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Continuous Flow Left Ventricular Assist Device Implant Significantly Improves Pulmonary Hypertension, Right Ventricular Contractility, and Tricuspid Valve Competence

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Abstract

Background—Continuous flow left ventricular assist devices (CF LVAD) are being implanted with increasing frequency for end-stage heart failure. At the time of LVAD implant, a large proportion of patients have pulmonary hypertension, right ventricular (RV) dysfunction, and tricuspid regurgitation (TR). RV dysfunction and TR can exacerbate renal dysfunction, hepatic dysfunction, coagulopathy, edema, and even prohibit isolated LVAD implant. Repairing TR mandates increased cardiopulmonary bypass time and bicaval cannulation, which should be reserved for the time of orthotopic heart transplantation. We hypothesized that CF LVAD implant would improve pulmonary artery pressures, enhance RV function, and minimize TR, obviating need for surgical tricuspid repair.

Methods—One hundred fourteen continuous flow LVADs implanted from 2005 through 2011 at a single center, with medical management of functional TR, were retrospectively analyzed. Pulmonary artery pressures were measured immediately prior to and following LVAD implant. RV function and TR were graded according to standard echocardiographic criteria, prior to, immediately following, and long-term following LVAD.

Results—There was a significant improvement in post-VAD mean pulmonary arterial pressures (26.6 ± 4.9 vs. 30.2 ± 7.4 mmHg, p = 0.008) with equivalent loading pressures (CVP = 12.0 ± 4.0 vs. 12.1 ± 5.1 p = NS). RV function significantly improved, as noted by right ventricular stroke work index (7.04 ± 2.60 vs. 6.05 ± 2.54, p = 0.02). There was an immediate improvement in TR grade and RV function following LVAD implant, which was sustained long term.

Conclusion—Continuous flow LVAD implant improves pulmonary hypertension, RV function, and tricuspid regurgitation. TR may be managed nonoperatively during CF LVAD implant.

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As perioperative management of patients following ventricular assist device (VAD) implant has evolved, there has been a marked improvement in short- and mid-term survival following LVAD.[1,2] Increasingly, mechanical support is being considered by both tertiary care and community heart failure specialists.[3] We are beginning to gain a clearer understanding of the optimal timing and contraindications for continuous flow left ventricular assist device (CF LVAD) therapy,[4,5] but many questions need to be resolved as this remains a relatively novel field. In the current era, without long-term continuous flow solutions for the right ventricle (RV), we are left relying on the native RV to provide pulmonary blood flow and LVAD preload. Several scoring systems have been devised to determine whether a patient will need biventricular mechanical support, but these data are based largely on pulsatile devices.[6-8] Incorrectly assessing RV function, and subsequent delayed implant of an RVAD, is associated with increased morbidity and mortality. In the case of a CF LVAD as destination therapy, the need for long-term RV support is not feasible in the present era.[9-11] Recent data suggest a low risk of RV failure following CF LVAD implant.[12] Our understanding of the alterations in right ventricular function, tricuspid valve competence—often a surrogate marker of RV failure, and pulmonary pressures following insertion of a continuous flow LVAD are limited. This often presents both a pre-operative and an intraoperative challenge. Namely, will a patient with moderate RV dysfunction tolerate a continuous flow LVAD? Should the tricuspid valvular incompetence be surgically corrected—both increasing cardiopulmonary bypass (CPB) time and disrupting the native tissue planes that may be needed for subsequent bicaval cannulation for orthotopic heart transplant? Will unloading of the pulmonary circulation lower pulmonary artery pressures and possibly reverse elevated pulmonary vascular resistance (PVR) that may traditionally preclude a patient from subsequent heart transplant? The answers to these questions are vital to further our understanding of the postcontinuous flow LVAD physiology and the intraoperative and perioperative management necessary to improve outcomes for this operation.

MATERIALS AND METHODS

Study design

From 1993 through 2011 we implanted 442 VADs at the University of Pennsylvania. In order to quantify the impact of a continuous flow LVAD on decompression of the pulmonary circulation, right ventricular function, and tricuspid valve competence, a retrospective review of a prospectively collected database was performed for all patients undergoing isolated implantation of continuous flow LVADs without concomitant tricuspid valve repair or right ventricular support (n = 114). Four patients underwent tricuspid valve repair at the time of LVAD implant and were excluded from the study (3 = torrential TR, 1 = moderate TR). Implanted continuous flow devices included: HeartMate II = 95, HeartWare = 13, VentrAssist = 6. Patient demographics, comorbidities, etiology of heart failure, invasive hemodynamics, and transesophageal echocardiography focusing on right ventricular function and tricuspid valve competence were examined preoperatively (Table 1). Invasive hemodynamics were measured postoperatively to quantify central venous pressure, pulmonary artery pressures, mixed venous oxygen saturation, cardiac output, and cardiac index. Hemodynamic measurements were made immediately upon arrival in the

ICU. Right ventricular function and tricuspid valve competence were quantified with transthoracic echocardiography prior to discharge and on mid-term follow-up. The study was approved by the Institutional Review Board of the University of Pennsylvania. After initial data accrual, all patient identifiers were removed from the database, prior to statistical analysis.

Operative technique

All patients underwent standard median sternotomy for exposure. The patients were placed on standard CPB utilizing ascending aortic arterial cannulation and dual-stage venous drainage via the right atrium. Patients with concomitant aortic valve insufficiency that required intervention and hence cross-clamp and cardioplegic arrest were excluded from the study. Moreover, patients who underwent intervention on the tricuspid valve were also excluded from the study. Once the LVAD was implanted, patients were weaned from CPB with the aid of the VAD. LVAD flow was optimized with transesophageal echocardiography. Optimum flow was determined by midline septal position, LV decompression, absence of mitral regurgitation, and none to minimal aortic valve opening.

Hemodynamic calculation

Invasive hemodynamic quantification was performed with a continuous cardiac output pulmonary artery catheter. The catheter was placed sterilely with ultrasound and echocardiographic guidance. The transducers were leveled with the middle of the heart prior to acquisition of all values. Mixed venous oxygenation (SVO_2) was confirmed by measurement of pulmonary venous arterial oxygen saturation in the laboratory. Cardiac output obtained from the continuous cardiac output box was confirmed by manual calculation utilizing the Fick equation. Right ventricular stroke work index (RVSWI) was calculated as SVI × (MPAP − CVP) × 0.136, where SVI is the stroke volume index, MPAP is the mean pulmonary artery pressure, and CVP is the central venous pressure.

Assessment of right ventricular function and tricuspid valve competence

Right ventricular function and tricuspid valve competence were quantified with transesophageal echocardiography immediately prior to the start of the operation. Function was subsequently quantified immediately following implant and optimization of LVAD settings, with a closed chest, prior to leaving the operating room. RV function was initially determined by a cardiac anesthesiologist board certified in echocardiography and confirmed by an independent cardiologist evaluating the same study in a separate setting. Standard short-axis, four-chamber long-axis, regurgitant valvular vena contracta, and tricuspid annular plane excursion were all considered in determining right ventricular and tricuspid valve function. In order to avoid undue sedation, mid-term follow-up and analysis of right ventricular function and tricuspid valvular competence were obtained with transthoracic echocardiography.

Statistical analysis

Measurements for quantitative variables and continuous variables were expressed as continuous mean ± SD. Categorical variables were presented as proportions. Differences

between groups were expressed using the Fisher's exact test for comparison of categorical data, the independent Student's *t*-test for normally distributed variables, and the Mann–Whitney *U* test was for comparison of non-normally distributed variables. Statistical analysis was performed using SPSS, version 18.0 (SPSS Inc., Chicago, IL, USA). A p-value of <0.05 was considered statistically significant for all analyses.

RESULTS

Overall, 40 patients (34.7%) subsequently went on to orthotopic heart transplant. Cumulative one-year survival for this cohort was 81%. None of the patients in this study required long-term RVAD support. Echocardiographic follow-up was performed at 3 (n = 71), 6 (n = 63), and 12 (n = 52) months. At follow-up echo four patients were on oral pulmonary vasodilators; two of these patients were preoperatively on pulmonary vasodilator therapy. No patients required inotropes on mid-term follow-up. None of the patients were mechanically ventilated on mid-term follow-up echocardiogram.

Continuous flow left ventricular assist devices significantly ameliorate pulmonary hypertension

Quantitative analysis of invasive hemodynamics revealed a marked and statistically significant reduction in mean pulmonary artery pressure immediately following implant of an isolated continuous flow LVAD. On average, mean pulmonary artery pressure decreased from 30.2 ± 7.4 to 26.6 ± 4.9 mmHg (p = 0.008), when the entire cohort of patients was analyzed. Preload was equivalent pre- and post-operatively, ensuring equivalent loading conditions (central venous pressure = 12.1 ± 5.1 vs. 12.0 ± 4.0 mmHg, p = 0.9). When isolated analysis was performed on patients with pulmonary hypertension (mean PA pressure ≥30 mmHg), there is a mean decrease of almost 12 mmHg (from 37.6 ± 5.3 to 26.1 ± 4.6 mmHg, p = 0.000000000003) following continuous flow LVAD implant. These findings demonstrate a significant decompression of the pulmonary circulation and subsequent decrease in pulmonary pressures following implant.

Impact of left ventricular mechanical support on right ventricular function

The critical question that has remained unclear is what physiologic impact a continuous flow LVAD will have on right ventricular dysfunction. The answer to this question remains paramount in deciding whether a patient will tolerate isolated left ventricular circulatory support or will need concomitant right ventricular support. Based upon our center's experience, CF LVAD therapy significantly improves right ventricular function as quantified by right ventricular stroke work index (7.04 ± 2.60 vs. 6.05 ± 2.54 g m/m^2/beat, p = 0.02). Echocardiographically graded severity of RV dysfunction improved significantly with LVAD implant (Table 2). This improvement was sustained mid-term at 3, 6, and 12 months (Table 3). These findings suggest that patients with marked RV dysfunction should still be evaluated for long-term mechanical circulatory support.

Tricuspid valve competence improves following implant of a continuous flow left ventricular assist device

Tricuspid valve competence appears to significantly improve immediately following CF LVAD implant (Table 2). Moreover, this improvement in valvular competence is maintained at three, six, and 12 months postimplant (Table 3), and continues to improve over time. In the absence of torrential TR (i.e., moderate–severe), it may be possible to expectantly manage tricuspid regurgitation, especially in the case of VAD as bridge to transplant.

DISCUSSION

There has been a significant shift in our management of heart failure in the post-REMATCH (Randomized Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure) era. When compared to medical management, mechanical circulatory support has proven to be far superior in regard to survival and quality of life.[13-16] As the technology advances and devices continue to demonstrate increased reliability and durability, the rate of LVAD implant should continue to increase. But, in order to optimize outcomes and patient survival there are several critical questions that need to be addressed. One of the major concerns for the majority of heart failure physicians is whether a patient with pulmonary hypertension and RV dysfunction will tolerate an isolated continuous flow LVAD. In the current era, where we are limited by long-term left-sided mechanical circulatory support, the answer to this question will often determine whether a patient can be offered life-saving therapy or whether he or she is palliated medically.

Based on the findings from this study, it is evident that active decompression of the left ventricle with a CF LVAD will further decompress the pulmonary circulation. We were very pleased to note the marked improvement in pulmonary artery pressures that result from LV decompression with a more significant benefit noted with greater pulmonary hypertension. This physiologic benefit has clinical implications for both destination and bridge-to-transplant LVAD therapy. For the destination cohort, a decompression of the pulmonary circulation will decrease afterload on the RV and improve RV function, as we have demonstrated. Long-term maintenance of RV function is essential for optimal long-term LVAD outcomes. Additionally, decompressing the RV can minimize central venous pressure, thereby significantly reducing venous congestion and positively impacting end organ function (i.e., hepatic, renal, mesenteric).

The improvement in pulmonary pressures may broaden the degree of right ventricular dysfunction that is acceptable and safe for isolated continuous flow LVAD implant. A large percentage of continuous flow LVADs are implanted as a bridge to heart transplant. Decompression of the pulmonary circulation acts to diminish pulmonary hypertension and potentially reverse elevated PVR that is detrimental to graft function. In fact, many patients are not candidates for heart transplant due solely to an elevated PVR. Traditional medical therapy, including phosphodiesterase inhibitors, nitric oxide, prostacyclin, and sildenafil, has had variable results in reversing PVR.[17] Limited studies have demonstrated a reversibility of fixed PVR following implant of a CF LVAD.[18-21] Expanding LVAD indications to include patients with an elevated PVR and concomitant severe LV dysfunction with the goal of

temporary VAD insertion to reverse fixed PVR and allow optimal transplant outcomes should be considered.

Interestingly, we also noted a significant improvement in severity of tricuspid regurgitation following CF LVAD implant. There has been a lot of controversy and disagreement about whether to surgically correct tricuspid regurgitation at the time of LVAD implant. All authors will agree that it does increase CPB time. But, there is disagreement as to whether it will be beneficial long term.[2,22-25] The group at the Mayo Clinic, a highly experienced VAD center, has demonstrated beneficial right ventricular reverse remodeling associated with an elimination of tricuspid regurgitation, though a significant benefit from a functional standpoint was not demonstrated.[25] It should be noted that the patients who underwent tricuspid valve surgery in this study likely had significant TR hemodynamically based upon significantly elevated CVP values. In their experience evaluating stable patients who underwent HM II LVAD implant with or without concomitant tricuspid valve surgery, the Mayo clinic group has additionally published improved RV performance, LV unloading, and improved mitral regurgitation following LVAD implant based upon echocardiographic analysis three and six months postsurgery.[26] They have additionally noted improvements in tricuspid regurgitation only following TV surgery. The Duke group has recently demonstrated, in their series of 61 patients with TR, a significant improvement in RV function following surgical correction of tricuspid regurgitation.[27] However, they did not note any difference in duration of hospitalization, need for rehospitalization, 30-day mortality or one-year mortality between patients with and without concomitant triscupid valve surgery at the time of LVAD implant. Triscupid valve surgery was associated with a 68 minutes longer CPB time.

The Northwestern group has demonstrated that prolonged CPB time associated with tricuspid valve repair has been associated with enhanced bleeding, transfusion requirement, and worse renal function without a benefit to long-term right ventricular function.[22] Collectively, whether the benefits of tricuspid valve repair outweigh the added risk, though minimal, is difficult to ascertain. From the data presented in this study, it appears that there is an improvement in RV function and probably TR grade following CF LVAD implant; therefore, surgeons should carefully weigh the risks and benefits before making a decision. However, based upon these isolated small series from various centers with varying management styles, it may be difficult to make a conclusive decision without the benefits of a large, prospective multicenter study. Additionally, overly aggressive tricuspid reduction annuloplasty could risk creating functional tricuspid stenosis and compromising hepatic and renal function.

Tricuspid valve regurgitation is often a surrogate measure of RV dysfunction. The decision to surgically correct tricuspid valve incompetence during CF LVAD implant can be difficult to make. Operative repair of the tricuspid valve is associated with prolonged CPB time, potential AV node dysfunction, and disruption of native tissue plans surrounding the inferior and superior vena cava that are utilized during bicaval orthotopic heart transplant. Most surgeons would agree that torrential TR should be corrected at the time of LVAD implant. However, the optimal management of less severe degrees of TR remains unclear.

In our practice, we have liberalized patient selection for CF LVAD therapy based upon our and others' prior work determining the risk factors for RV failure post LVAD implant.[6] We have found that patients with marked RV dysfunction can be reasonable candidates for CF LVAD implant. Aggressive post-operative medical management including afterload reduction with pulmonary vasodilators (i.e., inhaled prostacylin or nitric oxide), inotropic therapy (i.e., milrinone), optimization of volume status, and RV unloading achieved with a CF LVAD can allow a patient with marked RV dysfunction to benefits from LVAD therapy. The few patients that fail to have enough RV reserve can usually be bridged for a short period of time with a temporary CF RVAD tunneled through the chest wall.[28] Over the ensuing post-operative period the RV will often regain function, allowing RVAD explants and isolated LVAD DT therapy. This strategy has been studied by Dr. Sawa and collegues and demonstrated to be successful, without compromising survival.[29] A failure to expand LVAD therapy to this population will leave thousands of heart failure patients without options for therapy.[30,31] As the field of mechanical circulatory support continues to evolve, we need to find means of providing therapy to all patients with end-stage heart failure. The advent of continuous flow, biventricular support will ensure that severe RV failure is no longer a contraindication to LVAD implant. In the meantime, we need to ensure that any and all patients who will tolerate an isolated LVAD, even with moderate or moderate to severe RV dysfunction, are offered this life-saving therapy.

Study limitations

By the nature of the study design, data collection was not complete for every variable nor as predictive as a randomized prospective study. Additionally, this study was performed at a single institution and therefore incorporated some degree of institutional selection bias.

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TABLE 1

Preoperative Patient Demographics

Variable	
Age (years)	56.4 ± 13.0
Body mass index (kg/m^2)	27.0 ± 5.0
Diabetes mellitus (%)	51.3%
Pulmonary hypertension (%)	41.7%
Chronic obstructive pulmonary disease (%)	18.3%
Prior smoker (%)	50.4%
Hypertension (%)	67.0%
Hypercholesterolemia (%)	75.7%
Carotid stenosis (%)	3.5%
Prior stroke (%)	14.8%
Chronic renal insufficiency (%)	41.7%
Prior cardiac surgery	33.9%
Intraaortic balloon pump	14.0%
Intubated	9.6%
Preoperative inotropic support (epinephrine)	4.7%
Preoperative inotropic support (milrinone)	73.8%

TABLE 2
Pre- and Postoperative Hemodynamic and Laboratory Variables

Variable	Preimplant	Postimplant	P-Value
Heart rate (beats/min)	88 ± 14	96 ± 12	0.0004
Arterial blood pressure (mean, mmHg)	72.5 ± 9.1	76.0 ± 9.5	0.03
Central venous pressure (mmHg)	12.1 ± 5.1	12.0 ± 4.0	0.8
Pulmonary artery pressure (mean, mmHg)	30.2 ± 7.4	26.6 ± 4.9	<0.00000001
Cardiac index (L/min^{-1} m^{-2})	2.10 ± 0.4	3.12 ± 0.5	<0.00000001
Right ventricular dysfunction (%)			0.02
None	7.07%	12.50%	
Mild	23.89%	36.46%	
Moderate	43.37%	39.58%	
Severe	25.67%	11.45%	
Right ventricular stroke work index (g m^2/beat)	6.05 ± 2.54	7.04 ± 2.60	0.02
Tricuspid regurgitation (%)			0.0004
None	9.26%	4.35%%	
Mild	36.11%	65.22%	
Moderate	44.44%	27.17%	
Severe	10.19%	3.26%	
Sodium	135 ± 4	137 ± 3	0.0008
Total bilirubin	1.37 ± 0.61	1.80 ± 1.63	0.2
Aspartate aminotransferase	44.80 ± 27.55	79.37 ± 77.55	0.1
Alanine aminotransferase	40.29 ± 24.03	45.92 ± 35.92	0.4
Creatinine	1.50 ± 0.47	1.30 ± 0.46	0.01

Postoperative hemodynamic variables were acquired immediately upon arrival to the intensive care unit. Postoperative echocardiography parameters were determined immediately prior to leaving the operating room (n = 114).

TABLE 3

Mean Preoperative, Immediate Postoperative, and Follow-Up Right Ventricular Dysfunction and Tricuspid Regurgitation Following Continuous Flow Left Ventricular Assist Device Implant

	Pre-Operative (n = 114)	Post-Operative (n = 114)	3-Month Follow-Up (n=71)	6-Month Follow-Up (n = 63)	12-Month Follow-Up (n = 52)	P = (Post-Op vs. Pre-Op)
Right ventricular dysfunction (all patients)	2.09 ± 0.64	1.65 ± 0.71	1.67 ± 0.77	1.36 ± 0.88	1.64 ± 0.79	0.001
Right ventricular dysfunction (pre-op moderate or severe, n = 58)	2.46 ± 0.49	1.89 ± 0.55	1.79 ± 0.74	1.48 ± 0.80	1.75 ± 0.80	<0.00001
Tricuspid regurgitation (all patients)	1.48 ± 0.75	1.24 ± 0.50	1.05 ± 0.53	1.04 ± 0.42	0.75 ± 0.58	0.001
Tricuspid regurgitation (pre-op moderate or severe, n = 59)	2.17 ± 0.28	1.38 ± 0.60	1.14 ± 0.61	1.17 ± 0.47	0.71 ± 0.57	<0.000001

A subgroup of patients with moderate or severe right ventricular dysfunction and tricuspid regurgitation is analyzed in addition to the entire cohort (0 = none, 1 = mild, 2 = moderate, 3 = severe).