Cavaortic shunt improves hemodynamics with preserved oxygen delivery in experimental right ventricular failure during left ventricular assist device therapy

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Objective: Right heart failure is a major cause of morbidity and mortality after left ventricular assist device (LVAD) implantation. This study evaluated the approach of a cavaortic shunt included in the LVAD circuit, which would aim to relieve venous congestion and improve hemodynamics with preserved oxygen delivery during induced right ventricular failure.

Methods: Right ventricular failure was induced by coronary ligation in 10 pigs. An LVAD was implanted and a cavaortic shunt was created from the right atrium and included in the assist circuit. Hemodynamic measures and blood gas analyses were analyzed. Oxygen delivery and oxygen consumption were estimated.

Results: Right atrial pressure decreased from more than 20 mm Hg to 17.2 mm Hg (14.8-18.4) with the LVAD and to 14.1 mm Hg (11.2-15.5) ($P < .01$) with the LVAD and cavaortic shunt. Mean arterial pressure increased from 70.9 mm Hg (67.6-79.8) to 81.5 mm Hg (70.8-92.6) ($P = .02$) with addition of the shunt into the assist circuit. Cardiac output increased from 3.5 L/min (2.6-4.2) to 4.9 L/min (3.5-5.6) ($P < .01$) with cavaortic shunting. Oxygen delivery with the cavaortic shunt was 337 mL/min (± 70) as compared with left ventricular assist alone at 258 mL/min (± 52) ($P < .01$). Oxygen consumption was restored during use of the cavaortic shunt.

Conclusions: A cavaortic shunt combined with an LVAD during right ventricular failure reduces central venous pressures, increases systemic arterial pressure, and enables increased cardiac output compared with device therapy alone. This was feasible with preserved oxygen delivery. (J Thorac Cardiovasc Surg 2014;147:625-31)

Left ventricular assist device (LVAD) therapy has become an established treatment for end-stage heart failure.^{1,2} In these patients, right ventricular (RV) failure of various degrees is one of the most dreaded complications known to cause significant morbidity and mortality.^{3,4} Today, although efforts have been made, there is a lack of reliable predictors of which patients will have RV failure undergoing surgery.^{5,6} Irrespective of the preoperative condition of the RV, the LVAD implantation is at times a venture inasmuch as the evolution of RV function during surgery is somewhat unpredictable.

Once severe RV failure occurs, the only reliable treatment is the placement of an RV assist device (RVAD). Patients who receive RVADs have poorer prognoses both in short- and long-term perspectives. However, they more commonly have severe RV failure, venous congestion, and end-organ failure preoperatively.^{3,7} We have earlier evaluated a complementary approach to RVAD, the concept of a modified Glenn shunt (superior vena cava to main

pulmonary artery) in experimental RV failure.⁸ This approach was feasible with a positive effect on hemodynamics such as central venous pressures and cardiac output (CO).

However, in cases of elevated pulmonary vascular resistance (PVR), the concept of Glenn is not an option when RV failure occurs during LVAD surgery.

Elevated PVR is present in some LVAD candidates as a consequence of severe and longstanding congestive heart failure.⁹⁻¹¹ In these patients, the resistance in the pulmonary vascular bed might be even further elevated with prolonged surgery, coagulopathy, and bleeding necessitating transfusion. This viscous circulatory state with increasing PVR during LVAD implantation might result in a situation that requires temporary decompression of both the RV and the pulmonary circulation.

We sought to evaluate a complementary approach to bi-ventricular failure that would aim at properly unloading the RV, relieving central venous congestion, and unloading the pulmonary circulation. We hypothesized that a cavaortic shunt included in the LVAD circuit would reduce central venous pressures and improve circulation (CO and mean arterial pressure [MAP]) with maintained oxygen delivery (Do_2) in acute RV failure during LVAD therapy.

MATERIAL AND METHODS

The Uppsala Ethical Committee on Laboratory Animal Research approved the study and all animals received humane care in compliance with the European Convention on Animal Care.

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Abbreviations and Acronyms

Cao ₂	= arterial oxygen content
CO	= cardiac output
Do ₂	= oxygen delivery
LA	= left atrium
LV	= left ventricular
LVAD	= left ventricular assist device
MAP	= mean arterial pressure
Pao ₂	= arterial oxygen partial pressure
PVR	= pulmonary vascular resistance
RA	= right atrium
RV	= right ventricular (ventricle)
RVAD	= right ventricular assist device
Sao ₂	= arterial oxygen saturation
Svo ₂	= mixed venous oxygen saturation
Vo ₂	= oxygen uptake

Anesthesia

The study comprised 11 pigs of Swedish country breed with a mean weight of 34.9 kg (± 1.6).

Anesthesia was induced by a subcutaneous injection of xylazine (Rompun, 2.2 mg/kg; Bayer A/S, Lyngby, Denmark) and tiletamine/zolazepam (Zoletil, 100 6.0 mg/kg; Virbac SA, CarrosCedex, France) and maintained by infusion of a buffered glucose solution carrier (Rehydrex with glucose, 25 mg/mL; Fresenius Kabi AB, Uppsala, Sweden) with ketamine, 30 mg/kg/h (Ketaminol Vet 100 mg/mL; Intervet AB, Stockholm, Sweden), fentanyl, 0.04 mg/kg/h (Fentanyl 50 μ g/mL; B Braun Medical AB, Danderyd, Sweden), midazolam, 0.1 mg/kg/h (Midazolam Hameln, 1 mg/mL; AlgotPharma AB, Kista, Sweden), and pancuronium bromide, 0.3 mg/kg/h (Pavulon 2 mg/mL; ScheringPlough AB, Stockholm, Sweden).

Body temperature was controlled with a heating pad, aiming for a core temperature of 37°C.

The pigs were intubated and mechanically ventilated with 21% oxygen by a Siemens Servo-i ventilator (Maquet GmbH & Co KG, Rastatt, Germany). Volume-controlled ventilation was used aiming for an arterial Pco₂ within the range of 5.0 to 5.5 kPa, and a positive end-expiratory pressure of 5 cm H₂O was applied. The bladder was catheterized with a silicone catheter.

A catheter was inserted through the right external jugular vein (BD Careflow 17G; Becton-Dickinson AB, Stockholm, Sweden) for central venous pressure, blood sampling, and drug administration. A thermodilution pulmonary artery catheter (BD Criticath Pulmonary Artery/Thermodilution Catheter; Becton-Dickinson) was introduced through the right external jugular vein and advanced to the pulmonary artery for measurements of CO and mixed venous oxygen saturation (Svo₂).

A catheter was inserted into the left femoral artery for continuous monitoring of oxygen saturation (Pao₂), blood gas extraction, and for arterial blood pressure with a pressure transducer (BD Careflow 20G; Becton-Dickinson).

Surgical Preparation

A median sternotomy was performed. The pericardium was opened and pericardial stay sutures were placed. The azygos and hemiazygos veins were ligated. Pressure transducer catheters were surgically placed in the right atrium (RA), RV, and the left atrium (LA). The catheters were secured with fine sutures and connected to a monitoring system (Dräger Infinity Delta; Dräger Medical; Lübeck, Germany). Animals were heparinized with 7500 IU once and repeatedly every 2 hours with 5000 IU. The RA

was cannulated with a 24F venous cannula (Malleable Single Stage Venous Cannula; Edwards Lifesciences, Irvine, Calif), as shown in Figure 1. The left ventricular (LV) apex was cannulated with a 28F venous cannula (Malleable Single Stage Venous Cannula; Edwards Lifesciences). A 16F arterial cannula (FemFlex; Edwards Lifesciences) was inserted into the ascending aorta. All cannulas were secured with purse-string sutures. A Bio-Pump flow probe connector (Medtronic, Inc, Minneapolis, Minn) was connected to the RA cannula. The RA and the LV apical cannula were deaired and connected with a Y-connector to the inflow tubing of a preprimed Bio-Pump connector (Medtronic). The aortic cannula was deaired and connected to the outflow tubing of the Bio-Pump connector.

Experimental Protocol

RV failure was created by subsequent ligation of 4 to 6 branches of the right coronary artery and all visible branches to the RV from the left coronary artery. The RV was further challenged with volume overload achieved by rapid infusion of 2000 mL HESRA (Baxter AB, Knivsta, Sweden). RV failure was considered present at an RA pressure of 20 mm Hg or more.

When the pressure was 20 mm Hg or more, the LVAD was started and set to a flow corresponding to the baseline CO value. The RA cannula was opened and the flow through this cannula was set to one third of total LVAD flow by an adjustable clamp, so that one third of the CO was shunted from the RA, via the Bio-Pump connector, into the ascending aorta. After 20 minutes of shunting, measurements were made.

Measurements

The experimental protocol required right heart failure to be established. Data were recorded during 4 time periods: (1) baseline, (2) severe RV failure, (3) LVAD with closed RA cannula, and (4) LVAD and one third of the total CO through the RA cannula (Figure 1).

A 20-minute stabilization period was followed by repeated measurements every minute for 5 consecutive minutes.

Standard lead II electrocardiogram, systemic arterial blood pressure, RA pressure, RV pressure, LA pressure, and pulmonary artery pressure were monitored continuously and recorded. CO was measured, using the thermodilution technique. During LVAD and LVAD with cavaortic shunting, the total LVAD flow was used as CO. Blood samples were collected at the same time periods for arterial and venous blood gas analysis. Arterial blood gases, hemoglobin concentration, and Svo₂ were measured with an ABL 500 Radiometer (Radiometer Medical ApS, Bronshøj, Denmark).

The arterial oxygen content (Cao₂) was calculated using the formula $(0.0138 \times \text{hemoglobin} \times \text{Sao}_2) + (0.0031 \times \text{Pao}_2)$ and the Do₂ using the formula $\text{Cao}_2 \times \text{CO} \times 10$. Vo₂ was calculated using the formula $\text{Vo}_2 = \text{CO} \times (\text{Cao}_2 - \text{Cvo}_2)$.

Statistical Analysis

Nonparametric tests were used inasmuch as normality assumption was subject to question. For hemodynamic measurements such as RA pressure, RV pressure, LA pressure, MAP, and CO, the value for each animal was the mean of all measurements of each period and data were expressed as medians with 95% confidence interval. For blood gas analysis, and calculated parameters (Cao₂ and Do₂), data are expressed as means ($\pm$ SD). The Wilcoxon matched pairs signed-rank test was used to compare differences between the periods: baseline, RV failure, RV failure and LVAD without shunt, and RV failure and LVAD with shunt. Statistical calculations were performed with the SPSS statistical package 20.0 (SPSS, Inc, Chicago, Ill).

RESULTS

All 10 animals survived the study period and constituted the study group. RV failure was achieved in all pigs leading to significantly elevated right-sided filling pressures.

FIGURE 1. Animal preparation and flow chart of the experimental protocol in the model of right ventricular failure in pigs ($n = 10$). An LVAD was implanted and a cavaortic shunt was created from the right atrium (the cannulas into the superior vena cava) to the aorta. LVAD, Left ventricular assist device.

RV Failure

After induced RV failure, CO decreased from 3.4 L/min (2.6-4.2) to 2.0 L/min (1.5-2.5) ($P = .01$; Table 1). Mean RA pressure increased from 10.8 mm Hg (9.0-11.0) at baseline to 20.4 mm Hg (20.0-22.2) ($P < .01$) at RV failure as depicted in Figure 2 and Table 1. The diastolic RV pressure increased from 9.0 mm Hg (8.7-10.3) at baseline to 13.4 mm Hg (12.4-16.6) ($P < .01$; Figure 2).

No significant changes were observed in the LA pressure or systemic MAP. In addition, the SaO_2 and Svo_2 remained unchanged during establishment of RV failure (Figure 3). The Do_2 was reduced by 43% from 387 mL/min (± 96) at baseline to 171 mL/min (± 40) ($P < .01$) at RV failure (Table 1 and Figure 4). Finally, oxygen uptake (Vo_2) was reduced from 180 mL/min (± 53) to 86 mL/min (± 17) ($P < .01$) during RV failure (Figure 4).

LVAD Therapy Only

When LVAD therapy was instituted, the pump was set to produce a CO that was equal to the baseline value at 3.4 L/min (2.6-4.2). This resulted in a decrease in RA pressure from 20.4 mm Hg (20.0-22.2) to 17.2 mm Hg (14.8-18.4) ($P < .01$; Figure 2). Also, the diastolic RV pressure decreased from 13.4 mm Hg (12.4-16.6) at RV failure to

11.9 mm Hg (11.0-15.0) ($P = .02$) during LVAD therapy, as shown in Table 1 and Figure 2.

Compared with the period with RV failure, MAP was unaffected, but both Do_2 and Vo_2 were increased when LVAD therapy was put in use, as depicted in Table 1.

LVAD and Cavaortic Shunt

There was an increase in CO when LVAD therapy was combined with the cavaortic shunt as compared with LVAD alone, from 3.5 L/min (2.7-4.0) to 4.9 L/min (3.5-5.6) ($P < .01$; Table 1). The RA pressure decreased even further when the cavaortic shunt was put in use to 14.1 mm Hg (11.2-15.5) ($P < .01$) as compared with LVAD alone (Table 1 and Figure 2). The RV pressures did not change significantly, nor did the LA pressure with use of the shunt. There was an elevation of MAP from 70.9 mm Hg (67.6-79.8) with LVAD only to 81.5 mm Hg (70.8-92.6) ($P = .02$) with the use of LVAD and cavaortic shunt, as described in Table 1.

A decrease in SaO_2 from 94.4% (± 6.4) to 86.2% (± 6.5) ($P < .01$) was observed when the cavaortic shunt was in use. The Svo_2 , on the other hand, remained unchanged, as shown in Figure 3 and Table 1. The Do_2 during LVAD and the cavaortic shunt were higher when compared with LVAD only, at 337 mL/min (± 70) and 258 mL/min

TABLE 1. Hemodynamic values, blood gas values, and calculations of CaO_2 and Do_2 at baseline, RV failure, LVAD treatment, and LVAD treatment plus cavaoortic shunting (n = 10)

	Baseline	Versus (P value)	Right heart failure	Versus (P value)	LVAD	Versus (P value)	LVAD plus cavaoortic shunt
RAP (mm Hg)	10.8 (9.0-11.0)	<.01	20.4 (20.0-22.2)	<.01	17.2 (14.8-18.4)	<.01	14.1 (11.2-15.5)
DRVP (mm Hg)	9.0 (8.7-10.3)	<.01	13.4 (12.4-16.6)	.02	11.9 (11.0-15.0)	.41	11.6 (10.0-16.0)
SRVP (mm Hg)	31.9 (27.8-41.8)	.04	37.2 (35.6-39.2)	.17	35.7 (34.3-43.8)	.45	35.9 (32.2-43.8)
mPAP (mm Hg)	22.4 (18.8-24.8)	<.01	28.6 (26.4-31.2)	.06	23.9 (21.0-31.4)	.72	24.0 (19.8-34.8)
LAP (mm Hg)	15.8 (13.0-20.0)	.11	17.2 (15.3-26.2)	<.01	13.6 (11.0-16.2)	.42	12.1 (10.5-17.8)
MAP (mm Hg)	65.4 (60.8-71.8)	.07	71.7 (62.0-79.6)	.51	70.9 (67.6-79.8)	.02	81.5 (70.8-92.6)
CO (L/min)	3.4 (2.6-4.2)	<.01	2.0 (1.5-2.5)	N/A	N/A	N/A	N/A
LVAD (L/min)	N/A	N/A	N/A	N/A	3.5 (2.7-4.0)	<.01	4.9 (3.5-5.6)
Do_2 (mL/min)	387 (± 96)	<.01	171 (± 40)	<.01	258 (± 52)	<.01	337 (± 70)
Vo_2 (mL/min)	180 (± 53)	<.01	86 (± 17)	<.01	138 (± 32)	.07	160 (± 39)
Sao_2 (%)	96.0 (± 2.2)	.51	95.2 (± 3.8)	.65	94.4 (± 6.4)	<.01	86.2 (± 6.5)
Svo_2 (%)	50.2 (± 11.8)	.17	46.4 (± 8.2)	.84	44.2 (± 6.2)	.24	44.1 (± 12.3)
CaO_2 (mL/dL)	11.5 (± 1.5)	<.01	8.6 (± 0.9)	<.01	7.5 (± 0.6)	.16	7.3 (± 1.1)
Cvo_2 (mL/dL)	6.0 (± 1.4)	<.01	4.2 (± 0.9)	.02	3.5 (± 0.3)	.76	3.8 (± 1.6)
Hemoglobin (g/dL)	8.7 (± 1.3)	<.01	6.6 (± 0.8)	<.01	5.8 (± 0.6)	.06	6.1 (± 1.0)
PaO_2 (kPa)	13.6 (± 3.2)	.39	12.9 (± 2.7)	.36	12.3 (± 2.5)	<.01	7.7 (± 0.9)
Paco_2 (kPa)	5.0 (± 0.5)	.42	5.1 (± 0.7)	.76	5.2 (± 0.6)	.09	5.4 (± 0.8)
pH	7.46 (± 0.06)	.05	7.44 (± 0.06)	.30	7.43 (± 0.07)	.55	7.43 (± 0.07)
SBE (mmol/L)	2.9 (± 2.2)	<.01	1.1 (± 1.4)	.33	1.5 (± 2.0)	.04	2.0 (± 2.2)

Hemodynamic values presented as median with 95% confidence interval, and blood gas derived values as mean $\pm$ standard deviation. CaO_2 , Arterial oxygen content; Do_2 , oxygen delivery; RV, right ventricular; LVAD, left ventricular assist device; RAP, right arterial pressure; DRVP, diastolic right ventricular pressure; SRVP, systolic right ventricular pressure; mPAP, mean pulmonary artery pressure; LAP, left atrial pressure; MAP, mean arterial pressure; CO, cardiac output; Vo_2 , oxygen uptake; Sao_2 , arterial oxygen saturation; Svo_2 , mixed venous oxygen saturation; Cvo_2 , venous oxygen content; PaO_2 , arterial oxygen partial pressure; Paco_2 , arterial carbon dioxide partial pressure; SBE, arterial standard base excess; N/A, not applicable.

(± 52), respectively ($P < .01$; Figure 4 and Table 1). Moreover, the Vo_2 was unaffected at 138 mL/min (± 32) with LVAD only as compared with 160 mL/min (± 39) when the shunt was in use ($P = .07$).

Blood Gas Analysis

Initially, the hemoglobin decreased during the experiment, but then there was a slight increase in hemoglobin during cavaoortic shunting, as shown in Table 1. In addition, the CaO_2 decreased throughout the experiment from

FIGURE 2. Right atrial and right ventricular pressures at baseline, RV failure, LVAD, and LVAD + cavaoortic shunt (n = 10). RAP, Right atrial pressure; DRVP, diastolic right ventricular pressure; SRVP, systolic right ventricular pressure; LVAD, left ventricular assist device.

11.5 mL/dL (± 1.5) at baseline to finally 7.3 mL/dL (± 1.1) ($P < .01$) with LVAD and use of the cavaoortic shunt, as shown in Table 1. Arterial standard base excess decreased from baseline to RV failure but increased during LVAD to 1.5 mmol/L (± 2.0) and even further when the cavaoortic shunt was used to 2.0 mmol/L (± 2.2) ($P = .03$; Table 1).

DISCUSSION

This experimental study shows that a cavaoortic shunt combined with LVAD therapy reduces central venous pressure, increases MAP, and enables increased CO when compared with LVAD without the shunt in a model of induced RV failure. This was feasible with preserved systemic Do_2 .

A cavaoortic shunt included in the LVAD circuit increased systemic Do_2 by approximately 30% when compared with LVAD alone in a situation with severe RV failure. The cavaoortic shunt produced a level of Do_2 at 87% of the baseline value as compared with 66% with LVAD alone. Interestingly, this level of Do_2 was accomplished even though nonsaturated blood (shunt flow) was added into the arterial circulation. We believe that the main reason for this increase in Do_2 is the increase in CO, which is enabled by the cavaoortic shunt added into the LVAD circuit. This increase in CO was not possible to obtain without the shunt inasmuch as the failing RV is the limiting factor, unable to increase its workload. Moreover, in a clinical setting, where the 30% reduction in hemoglobin, which occurred

FIGURE 3. Systemic oxygen saturation (arterial and venous) at baseline, right ventricular failure, LVAD, and LVAD + cavaortic shunt ($n = 10$). SaO_2 , Arterial oxygen saturation; SvO_2 , venous oxygen saturation; LVAD, left ventricular assist device.

from baseline to LVAD and shunt could be restored by transfusion, the DO_2 with the shunt would most certainly be even further improved. Furthermore, the SvO_2 is low but sustained throughout the experiment despite the fact that one third of CO is shunted directly into the arterial circulation. In addition, VO_2 decreases during RV failure and is restored during LVAD and cavaortic shunt. Making the assumption that oxygen demand is constant throughout the experiment, this reflects increased tissue oxygenation during shunting.

Consequently, the addition of a cavaortic shunt into the LVAD circuit enables an increase in CO and thereby systemic DO_2 in a state of severe RV failure.

An increasing number of studies propose alternative strategies to conventional RVADs.^{3,4,12-14} For example, the use of extracorporeal centrifugal pumps as temporary RVADs is reported more frequently.^{12,13} In our model, the idea is to circumvent the pulmonary circulation to unload the RA and RV, relieve further congestion of the pulmonary vascular bed, and allow adequate filling of the LVAD. In a recent study, a similar concept was evaluated with a bypass from the RA to the LA,¹⁵ and this shunt could successfully reverse RV failure, increase MAP, and reduce central venous pressures. However, in that experimental

FIGURE 4. Oxygen delivery (DO_2) and oxygen consumption (VO_2) at baseline, right ventricular failure, LVAD, and LVAD + cavaortic shunt ($n = 10$). LVAD, Left ventricular assist device.

model, the interatrial shunt was not evaluated together with LVAD treatment and the system contained an oxygenator. The inclusion of the shunt into the LVAD circuit adds additional information, which we believe motivates our study. Moreover, Slater and associates¹⁶ have previously demonstrated the feasibility of right-to-left shunting to reduce right-sided filling pressures in experimental RV failure, also without LVAD treatment. They compared central with peripheral shunting with no statistical difference. They concluded that in a situation with normal pulmonary resistance a shunt of almost 50% can be tolerated with sustained SaO_2 . Also, Goldstein and colleagues¹⁷ conducted a similar study with venoarterial shunting, also with measures of arterial saturation only. However, SaO_2 is not a reliable sole indicator of end-organ perfusion, and no estimations of actual systemic DO_2 were performed in these studies. Finally, a case report from Toole and coworkers¹⁸ illustrates a clinical biventricular assist device situation with escalation of pulmonary pressures not responsive to medical management. They successfully used a right-to-left shunt to circumvent the pulmonary circulation.

Although the field of assist device treatment has made great progress over the past 10 years, preoperative RV failure is associated with poor results. Preoperative RV failure reduces survival to transplant in LVAD patients from 85% to approximately 57%.¹⁰ Preoperative prediction of RV failure is still somewhat unreliable, and attempts to develop useful preoperative risk scores are still a struggle. Once present, RV failure is associated with increased perioperative bleeding, more blood transfusions, higher mortality and morbidity, increased incidence of dialysis, worsening of renal and liver function, and increased postoperative lengths of stay.^{19,20} We⁸ have recently shown improved hemodynamics and reduction of central venous pressures in RV failure with volume exclusion of the RV with a modified Glenn connection (shunt from the superior vena cava to the pulmonary artery). However, the concept of a Glenn connection in RV failure requires normal resistance in the pulmonary vascular bed inasmuch as the shunt is not driven mechanically. Thus the Glenn procedure is not an option in patients with increased PVR.

The reestablishment of CO that an LVAD offers without relieving venous congestion may not necessarily avert the clinical state of end-organ failure. There has been a conceptual shift regarding the view of end-organ function in patients with heart failure implying that end-organ function such as liver and kidney is even further worsened by high venous pressures than solely on the impairment of CO. Mullens and associates,²¹ in a study of patients with regular heart failure, showed that preservation of CO without relieving central venous pressures may not avert development of worsening renal function. The addition of the cavaortic shunt into the LVAD circuit in our model reduced the central venous pressures even further than use of the LVAD only. In

RVAD candidates with elevated PVR, it might be difficult to accomplish an adequate reduction of central venous pressures during RVAD treatment. If PVR is even further increased owing to cardiopulmonary bypass and blood transfusion,²² the congestion in the pulmonary vascular bed increases and even the RVAD treatment will resolve in suboptimal loading of the LVAD, inasmuch as the transpulmonary blood flow will be the limiting factor. In these desolate cases, a bypass of both the RV and the pulmonary vascular bed might offer a beneficial clinical situation inasmuch as it will unload the pulmonary circulation and thereby relieve further pulmonary congestion. The clinical situation in which a cavo-aortic shunt could be of use could be pulmonary dysfunction owing to an increase in PVR (nonresponsive to medical treatment), as described in the previously mentioned case report on a patient with a biventricular assist device.¹⁸

The major advantage of the cavo-aortic shunt included in the LVAD circuit, compared with standard RVAD placement, is the simplicity of its removal. Multiple reoperations in these critically ill patients are preferably avoided. In the setting of biventricular failure after cardiopulmonary bypass, the addition of an RA cannula to the LVAD circuit is a very simple and safe method to decompress the RV without the need to use a separate pump and/or oxygenator. There is no need for cannulation of the RV, which can be technically complicated. When the RV recovers, the clamp on the RA cannula enables controlled weaning and the RA cannula can easily be removed. Furthermore, if the oxygen demand increases, it is simple to insert an oxygenator in the system and thereby provide excellent oxygenation support. Moreover, our findings suggest that current guidelines recommending closing of atrial septal defects to prevent shunting and hypoxia can be discussed. Given the overall poor state of health of the typical LVAD candidate, the shortening of the surgical procedure and bypass time is of importance.

Limitations

The major limitation of our study is that the long-term patency of the results cannot be secured in this model of acute RV failure. The effects of shunting on P_{aO_2} and blood pH may not fully manifest during the time course studied. In addition, the use of pressure-volume loops or echocardiography could have allowed for assessment of RV volumes and loading conditions. The Do_2 is calculated based on CO and CaO_2 . A more reliable method to measure the actual perfusion of end organs would secure data regarding the actual state of perfusion.

An additional limitation is that each animal acts as its own control throughout the protocol. This makes the measurements not completely comparable owing to the physiologic changes that might occur in response to the various steps of the protocol. Finally, the cavo-aortic approach

should be compared and validated to a model with classic RVAD or extracorporeal membrane oxygenation.

In conclusion, a cavo-aortic shunt included in the LVAD circuit improved hemodynamics and enabled increased CO, compared with LVAD alone, in an experimental model of RV failure. Moreover, the shunt decreased central venous pressures even further than only LVAD. This approach was feasible with preserved Do_2 . The concept of a right-to-left shunt included in the LVAD circuit can be used as short-term support for RV failure when there is a need for RV support in combination with decompression of a congested pulmonary vascular bed.

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