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In-Vitro Evaluation of a Compliant Inflow Cannula Reservoir to Reduce Suction Events with Extracorporeal, Rotary VAD Support

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Abstract

Limited preload sensitivity of rotary left ventricular assist devices (LVADs) renders patients susceptible to harmful atrial or ventricular suction events. Active control systems may be used to rectify this problem, however, they usually depend on unreliable sensors or potentially inaccurate inferred data from, for example, motor current. This study aimed to characterize the performance of a collapsible inflow cannula reservoir as a passive control system to eliminate suction events in extracorporeal, rotary LVAD support. The reservoir was evaluated in a mock circulation loop against a rigid cannula under conditions of reduced preload and increased LVAD speed in both atrial and ventricular cannulation scenarios. Both cases demonstrated the ease with which chamber suction events can occur with a rigid cannula and confirm that the addition of the reservoir maintained positive chamber volumes with reduced preload and high LVAD speeds. Reservoir performance was dependant on height with respect to the cannulated chamber, with lower placement required in atrial cannulation due to reduced filling pressures. This study concluded that a collapsible inflow cannula is capable of minimizing suction events in extracorporeal, rotary LVAD support.

Keywords

Venous reservoir, ventricular assist device, extracorporeal circulation, suction event, heart failure.

Introduction

Ventricular assist devices (VADs) have undergone decades of improvement since the first implant by DeBakey et al. in 1963 and are now employed to provide mechanical circulatory support throughout the world [1, 2]. VADs may be employed to support the left (LVAD), right (RVAD) or both (BiVAD) sides of the heart for short or long term support, and more recently for myocardial recovery [3]. The use of long term, intracorporeal devices as a bridge to transplant or recovery has been investigated previously with moderate success [4, 5]. However, patients presenting in a moribund state can be difficult to manage and require early decision making and intervention [6]. Use of short term, extracorporeal devices provides a suitable platform for patient stabilization and assessment before opting for a long term device or medical management with a view to ventricular recovery [6, 7].

The pulsatile flow Abiomed BVS5000 VAD is an example of a clinically approved, short term, extracorporeal VAD [8]. The passively filling BVS5000 is attached to an IV-type pole by the patient's bedside, with filling pressures dependant on the device height with respect to the patient. However, significant advantages of continuous flow devices over pulsatile flow devices, including reduced

thrombus formation and improved device lifetime, have been presented previously in detail [9]. The Levitronix CentriMag was developed to address these issues, operating as a continuous flow, extracorporeal VAD with magnetic drive and suspension system [6]. Both BVS5000 and CentriMag devices may be used in an extracorporeal membrane oxygenation (ECMO) circuit when combined with an oxygenator [10, 11].

Unlike pulsatile flow VADs, continuous flow devices are not passively filled and low inlet pressures due to imbalances in the systemic and pulmonary circulations may result in negative atrial or ventricular pressure causing chamber suction [12]. The inflow cannulation site has been shown to influence LVAD performance and patient haemodynamics, however, the choice of site is often dependant on the surgeon's preference and constraints in patient anatomy [13]. Suction of the cannulated chamber may draw the septum or free wall onto the cannula, resulting in intermittent LVAD flow stoppages, endocardial damage and ventricular arrhythmias [14-17]. If left untreated, this septal shift may impair right ventricular function and potentially result in right ventricular failure requiring RVAD support [18].

Previous studies have shown active control systems may be used to achieve flow balancing and reduce suction events with rotary blood pumps [19, 20]. These systems use either sensor or sensor-less feedback control to alter VAD rotational speed depending on a controller input variable. Use of sensors, particularly pressure sensors, for VAD control presents several disadvantages. Pressure sensors pose an ideal location for thrombus to form, while to the author's knowledge no pressure sensor has been developed with zero drift [21]. Meanwhile, the costs associated with reliable sensor development and implementation only adds to the already high costs of VAD support. Sensor-less control systems have also been employed previously in VADs and usually rely on the relationship between VAD flow and a non-invasive measurement which can be easily taken from the VAD itself, such as VAD motor current [22]. However, these measurements may present gross inaccuracies, particularly if there is a blockage in the VAD circuit or thrombus attachment to the impeller [23].

Flexible venous reservoirs which collapse with reduced inlet pressure have been previously employed in ECMO systems to reduce chamber suction events. These systems may be active control systems which include pressure sensors to provide feedback to the pump [24], or passive control systems which rely on the increased resistance of the collapsed inflow cannula to reduce pump flow (R-38 venous reservoir, Medtronic, Minneapolis, USA). Compliant inflow cannulae have been shown to improve the performance of pulsatile devices [25], however no literature exists which

demonstrates the use of a collapsible inflow cannula reservoir in an extracorporeal, rotary LVAD circuit. Therefore, this study aimed to characterize the performance of a collapsible inflow cannula reservoir to eliminate suction events in extracorporeal, rotary LVAD support with both atrial and ventricular inflow cannulation. These results can be used as a guide to assist the clinician with inflow cannula reservoir operation when used in an extracorporeal LVAD circuit.

Methods

Reservoir Construction

An inner mould for reservoir manufacture was designed in SolidWorks (Concord, Massachusetts, USA) and printed with ABS plastic using a rapid prototyping machine (Dimension SST768, Stratasys Inc, Eden Prairie, USA). The mould was then coated with three 20mL layers of de-aired silicone (Silastomer P15, Dalchem, Cheltenham, Australia), with each layer allowed to set prior to adding the subsequent layer. The rapid prototyped mould was then destroyed and removed from inside the silicone reservoir, leaving only the thin-walled flexible reservoir. The reservoir is shown attached to a mock circulation loop (MCL) in Figure 1. The reservoir had a 2mm average wall thickness, 45mm outer diameter at the centre, 19mm outer diameter at the connecting ends, 100mm total length, 50mL priming volume and compliance of 0.5mL/mmHg.

Mock Circulation Loop

A physical five element Windkessel MCL including systemic and pulmonary circulations was used for this study [26, 27]. In brief, ventricular systole was controlled through a series of electropneumatic regulators (ITV2030-012BS5, SMC Pneumatics, Brisbane, AUS) and 3/2 way solenoid valves (VT325-035DLS, SMC Pneumatics, Brisbane, AUS) to provide passively filled heart chambers and variable contractility, heart rate and systolic time. Mechanical check valves were used to simulate the mitral, aortic, tricuspid and pulmonary valves to ensure unidirectional flow throughout the circuit. Four independent Windkessel chambers were employed to simulate lumped systemic and pulmonary arterial and venous compliance. Proportional control valves (EPV-375B, HASS Manufacturing, NY, U.S.A.) allowed easy manipulation of systemic and pulmonary vascular resistance. Mechanical circulatory assistance was incorporated with cannulation through the left atrium or ventricle for LVAD inflow and the ascending aorta for LVAD outflow. The working fluid throughout this study was a water/glycerol mixture (60/40% by mass) with similar viscosity and density to that of blood.

Reservoir Evaluation

The MCL was initially configured to represent a medically treated left heart failure situation without LVAD support. Haemodynamics for this condition are defined in Table 1. A Levitronix CentriMag LVAD (Levitronix LLC, Waltham, USA) was connected to the MCL with inflow cannulation evaluated in both the left atrium and left ventricle and outflow location in the ascending aorta. Cannula length (3m) and inside diameter (12mm) were representative of an extracorporeal LVAD circuit. The reservoir was placed 0.6m from the cannulated chamber to allow an appropriate distance for the cannula to exit the patient. The reservoir was placed at a height which allowed sufficient perfusion during normal operation while preventing chamber suction events when preload was reduced. LVAD support was initiated by increasing LVAD speed and unclamping the cannulae. MCL resistances were then altered to restore the haemodynamics to physiologically healthy levels.

Reservoir performance in atrial cannulation (AC) and ventricular cannulation (VC) was evaluated firstly through a reduction in left atrial/ventricular preload and secondly through an increase in LVAD speed. Reduced preload was simulated through a sudden increase in pulmonary vascular resistance (PVR) from 130 to 400dyne.s.cm^{-5}. The MCL was allowed to settle before data acquisition was stopped. PVR was then returned to normal levels before the LVAD speed was increased in increments of 100rpm until a maximum speed of 3800rpm was reached. The tests were then repeated without the reservoir, using a rigid cannula, for comparison. Reservoir performance at different heights with respect to the cannulated chamber was then evaluated with PVR and LVAD speeds returned to normal levels. For this test, reservoir height was initially set at the height of the cannulated chamber and varied to determine the ideal reservoir height to reduce chamber suction events in AC and VC.

Data acquisition

Haemodynamic and LVAD parameters were captured at 100Hz using a dSPACE acquisition system (DS1104, dSPACE, MI, USA). Systemic and pulmonary flow rates were recorded using magnetic flow meters (IFC010, KROHNE, Sweden) while LVAD inlet and outlet flow rates were recorded with clamp on ultrasonic flow meters (TS410-10PXL, Transonic Systems, NY, USA). Circulatory and LVAD pressures were recorded using silicon-based transducers (PX181B-015C5V, Omega Engineering, Connecticut, USA). Ventricular volume was recorded using a magnetostrictive level sensor (IK1A, GEFRAN, Italy) which, when combined with the left ventricular pressure trace, produced pressure-volume loops. Post processing of the pressure-volume loops enabled capture of left ventricular ejection fraction and stroke work.

Results

Results were compared in AC and VC for increased PVR to characterize the performance of the reservoir. An example of how the reservoir prevented a chamber suction event is demonstrated in Figure 2 which shows the left ventricular volume (LVV) before and after an increase in PVR. Further recorded parameters for the increased PVR test are presented in Table 1 for both AC and VC. In VC, the increased PVR reduced LVAD preload and collapsed the reservoir, thus increasing the LVAD inlet resistance ($LVADR_{in}$) (500 to 750dyne.s.cm^{-5}). This reduced the mean systemic flow rate (MSQ) from 4.8 to 4.4L/min which assisted in maintaining a sufficient end systolic left ventricular volume (LVV_{sys}) of 36mL. Without the reservoir, reduced LVAD preload had minimal effect on the LVAD flow rate, thus causing a ventricular suction event shortly after the PVR increase. Similar results were noted in AC, with the reservoir increasing LVAD circuit resistance to 1400dyne.s.cm^{-5} which maintained left atrial pressure (LAP) at positive levels following a reduction in preload. Without the reservoir, atrial pressure dropped below zero, indicating atrial suction. Before the PVR increase, left ventricular ejection fraction (EF) was higher with the reservoir (35%) compared to without the reservoir (28%) in VC. This was due to the left ventricular pressure pulse ejecting fluid into a compliant vessel capable of expanding with increased inlet pressure. Combined with fairly consistent left ventricular systolic and diastolic pressures, the increased EF resulted in higher ventricular stroke work (SW) in VC with the reservoir (0.35W) compared to without the reservoir (0.25W). EF and SW before the PVR increase in AC were similarly small with and without the reservoir as the ventricular volume barely changed over each cardiac cycle. However, after the PVR increase with the reservoir included in the circuit, the aortic pressure dropped which allowed some ejection through the aortic valve and an increased stroke volume and EF. An increase in EF was also noted in AC without the reservoir after the PVR increase, however this was due to much lower LVV as the atrium collapsed.

Results were then compared with and without the reservoir in AC and VC to observe the point of chamber suction when LVAD speed was increased. LVV for VC and LAP for AC are plotted against LVAD speed in Figure 3, while haemodynamic parameters for each case are shown in Table 2. With VC, the reservoir maintained LVV_{sys} above 75mL for all VAD speeds up to 3800rpm, however when the reservoir was removed ventricular suction occurred at 3100rpm. With the reservoir, MAP and MSQ increased slightly with increased LVAD speed, however the drastically increased $LVADR_{in}$ due to reservoir collapse restrained this increase. Once again, similar results were recorded with AC as the reservoir maintained LAP at 6mmHg or above for all LVAD speeds up to 3800rpm. Reservoir collapse prevented atrial suction by passively increasing $LVADR_{in}$ from 700 to 4250dyne.s.cm^{-5} over a LVAD

speed increase from 2800 to 3800rpm. Without the reservoir, negative atrial pressures were recorded when the LVAD speed approached 3400rpm, indicating atrial suction.

Increased reservoir height resulted in reduced reservoir filling pressure for both cannulation sites, thus increasing the $LVADR_{in}$. This was particularly noticeable in AC due to the lack of a systolic pulse to aid with reservoir filling, where the $LVADR_{in}$ increased from 650 to 3245dyne.s.cm^{-5} when the reservoir height was increased from 0 to 150mm above the atrium (Figure 4). Meanwhile, little change was noted in $LVADR_{in}$ with VC until the reservoir height was increased to 200mm above the ventricle. Therefore, lower reservoir placement was required in AC compared to VC. In this study, the ideal reservoir height to eliminate suction events while preventing unnecessary reservoir collapse was found to be 150mm above the ventricle for VC and at the height of the atrium for AC. Reservoir placement high above the cannulated chamber resulted in reservoir collapse well before chamber suction, thus unnecessarily reducing VAD flows while chamber pressures were still at sufficient levels. However, reservoir placement well below the cannulated chamber increased the hydrostatic pressure within the reservoir, thus suction of the chamber occurred prior to reservoir collapse.

Discussion

The limited preload sensitivity of rotary VADs increases the likelihood of suction events, particularly in low volume situations. Reesink et al. (2007) [16] demonstrated the ease with which ventricular suction can occur with increased LVAD speed or pulmonary resistance, which was also noted in our study. Reesink also reported that the usual method of avoiding suction by a trial and error based approach of manipulating LVAD speed is not always successful, demonstrating the requirement for a control system to prevent suction events. Despite improvements in active control systems for LVADs, limitations still exist. The control system must operate in a similar manner to the native heart by reducing cardiac output when preload is reduced. The strategy appears simple; however, as discussed in the introduction, accurate, invasive preload measurement requires sensors which are still costly and unreliable, while non-invasive preload measurements rely on inferred data which can be grossly inaccurate in some circumstances. This study examined whether a passively collapsible inflow cannula reservoir could overcome the requirement for sensors, while also providing an inexpensive and reliable solution for suction prevention.

The inflow cannula reservoir eliminated chamber suction events by passively increasing the LVAD inlet resistance due to reservoir collapse as the preload decreased. Manipulation of LVAD outflow resistance to alter performance has been presented previously which demonstrated that by increasing LVAD outlet resistance to reduce outlet pressure, an LVAD may be used to support the pulmonary circulation [28]. As expected, our study demonstrated that manipulation of inlet resistance reduced LVAD outlet pressure along with LVAD outflow. The long cannulae required for extracorporeal LVAD support produced a large inlet resistance with a slight increase of approximately 50dyne.s.cm^{-5} in all cases when the full reservoir was added to the circuit. The increased resistance due to reservoir addition can be attributed to the various fittings required to secure the reservoir to the cannula. Meanwhile, AC consistently resulted in higher inlet resistance compared to VC due to additional fittings and tubing length on the AC site in the MCL. Increased PVR resulted in partial reservoir collapse which increased the $LVADR_{in}$ in AC more than that seen in VC with values of 1400 and 750dynes.s.cm^{-5} respectively. Variations in LVAD speed once again revealed higher $LVADR_{in}$ for AC compared to VC, particularly at high VAD speeds. This can be attributed to the left ventricular systolic pressure filling the reservoir in VC which is not experienced in AC.

With the reservoir in VC, higher EF and SW were recorded compared to AC both before and after the increase in PVR. When LVAD speed was incrementally increased with the reservoir from 2800 to 3800rpm, MAP increased with VC (95 to 101mmHg) however decreased with AC (96 to 90mmHg). Similar trends were noted with MSQ and could be attributed to the dramatically increased $LVADR_{in}$ with AC. This may appear to restrict the LVAD output with the reservoir, however if increased cardiac output is required, the reservoir could simply be placed at a reduced height to decrease the $LVADR_{in}$. Reductions in MAP and MSQ appeared more apparent during reservoir collapse with AC, indicating the reservoir may be slightly more suitable for VC. Despite the reported advantages of one cannulation site over another, preference of the surgeon and surgical or patient constraints may apply, indicating LVADs and their related products should be suitable or adaptable for both cannulation sites. This study suggests that a collapsible inflow cannula reservoir can be used to prevent suction events in either inflow cannulation site.

An ideal reservoir height with respect to the cannulated chamber in both AC and VC was determined in this study. However, this height was based on a trial and error method for one particular degree of left heart failure, and therefore must be addressed clinically on a patient by patient basis. Patient volume status, ventricular contractility, and cannulation site, amongst other things, will influence the ideal reservoir height. As a guide for clinical implementation of the reservoir, the ideal reservoir

height for both cannulation sites was the highest point at which the reservoir had not started to collapse. Therefore, an adjustable height mechanism is required, similar to the passively filled Abiomed BVS5000 pulsatile VAD. The BVS5000 was particularly susceptible to thrombus formation, as the reservoir may also be if not designed to optimize the blood flow path [8]. The additional volume in the reservoir served to account for only minimal flow imbalances and could be replaced by an in-line collapsible inflow cannula which may also be applicable for use with intracorporeal LVADs. Meanwhile, this design change may reduce the device's potential for thrombus formation, which is of particular importance due to the risk of mortality associated with post-LVAD stroke [29]. Future studies on the flow dynamics within the modified collapsible cannula are required before progression of the device to the clinical setting.

In-vitro evaluation in a MCL proved an appropriate method for an initial characterization of the collapsible inflow cannula reservoir. This setting enabled controlled, repeatable testing and data collection; however, it also presented several disadvantages over an in-vivo study. Firstly, suction typically occurs as the free wall, septum or valve is pulled onto the cannula, obstructing the fluid path. In our study, suction events were based on fluid levels in each chamber reaching a preset zero level with no physical suction occurring after this point, thus the physiological response during or after suction could not be observed. Meanwhile, the lack of native autoregulation mechanisms such as the Frank-Starling and baroreceptor responses in the MCL may influence the presented results, even though these responses are typically reduced in heart failure [30, 31].

Conclusion

Continuous flow LVADs present several advantages over pulsatile flow devices, however limited preload sensitivity renders patients susceptible to harmful atrial or ventricular suction events. Evaluation of a rigid cannula demonstrated the ease with which chamber suction events can occur after a preload reduction or LVAD speed increase; however these were not experienced when a collapsible inflow cannula reservoir was added to the circuit. VC appeared slightly more suitable for reservoir use as haemodynamics were maintained at higher levels before and after reservoir collapse. The key mechanism in the reservoir's operation was a passively increased LVAD inlet resistance due to reservoir collapse when LVAD, and hence reservoir, preload was reduced. However, this added volume could induce thrombus formation. Therefore, the reservoir should be redesigned as an in-line device without the additional reservoir volume. In conclusion, this study demonstrated that the addition of a collapsible inflow cannula to an extracorporeal LVAD circuit

acted as a passive control system to reduce chamber suction events in both AC and VC, thus reducing potential endocardial damage, pump flow stoppages and ventricular arrhythmias.

Acknowledgements

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Figure 1

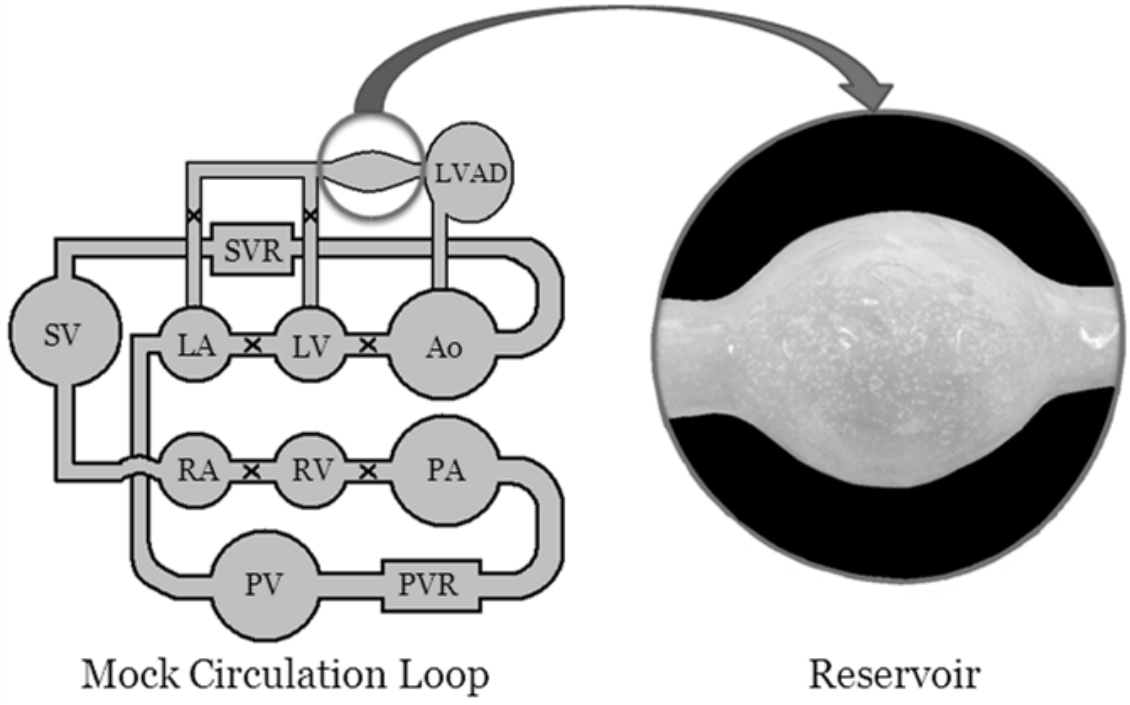

Figure 1 – Diagrammatic representation of the collapsible inflow cannula reservoir attached to either the left atrium or left ventricle in the mock circulation loop with the real reservoir shown in the inset. LA – left atrium, LV – left ventricle, Ao – aortic compliance chamber, LVAD – left ventricular assist device, SVR – systemic vascular resistance valve, SV – systemic venous compliance chamber, RA – right atrium, RV – right ventricle, PA – pulmonary arterial compliance chamber, PVR – pulmonary vascular resistance valve, PV – pulmonary venous compliance chamber, x – valve.

Figure 2

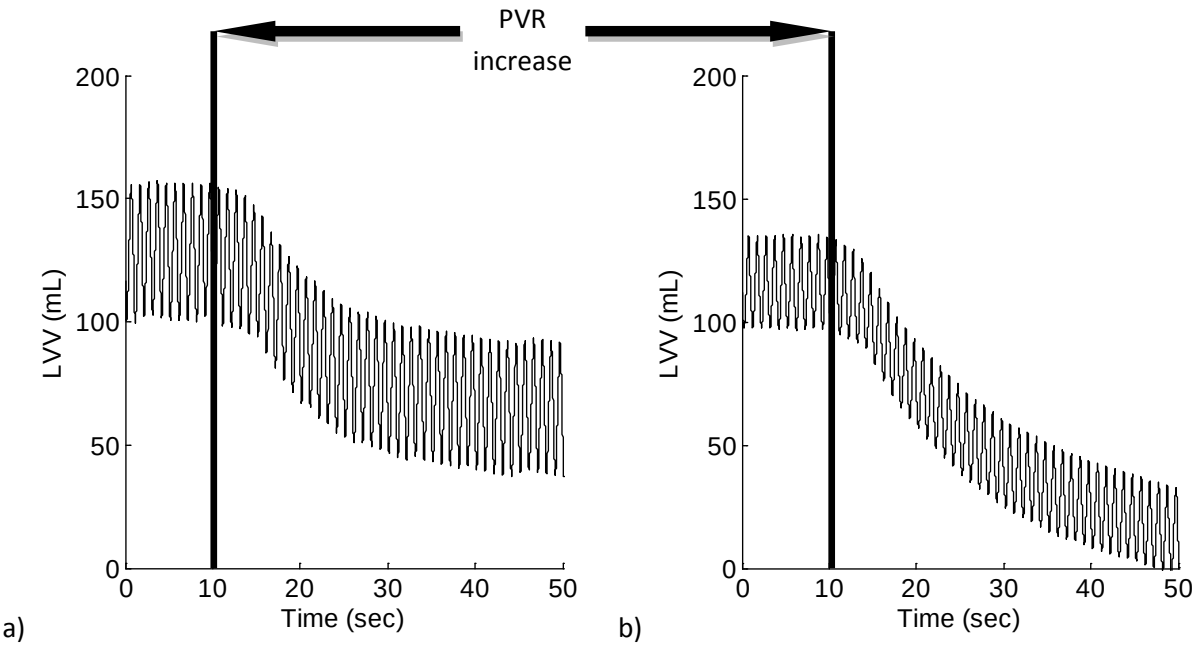

Figure 2 – Left ventricular volume (LVV) vs. time (a) with and (b) without the reservoir in ventricular cannulation before and after an increase in pulmonary vascular resistance. Note ventricular suction has occurred during end systolic periods after 50 seconds without the reservoir in the circuit.

Figure 3

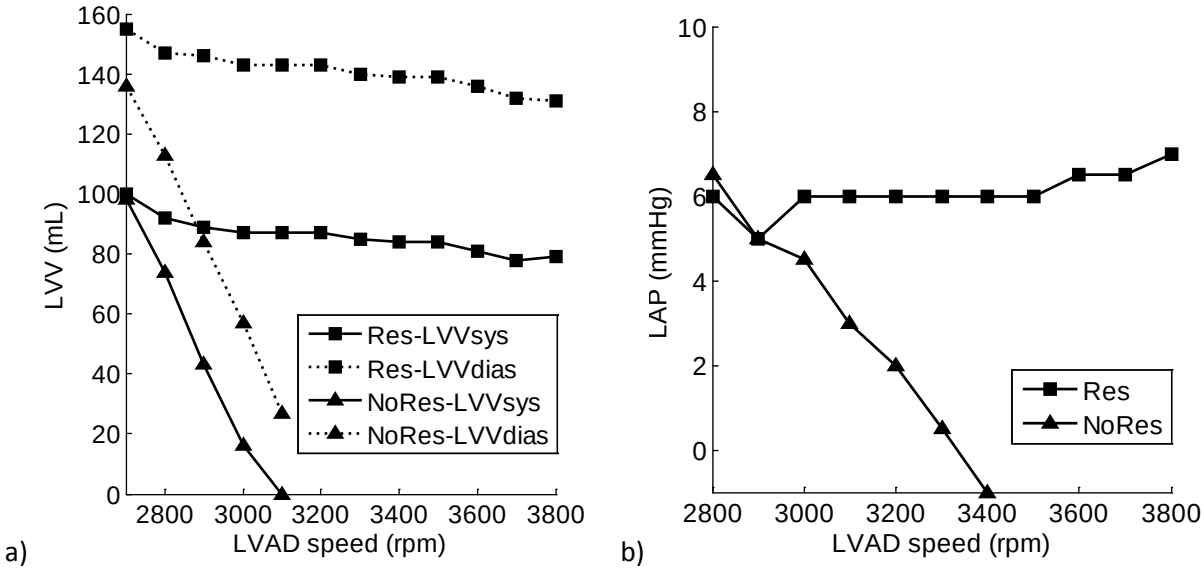

Figure 3 – Trends with and without the reservoir for (a) left ventricular volume vs. LVAD speed in ventricular cannulation and (b) left atrial pressure vs. LVAD speed in atrial cannulation. LVV – left ventricular volume, LAP – left atrial pressure, rpm – revolutions per minute, LVVsys – systolic left ventricular volume, LVVdias – diastolic left ventricular volume, Res – with the reservoir, NoRes – without the reservoir.

Figure 4

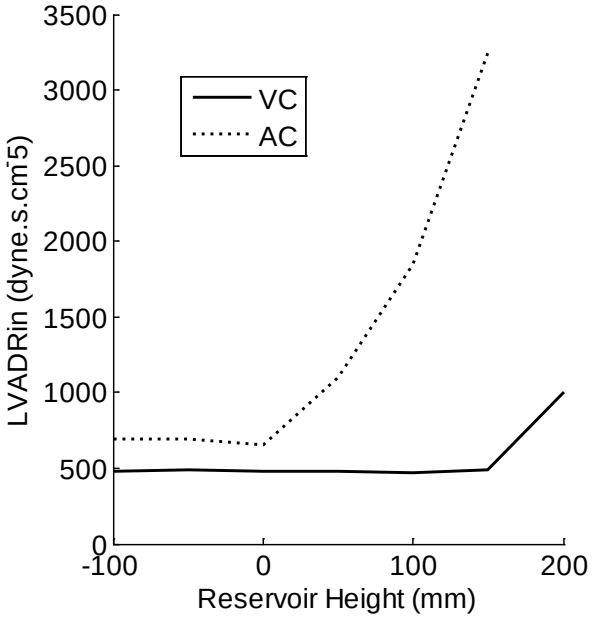

Figure 4 – Plot of LVAD inflow cannula resistance (LVADRin) vs. reservoir height with respect to the cannulated chamber for ventricular (VC) and atrial (AC) cannulation.

List of Tables

Table 1

Parameter	LHF	Ventricular Cannulation				Atrial Cannulation			
	No VAD	Reservoir		No Reservoir		Reservoir		No Reservoir	
	NPVR	NPVR	EPVR	NPVR	EPVR	NPVR	EPVR	NPVR	EPVR
LAP (mmHg)	12	9	6	8	3	6	2.5	6.5	-2
MAP (mmHg)	60	95	86	95	95	96	83	96	93
MSQ (L/min)	2.5	4.8	4.4	4.9	4.8	4.8	4.2	4.9	4.7
LVV_{sys} (mL)	197	100	36	98	0	156	37	148	22
LVV_{dias} (mL)	247	155	91	136	20	159	47	154	25
EF (%)	20	35	60	28	100	2	21	4	12
SW (Watts)	0.34	0.35	0.35	0.25	0.24	0.00	0.06	0.02	0.00
$LVADR_{in}$ ($dyne.s.cm^{-5}$)	N/A	500	750	400	400	700	1400	650	650

Table 1 – Recorded parameters in ventricular and atrial cannulation with and without the reservoir before and after an increase in pulmonary vascular resistance. LHF – left heart failure, NPVR – normal pulmonary vascular resistance, EPVR – elevated pulmonary vascular resistance, LAP – left atrial pressure, MAP – mean aortic pressure, MSQ – mean systemic flow rate, LVV_{sys} – systolic left ventricular volume, LVV_{dias} – diastolic left ventricular volume, EF – ejection fraction, SW – stroke work, $LVADR_{in}$ – LVAD inflow cannula resistance.

Table 2

Ventricular Cannulation										
LVAD	**Reservoir**					**No Reservoir**				
Speed	**LAP**	**MAP**	**MSQ**	**LVV_{sys}**	**$LVADR_{in}$**	**LAP**	**MAP**	**MSQ**	**LVV_{sys}**	**$LVADR_{in}$**
rpm	mmHg	mmHg	L/min	mL	dyne.s.cm^{-5}	mmHg	mmHg	L/min	mL	dyne.s.cm^{-5}
2700	9	95	4.8	100	500	8	95	4.9	98	400
2800	8	97	4.9	92	650	7	102	5.1	74	400
3000	8	97	4.9	87	1100	5	116	5.5	16	450
3200	8	97	4.9	87	1600	-	-	-	-	-
3400	8	98	4.9	84	2100	-	-	-	-	-
3600	8	99	4.9	81	2650	-	-	-	-	-
3800	8	101	4.9	77	3250	-	-	-	-	-
Atrial Cannulation										
LVAD	**Reservoir**					**No Reservoir**				
Speed	**LAP**	**MAP**	**MSQ**	**LVV_{sys}**	**$LVADR_{in}$**	**LAP**	**MAP**	**MSQ**	**LVV_{sys}**	**$LVADR_{in}$**
rpm	mmHg	mmHg	L/min	mL	dyne.s.cm^{-5}	mmHg	mmHg	L/min	mL	dyne.s.cm^{-5}
2800	6	96	4.8	156	700	6.5	96	4.9	148	650
3000	6	93	4.8	151	1300	4.5	109	5.3	143	700
3200	6	93	4.8	149	1800	2	123	5.7	125	700
3400	6	95	4.85	142	2300	-	-	-	-	-
3600	6.5	93	4.8	142	3100	-	-	-	-	-
3800	7	90	4.7	152	4250	-	-	-	-	-

Table 2 – Recorded parameters in ventricular and atrial cannulation with and without the reservoir under various LVAD rotational speeds. LAP – left atrial pressure, MAP – mean aortic pressure, MSQ – mean systemic flow rate, LVV_{sys} – systolic left ventricular volume, $LVADR_{in}$ – LVAD inflow canula resistance, rpm – revolutions per minute. Dashed lines indicate chamber suction had occurred at a lower LVAD speed.

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