Published in final edited form as:

J Heart Lung Transplant. 2010 February ; 29(2): 201–208. doi:10.1016/j.healun.2009.09.013.

Pulsatile left ventricular assist device support as a bridge to decision in patients with end-stage heart failure complicated by pulmonary hypertension

Pradeep K. Nair, MD^a, Robert L. Kormos, MD^{b,c}, Jeffrey J. Teuteberg, MD^a, Michael A. Mathier, MD^a, Christian A. Bermudez, MD^{b,c}, Yoshiya Toyoda, MD, PhD^{b,c}, Mary Amanda Dew, PhD^{c,d}, and Marc A. Simon, MD^{a,e}

^aCardiovascular Institute, University of Pittsburgh, Pittsburgh, Pennsylvania.

^bHeart, Lung, Esophageal Surgery Institute, University of Pittsburgh, Pittsburgh, Pennsylvania.

^cArtificial Heart Program, University of Pittsburgh, Pittsburgh, Pennsylvania.

^dDepartments of Psychiatry, Psychology, Epidemiology and Biostatistics, University of Pittsburgh, Pittsburgh, Pennsylvania.

^eDepartment of Bioengineering, University of Pittsburgh, Pittsburgh, Pennsylvania.

Abstract

BACKGROUND—Severe pulmonary hypertension (PH) in heart failure (HF) is a risk factor for adverse outcomes after heart transplantation (HTx). Left ventricular assist devices (LVADs) improve pulmonary hemodynamics, but our understanding of the degree of improvement and the effect on outcomes is still evolving.

METHODS—We reviewed invasive pulmonary hemodynamics from 58 consecutive patients receiving LVAD support as a bridge to HTx from 1996 to 2003. The primary outcome was change in baseline transpulmonary gradient (TPG) during LVAD support and after HTx/recovery. The secondary outcome was post-HTx survival.

RESULTS—All patients (age, 49 ± 14 years, 79% male, 40% ischemic) received a pulsatile LVAD (median support, 97 days; interquartile range [IQR], 31–222). Hemodynamic measurements were obtained at baseline (median, 1 day; IQR, 1–3), during early (median, 1 day; IQR, 0–4) and late (median, 75 days; IQR, 24–186) LVAD support, and after HTx/recovery (median, 28 days; IQR, 17–40). Improvement in TPG occurred throughout LVAD support and was sustained after HTx/recovery. Levels of TPG reductions in patients with a baseline TPG in the highest quartile (14.1–26.0 mm Hg) were 8.6 ± 3.5 vs 6.5 ± 3.1 mm Hg in the lowest quartile (2.0–7.7 mm Hg) during LVAD support ($p = 0.102$), with 90% vs 100% 30-day post-HTx survival ($P = 0.113$).

CONCLUSION—Pulmonary hemodynamics and post-HTx survival were similar after pulsatile LVAD support in patients with and without pre-implant PH. LVAD support may be a useful strategy to reverse PH in carefully selected patients, thus improving candidacy for HTx.

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Reprint requests: Marc A. Simon, MD, FACC, Assistant Professor of Medicine, Cardiovascular Institute, University of Pittsburgh, Scaife Hall S554, 200 Lothrop St, Pittsburgh, PA 15213. Telephone: 412-647-1174. Fax: 412-647-3913. simonma@upmc.edu.

Disclosure statement

None of the other authors has a financial relationship with a commercial entity that has an interest in the subject of the presented manuscript or other conflicts of interest to disclose.

Keywords

heart-assist devices; pulmonary hypertension; heart failure; transplantation; outcomes; adult

Heart transplantation (HTx) is an established therapy for select patients with end-stage heart failure (HF).¹ Unfortunately, the demand for donor hearts far exceeds the supply. With progressive organ shortage and wait times comes a higher chance of developing pulmonary hypertension (PH), a major risk factor for right ventricular (RV) failure after HTx.^{2–7}

Chronic elevations of left ventricular filling pressures lead to PH, which if long-standing can result in histologic changes in the pulmonary vasculature.⁸ These changes can be reversible or irreversible after aggressive vasodilator therapy. The reversibility of PH is determined by the ability to lower the transpulmonary gradient (TPG) or pulmonary vascular resistance (PVR) during the infusion of selective pulmonary vasodilators and is associated with a decreased risk of RV failure after HTx.^{2,9–12} Consensus is lacking regarding the absolute cutoff for PVR and TPG. However, an association with the greatest risk has been suggested for PVR >2.5 Wood units (WU) and TPG >12 mm Hg before HTx.^{3,9,13}

Left ventricular assist devices (LVADs) have an established history as bridges to HTx.^{14,15} They lower pulmonary pressures by unloading the LV.^{16–20} Although data have emerged on the efficacy of LVADs to improve pulmonary hemodynamics and successfully bridge patients to HTx, several questions remain unanswered. In particular, what degree of PH can be successfully bridged to HTx with a LVAD, and what are the transplant outcomes of such patients? The role of pre-operative intraaortic balloon pumps (IABP) is as yet uncertain. This study evaluated the pulmonary hemodynamic response to pulsatile LVAD support and the effect on clinical outcomes after HTx.

Methods

Study design and data collection

This retrospective study was approved by the University of Pittsburgh's Institutional Review Board. Data on all patients who receive mechanical support are prospectively entered into our transplantation program's Transplant Patient Management System. This study included all patients aged >16 years with chronic end-stage HF who underwent LVAD support from 1996 to 2003 as a bridge to HTx. Exclusion criteria were LVAD implantation within 30 days of an acute myocardial infarction or post-cardiotomy failure, congenital heart disease, or insufficient hemodynamic measurements.

Pulmonary hemodynamics were analyzed from right heart catheterization data (1) immediately before LVAD implant (defined as baseline), (2) the earliest time after LVAD implant, (3) the latest time on LVAD support (closest to explant), and (4) 1 month after HTx/recovery. If a patient required an IABP immediately before LVAD implantation, hemodynamic measurements were also obtained before IABP insertion.

Patients in the cohort received 1 of 4 LVAD systems: Thoratec LVAD, IVAD, or HeartMate XVE (all Thoratec Corp, Pleasanton, CA), or Novacor LVAS (World Heart Corporation, Oakland, CA).

Statistical analysis

The primary outcome was the change in the TPG. Secondary outcomes were changes in mean pulmonary artery pressure (MPAP) and PVR, as well as survival to HTx and after HTx. Data are presented as mean $\pm$ standard deviation. Variables with a wide skew are

presented as median with interquartile range (IQR). The sample was divided into 4 groups for analysis, based on quartiles of TPG values. Baseline characteristics across the 4 groups were compared using 1-way analysis of variance (ANOVA) for continuous variables and chi-square or Fisher exact tests for categorical variables. Repeated measures ANOVA was used to assess for change in hemodynamics over time. Bonferroni post hoc pairwise comparison was used to evaluate differences of baseline to early and late follow-up at each time point. Survival was assessed by Kaplan-Meier analysis with a log-rank test. Statistical analysis was performed using SPSS 15 software (SPSS Inc, Chicago, IL).

Results

Baseline characteristics

Between 1996 and 2003, 75 patients received a pulsatile LVAD, and 58 patients remained for analysis after 17 patients were excluded (8 acute myocardial infarctions, 6 post-cardiotomy failures, 1 congenital heart disease, 2 insufficient hemodynamic measurements). Characteristics of the cohort are described in Table 1. Four patients received an unplanned temporary RVAD at the time of LVAD implant (Table 2). Their baseline right atrial (RA) pressure (13.8 ± 3.4 mm Hg), MPAP (37.3 ± 4.6 mm Hg), TPG (8.8 ± 5.7 mm Hg), and PVR (2.77 ± 1.91 WU) were similar to patients not requiring temporary RV support (RA, 10.1 ± 5.6 mm Hg; MPAP, 34.4 ± 8.2 mm Hg; TPG, 11.9 ± 5.1 mm Hg; PVR, 2.81 ± 1.52 WU; $P = \text{NS}$ for all).

Devices used were Novacor LVAS in 30 (52%), Thoratec LVAD in 15 (26%), Heartmate XVE in 12 (20%), and Thoratec VAD in 1 (2%). Median duration of support was 97 days (IQR, 31–222 days). Overall, 46 patients (79%) survived to HTx, 4 (7%) patients were weaned from LVAD after myocardial recovery, and 8 (14%) died during LVAD support.

Baseline hemodynamic measurements were obtained before LVAD implant (median, 1 day; IQR, 1–3 days). Hemodynamic variables were again measured during early (median, 1 day after implant; IQR, 0–4 days) and late (median, 75 days after implant; IQR, 24–186 days) LVAD support. Finally, hemodynamic measurements were collected after HTx/recovery (median, 28 days after explant; IQR, 17–40 days).

All patients required inotrope support before LVAD implant. Milrinone and/or dobutamine use decreased from 39 (67%) in early LVAD support to 5 (9%) in late LVAD support ($p < 0.001$).

Changes in pulmonary hemodynamics with LVAD support

Baseline MPAP, TPG, and PVR values were 34.6 ± 8.0 mm Hg, 11.7 ± 5.2 mm Hg, and 2.81 ± 1.53 WU, respectively. Compared with baseline, hemodynamic variables improved during early and late LVAD support (Figure 1). Reductions in MPAP, TPG, and PVR occurred early with LVAD support; however, only MPAP improved with ongoing support ($p = 0.028$; early vs late LVAD support). TPG and PVR improvements were sustained after HTx/recovery (Figure 2).

Effect of baseline pulmonary hemodynamic response to LVAD support

Characteristics of the cohort stratified by quartiles of baseline TPG are described in Table 1. Except for pulmonary hemodynamics, baseline characteristics, including RA pressure and milrinone usage, were similar across quartiles. Survival to HTx/recovery was unaffected by the degree of baseline PH. Baseline characteristics of the 25 patients in TPG quartiles 3 and 4 surviving to HTx/recovery were similar ($p = \text{NS}$ for all) to the 5 patients in the same group who died during LVAD support.

Survivors to HTx/recovery with the worst PH (quartile 4) had an early and sustained improvement in TPG with LVAD support (Figures 3 and 4). Patients in quartiles 2 and 3 achieved reductions in TPG before HTx/recovery. In addition, patients in the highest and lowest quartile had similar TPG levels before HTx/recovery (8.6 ± 3.5 vs 6.5 ± 3.1 mm Hg, respectively; $p = 0.102$).

Effect of IABP support on pulmonary hemodynamic response to LVAD

IABP support was initiated for a mean duration of 4 ± 3 days in 42 patients (72%) immediately before LVAD implant. Among the 8 patients who died during LVAD support, 7 received an IABP. IABP usage did not differ across quartiles of TPG (Table 1).

Values for MPAP (40.4 ± 8.7 mm Hg), TPG (13.0 ± 6.0 mm Hg), and PVR (3.58 ± 1.98 WU) were measured before IABP insertion (median, 6 days; IQR, 3–14). IABP support resulted in a modest improvement in MPAP (35.0 ± 7.8 mm Hg, $p < 0.001$) and PVR (2.63 ± 1.43 WU, $p = 0.001$), but not TPG (11.8 ± 4.6 mm Hg, $p = 0.158$). The 16 patients who did not receive an IABP had higher levels of serum albumin (3.5 ± 0.2 vs 3.2 ± 0.4 mg/dl, $p = 0.043$) and lower levels of serum creatinine (1.1 ± 0.4 vs 1.4 ± 0.5 mg/dl, $p = 0.01$) compared with those who required support. However, the baseline MPAP (34.3 ± 8.6 mm Hg, $p = 0.863$), TPG (11.7 ± 6.6 mm Hg, $p = 0.992$), and PVR (3.32 ± 1.74 WU, $p = 0.112$) of patients without an IABP were similar to those who received support.

Among the 35 survivors to HTx/recovery who received an IABP, TPG and PVR improved by the late LVAD support period but was not sustained after HTx/recovery (Figure 5). However, MPAP improvements (compared with baseline) were sustained after HTx/recovery. In contrast, the 15 survivors to HTx/recovery who did not receive IABP support achieved early improvements in pulmonary hemodynamics that were sustained throughout LVAD support and after HTx/recovery (Figure 6).

Effect of LVAD support on pulmonary hemodynamics based on survival to HTx or recovery

The 50 survivors to HTx/recovery showed improvement in MPAP, TPG, and PVR during early (MPAP, 35.2 ± 7.9 vs 23.1 ± 8.6 mm Hg; TPG, 11.5 ± 5.2 vs 8.6 ± 3.5 mm Hg; PVR, 2.80 ± 1.57 vs 1.71 ± 0.80 WU) and late (MPAP, 20.0 ± 6.8 mm Hg; TPG, 7.6 ± 3.2 mm Hg; PVR, 1.46 ± 0.75 WU) LVAD support compared with baseline ($p < 0.005$ for all). In contrast, the 8 patients who died on LVAD support did not show improvement in TPG or PVR during early (TPG, 12.7 ± 5.1 vs 10.2 ± 6.3 mm Hg; PVR, 2.86 ± 1.40 vs 1.80 ± 1.02 WU) or late (TPG, 10.8 ± 5.4 mm Hg; PVR, 2.30 ± 1.20 WU) mechanical support (baseline vs early/late LVAD support, $p = \text{NS}$ for all). Although mild improvement occurred in MPAP early after LVAD support, it was not sustained during long-term follow-up (31.3 ± 8.7 vs 25.9 ± 6.3 mm Hg, baseline vs late LVAD support; $p = 0.405$).

Survival and recovery after HTx

Fifty patients (86%) survived to HTx or recovery. The 30-day post-HTx/recovery survival of 44 patients (88%) was similar ($p = 0.113$) regardless of baseline TPG (Table 1). Among the early post-HTx/recovery deaths (<30 days), 4 patients in quartile 3 had the highest mortality that was attributed to failure of the donor heart to initiate spontaneous electrical activity (baseline TPG, 12.0 mm Hg), severe donor LV failure with normal RV function (Table 2), embolic stroke, and anoxic brain injury at 1, 1, 2, and 13 days after HTx, respectively. One patient in quartile 2 died of an embolic stroke 2 days after device explant for myocardial recovery. One patient in quartile 4 sustained an intracranial hemorrhage 23 days after HTx.

Overall, 5-year survival after HTx was 68%. Stratified by TPG quartiles, survival was 83%, 85%, 29%, and 82% for quartiles 1 to 4, respectively ($p = 0.001$). Among the 10 deaths occurring between 30 days and 5 years after HTx, 6 were directly attributed to infections. Six patients in quartile 3 died of (1) systemic infections from *Aspergillus fungemia*, vancomycin-resistant enterococcal sepsis, and pneumococcal sepsis at 96, 108 and 1123 days after HTx, respectively, (2) chronic allograft rejection 818 days after HTx, (3) post-HTx lymphoproliferative disease, and (4) suicide. The only death from TPG quartile 4 occurred 32 days after HTx and was a result of vancomycin-resistant enterococcal sepsis from an infected abdominal wound discovered post-mortem. No patients were reported to have RV failure after HTx.

Discussion

Our results provide further insight and expand our understanding of PH reversibility with LVAD support. Stratifying baseline PH severity by TPG quartiles resulted in several findings of clinical relevance. First, PH improves with pulsatile LVADs regardless of baseline pulmonary pressures, up to a TPG of 26 mm Hg. This is clinically significant because it suggests LVAD support may be considered as a bridge to candidacy among patients not initially considered for HTx due to PH. Second, hemodynamics in patients with the most severe PH improved early after LVAD implant (median, 1 day) and persisted with longer support (median, 75 days). Thus, if significant improvement is seen, it occurs within a reasonable timeframe to make decisions about activation on the transplant list; whereas, a lack of improvement by 2 months of support may be a marker for poor outcomes.

Several other clinically important findings should be noted. Baseline TPG was not associated with survival on LVAD, thus the degree of PH reported would appear not to be a contraindication to LVAD support. This is further supported by a lack of difference in several clinical variables known to increase the risk of death during LVAD support between survivors to HTx and non-survivors with high TPG.²¹ Finally, hemodynamic improvements in patients with severe baseline PH were sustained after HTx and no allograft RV failure occurred.

The effectiveness of IABP support before LVAD, specifically among patients with PH, remains uncertain. LVADs were more effective than IABP support for improving PH. Patients who received an IABP had a higher mortality on LVAD and required a longer support time to achieve PH reductions. Although this may reflect that the IABP patients are more acutely ill, the incidence of IABP usage was not different across TPG quartiles. Further investigation is necessary to establish the role of IABP support before LVAD implantation.

Reducing PVR to less than 2.5 WU may decrease the risk of death after HTx.^{9–11,22,23} Vasoreactive medications have been used to reverse PH before HTx but are not always successful.^{9–10,24–27} Some evidence suggests that LVADs may reverse medically refractory PH, which is a major risk factor for increased death after HTx.^{5,17–20} Our study did not specifically identify patients with medically refractory PH, but did provide longer follow-up of hemodynamics and post-HTx survival.

The 5-year post-HTx survival in our cohort was 68%, which is similar to published registry data.²⁸ Long-term survival was poor in patients from TPG quartile 3, but similar in quartiles 1, 2, and 4. This may reflect selection bias, but the observed deaths appeared to be unrelated to pre-existing PH. During the study period, our center performed transplants in 51 patients with a baseline TPG exceeding 14 mm Hg supported with inotropes rather than with a LVAD. Survival in these patients was 92% at 30 days and 63% at 5 years. Although our data

cannot conclusively establish that all patients with a high TPG will benefit from LVAD support, our study patients in the highest quartile of TPG had 82% survival at 5 years compared with those with elevated TPG supported with inotropes alone.

The transplant strategy for these patients is critical. We use several strategies as deemed necessary on a case-by-case basis. In particular, conservative bottom weight limits for donors are used (10% below recipient weight), with a preference for male donors. All patients receive inhaled nitric oxide immediately intraoperatively and are weaned off over 24 to 48 hours. Oversizing is occasionally used as well.

Four patients required an unplanned temporary RVAD (mean support, 6 ± 1 days) at the time of LVAD implantation due to severe RV dysfunction failing to provide enough pre-load to the LV. Baseline hemodynamic variables of these 4 patients (2 from quartiles 1 and 3, respectively) were similar to the remaining cohort (Table 2). All survived to HTx/recovery. However, 1 patient (quartile 3) died immediately after HTx due to severe donor LV failure despite normal RV function, and another death (quartile 1) was caused by acute allograft rejection 1 year after HTx (2 years after LVAD explant for myocardial recovery) because of non-compliance with immunosuppression. Although it may be a marker of worse outcomes, the need for temporary RV mechanical support was uncommon in this cohort and seemed unrelated to baseline TPG.

We excluded patients with acute HF after myocardial infarction or post-cardiotomy failure. Selecting HF patients with chronic elevations in pulmonary pressures was a primary aim of this study. Chronic pulmonary venous congestion due to elevated LV filling pressures triggers a neurohormonal imbalance that enhances pulmonary vasoconstriction, endothelial dysfunction, and remodeling leading to PH.^{8,29}

Limitations

The retrospective nature of this study cannot account for all sources of selection bias. The initial decreases in PH might have been due to the concomitant use of inotropes. However, the magnitude of this effect was likely small given the persistent decrease in PH at long-term follow-up when most patients were not receiving inotropes. Our cohort had a considerable range of pulmonary pressures, yet many other factors might have contributed to the final decision on which patients received mechanical support. The survival analysis was also limited by small sample sizes.

Finally, we analyzed only pulsatile LVADs, so the findings may not be applicable to rotary blood pumps. A few reports using non-pulsatile LVADs indicate that they may also be effective in controlling PH, and this represents an active area of investigation.^{17–20} Although technology is moving toward non-pulsatile LVADs, it is apparent that the pulsatile devices do provide significant PH improvements.

Conclusions

There is marked improvement in pulmonary hemodynamics with pulsatile LVAD support across a wide range of pulmonary pressures, allowing carefully selected patients to undergo transplantation with similar outcomes, irrespective of the severity of pre-implant PH. LVAD support appears to be useful for reversing moderate to severe PH, thus improving HTx candidacy. This approach is not risk-free and should be reviewed prospectively before it is endorsed as an absolute clinical strategy. We believe our results can serve as a benchmark from which to compare PH improvements achieved with future LVAD systems and technologies.

Acknowledgments

The authors thank Don Severyn, MS, Steve Winowich, BSChE, and Rick Schaub, PhD for clinical bioengineering support; Kathleen Lockard, RN, Lori DeGore, RN, Stephanie Weaver, RN, and Eileen Stanford, RN, for clinical management; and Michelle D. Navoney, BS, and Julianne Buchanan, MS, for transplant data management.

Dr Simon is supported by the National Institutes of Health Roadmap Multidisciplinary Clinical Research Career Development Award (KL2 RR024154).

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Figure 1.

Pulmonary hemodynamics in 58 patients at baseline and after left ventricular assist device (LVAD) support. Early improvements in hemodynamics were sustained during late support. The black, white, and striped bars represent baseline (median 1 day pre-implant), early (median 1 day post-implant) and late (median 75 days post-implant) LVAD support, respectively. Values are mean $\pm$ standard deviation. MPAP, mean pulmonary artery pressure (mmHg); TPG, transpulmonary gradient (mmHg); PVR, pulmonary vascular resistance (Wood units); * $p < 0.001$ compared with baseline; † $p = 0.028$ compared with early LVAD support.

Figure 2.

Pulmonary hemodynamics in patients who survived to heart transplantation (n = 46) or myocardial recovery (n = 4) before, during, and after left ventricular assist device (LVAD) support. There were sustained improvements in hemodynamics after LVAD support. Grey, white, black, and striped bars represent baseline (median 2 days pre-implant), early (median 1 day post-implant), and late (median 75 days post-implant) LVAD support, and post-transplant/recovery (median 28 days post-explant), respectively. Values are mean $\pm$ standard deviation. MPAP, mean pulmonary artery pressure (mm Hg); TPG, transpulmonary gradient (mm Hg); PVR, pulmonary vascular resistance (Wood units); * $p < 0.005$ compared with baseline, $^{\dagger}p = 0.004$ compared with post-transplant/recovery.

Figure 3.

Transpulmonary gradient (TPG) by quartiles of baseline value in patients surviving to heart transplantation/recovery after left ventricular assist device (LVAD) support. TPG improved in patients with severe pulmonary hypertension. Quartile 4, TPG 14.1–26 mm Hg; quartile 3, TPG 11.3–14.0 mm Hg; quartile 2, TPG 7.8–11.2 mm Hg; quartile 1, TPG 2.0–7.7 mm Hg; * $P < 0.01$, quartile 4 follow-up values compared with baseline; † $p < 0.01$, quartile 3 follow-up values compared with baseline; ‡ $p < 0.01$, quartile 2 follow-up values compared with baseline.

Figure 4.

Transpulmonary gradients (TPG) during left ventricular assist device (LVAD) support among the 30 patients within the highest 2 quartiles of baseline values. Solid and dashed lines represent survivors and non-survivors to heart transplant/recovery, respectively. Highest quartiles are TPG 14.1–26 mm Hg (quartile 4) and TPG 11.3–14.0 mm Hg (quartile 3).

Figure 5. Pulmonary hemodynamics in 35 patients supported with a left ventricular assist device (LVAD) who survived to heart transplantation/recovery and received a pre-implant intra-aortic balloon pump. Improvements in transpulmonary gradient (TPG, mm Hg) and pulmonary vascular resistance (PVR, Wood units) were not sustained after transplant/recovery. Grey, white, black, and striped bars represent baseline (median 1 day pre-implant), early (1 day post-implant), and late (68 days post-implant) LVAD support, and post-transplant/recovery (27 days post-explant), respectively. Values are mean $\pm$ standard deviation. MPAP, mean pulmonary artery pressure (mm Hg); * $p < 0.005$ compared with baseline; † $p = 0.012$ compared with early LVAD support; ‡ $p = 0.011$ compared with late LVAD support.

Figure 6.

Pulmonary hemodynamics in 15 patients supported with a left ventricular assist device (LVAD) who survived to heart transplantation/recovery and did not receive a pre-implant intra-aortic balloon pump. Early hemodynamic improvements were sustained after LVAD support. Grey, white, black, and striped bars represent baseline (median 2 days pre-implant), early (median 2 days post-implant) and late (median 75 days post-implant) LVAD support, and post-transplant/recovery (median 28 days post-explant), respectively. Values are mean $\pm$ standard deviation. MPAP, mean pulmonary artery pressure (mm Hg); TPG, transpulmonary gradient (mm Hg); PVR, pulmonary vascular resistance (Woods units); * $p < 0.05$ compared with baseline.

Table 1

Baseline Characteristics and Survival Stratified by Degree of Baseline Pulmonary Hypertension

Variables ^a	Transpulmonary gradient, mm Hg					p-value
	All	2.0–7.7 (Quartile 1)	7.8–11.2 (Quartile 2)	11.3–14.0 (Quartile 3)	14.1–26.0 (Quartile 4)	
No.	58	14	14	16	14	
Age, years	49 ± 14	50 ± 15	52 ± 12	50 ± 17	46 ± 13	0.721
Male gender, No. (%)	46 (79)	13 (93)	9 (64)	15 (94)	9 (64)	0.059
Ischemic etiology, No. (%)	23 (40)	7 (50)	4 (29)	8 (50)	4 (29)	0.427
Diabetes mellitus, No. (%)	15 (26)	4 (29)	2 (14)	6 (38)	3 (21)	0.511
ICD, No. (%)	30 (52)	7 (50)	8 (57)	7 (44)	8 (57)	0.861
Previous sternotomy, No. (%)	11 (19)	4 (29)	0 (0)	5 (31)	2 (14)	0.117
LVEF	0.19 ± 0.05	0.20 ± 0.05	0.16 ± 0.05	0.20 ± 0.05	0.21 ± 0.05	0.095
Inotropes, No. (%)	58 (100)	14 (100)	14 (100)	16 (100)	14 (100)	
Milrinone	3 (5)	1 (7)	1 (7)	0 (0)	1 (7)	0.752
Dobutamine	25 (43)	8 (57)	6 (43)	7 (44)	4 (29)	0.506
Both inotropes	30 (52)	5 (36)	7 (50)	9 (56)	9 (64)	0.481
IABP support, No. (%)	42 (72)	9 (64)	11 (79)	13 (81)	9 (64)	0.611
Serum creatinine, mg/dl	1.32 ± 0.52	1.43 ± 0.70	1.26 ± 0.51	1.42 ± 0.41	1.20 ± 0.45	0.484
WBC count, ×10 ⁹ /liter	8.9 ± 3.6	8.7 ± 3.7	7.5 ± 3.2	11.0 ± 4.3 ^b	8.2 ± 2.2	0.046
Hematocrit, %	33.5 ± 4.8	34.3 ± 4.6	31.5 ± 4.5	34.0 ± 6.0	33.9 ± 3.4	0.400
Total bilirubin, mg/dl	1.31 ± 0.97	1.36 ± 0.90	0.93 ± 0.37	1.26 ± 1.23	1.69 ± 1.06	0.240
Serum albumin, mg/dl	3.3 ± 0.4	3.3 ± 0.5	3.0 ± 0.3	3.5 ± 0.4	3.3 ± 0.4	0.079
Severe MR, No. (%)	6 (10)	1 (7)	2 (14)	2 (13)	1 (7)	0.891
Hemodynamics						
RAP, mm Hg	10.4 ± 5.5	10.4 ± 5.8	9.4 ± 4.1	9.6 ± 6.5	12.2 ± 5.4	0.508
MPAP, mm Hg	34.6 ± 8.0	28.8 ± 5.8	31.1 ± 5.5	35.7 ± 7.7 ^c	42.7 ± 5.5 ^{c,d,e}	<0.05
TPG, mm Hg	11.7 ± 5.2	5.5 ± 1.6	9.6 ± 0.9 ^c	12.8 ± 0.8 ^{c,d}	18.7 ± 3.5 ^{c,d,e}	<0.05
PVR, Wood unit	2.81 ± 1.53	1.30 ± 0.62	2.32 ± 0.72 ^c	2.78 ± 0.89 ^c	4.82 ± 1.19 ^{c,d,e}	<0.05
Survival, No. (%)						
Heart transplant	46 (79)	10 (71)	12 (86)	13 (81)	11 (79)	0.820
Recovery	4 (7)	2 (14)	1 (7)	1 (6)	0 (0)	0.524
Died on LVAD	8 (14)	2 (14)	1 (7)	2 (13)	3 (21)	0.745
Post-HTx recovery, survival, No. %						
30 days	44 (88)	12 (100)	12 (92)	10 (71)	10 (91)	0.113
5 years	34 (68)	10 (83)	11 (85)	4 (29)	9 (82)	0.001

HTx, heart transplant; IABP, intra-aortic balloon pump; ICD, implantable cardioverter-defibrillator; LVAD, left ventricular assist device; LVEF, left ventricular ejection fraction; MPAP, mean pulmonary artery pressure; MR, mitral regurgitation; PVR, pulmonary vascular resistance; RAP, right atrial pressure; TPG, transpulmonary gradient; WBC, white blood cell.

^a Continuous values are presented as the mean ± standard deviation.

^b $p = 0.052$ compared with quartile 2.

^c
 $p < 0.05$ compared with quartile 1.

^d
 $p < 0.05$ compared with quartile 2.

^e
 $p < 0.05$ compared with quartile 3.

Table 2

Details of Patients Requiring an Unplanned Temporary Right Ventricular Assist Device at Time of Left Ventricular Assist Device Implantation

Variables	Patient 1	Patient 2	Patient 3	Patient 4
RV support duration, days	7	7	4	6
Age, years	59	60	22	24
Male gender	+	+	–	+
Ischemic etiology	–	+	–	–
Diabetes mellitus	–	–	–	–
ICD	–	+	–	–
Previous sternotomy	–	+	–	–
LVEF, %	18	15	10	18
Inotropes	+	+	+	+
IABP support	+	+	–	–
Serum creatinine, mg/dl	1.3	1.6	0.9	1.9
WBC count, $\times 10^9$ /liter	13.0	11.1	11.5	6.6
Hematocrit, %	35.6	26.1	31.6	27.4
Total bilirubin, mg/dl	1.8	1.2	1.5	1.7
Serum albumin, mg/dl	2.5	2.9	3.3	3.8
Severe mitral regurgitation	–	–	–	–
Hemodynamics				
RAP, mm Hg	11	11	15	18
MPAP, mm Hg	38	43	32	36
TPG, mm Hg	13	14	6	2
PVR, WU	4.3	4.2	2.2	0.3
Survival				
Heart transplant	+	+	–	+
Recovery	–	–	+	–
Died on LVAD	–	–	–	–
Post-HTx/recovery survival				
30 days	+	–	+	+
5 years	+	–	–	+
Cause of death		a	b	

+, yes; –, no; HTx, heart transplantation; IABP, intraaortic balloon pump; ICD, implantable cardioverter-defibrillator; LVAD, left ventricular assist device; LVEF, left ventricular ejection fraction; MPAP, mean pulmonary artery pressure; PVR, pulmonary vascular resistance; RAP, right atrial pressure; RV, right ventricle; TPG, transpulmonary gradient; WBC, white blood cell; a, acute donor LV failure immediately post-HTx with normal RV function; b, received HTx 1 year after recovery, but died 1 year later from acute rejection secondary to non-compliance with medications.