdiaphragmatic flows often reverse during expiration. We used a patient-specific Fontan MCS with quiet respiration to study the effects of implanting a bovine valved conduit into the IVC. We varied system compliance and resistance to vary retrograde volume to study comparative hemodynamics during a respiration cycle.

Overall, the valve reduced the IVC retrograde flow volume to within 3% of antegrade flow volume in all cases. Hepatic vein retrograde flow volumes decreased. Improvements in antegrade flows were moderated by circulation impedances. The valve provided some relief of retrograde loading on the liver during expiration. Pressures within the TCPC increased and liver pressures decreased only during the duration of valve closure. Otherwise, mean IVC and liver pressures improved by less than 1 mm Hg during a respiration cycle. Power losses through the TCPC improved with the valve by 12–15%. For the host patient, increasing PVR increased mean system pressures regardless of whether a valve was implanted, but cardiac output stabilized with a valve. The results show that hemodynamic benefits of valve therapy will depend on patient vascular impedance and physiology.

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