Biventricular Assist Device Utilization for Patients with Morbid Congestive Heart Failure
A Justifiable Strategy

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Background—The rationale for the use of a biventricular assist device (BiVAD) for morbid congestive heart failure (MCHF) has been questioned because of historically unacceptable rates of postimplant and post-transplant mortality as well as perceived barriers to their outpatient management.

Methods and Results—All patients who received a Thoratec BiVAD from January 1990 to December 2003 at the University of Pittsburgh were studied retrospectively. There were a total of 73 patients (32% ischemic, 21% idiopathic, and 47% other) who had a BiVAD implanted. Before implantation, 100% were on inotropic agent, and 77% had an intra-aortic balloon pump. Overall survival was 69%; 42 patients (84%) received cardiac transplantation, 5 patients (10%) were weaned, and 3 (6%) remained supported on BiVAD. If the 14 patients with postcardiotomy failure and acute myocardial infarction with shock are excluded, the overall survival improves to 75%. Five-year actuarial survival after heart transplantation was 58%. Of the 29 patients implanted before 2000, the 4-month actuarial freedom from driveline infections, bloodstream infections, and neurological events was 10%, 54%, and 48%, respectively, whereas the rates of these events for the 44 patients implanted after 2000 improved to 70%, 79%, and 80%, respectively. Since 2000, 21 (48%) patients were discharged from the hospital, of whom 38% went to an outpatient residence, 33% to a skilled nursing facility, and 29% to home. Once discharged, ≥1 readmission occurred in 45% and ≥2 readmissions in 48%.

Conclusions—BiVAD support for MCHF has an acceptable overall mortality and survival to transplantation. Morbidity has been significantly reduced in the past 4 years, and management as an outpatient is achievable. (Circulation. 2005; 112[suppl 1]:I-65–I-72.)

Key Words: biventricular assist device ▪ cardiomyopathy ▪ heart failure

Nearly 300 000 patients die from heart failure each year in the United States despite optimal medical therapy; and although ≈10 000 qualify as transplant candidates, only ≈2000 cardiac transplants are performed each year. As a consequence, left ventricular assist devices (LVADs) are now increasingly used to bridge patients to cardiac transplantation. However, in many patients, their cardiomyopathy can result in substantial right ventricular as well as left ventricular dysfunction. In our early experiences with the Novacor Left Ventricular Assist System (LVAS; World Heart Inc.), we were able to define a population of patients who did poorly with support from an LVAD alone. We found that this group of patients required an extended period of inotropic support for the right ventricle or had a high mortality from multiorgan failure when the strategy for using a right ventricular assist device (RVAD) was delayed until persistent shock occurred on an LVAD. Thus, in patients with severely decompensated heart failure with signs of significant right ventricular failure in whom an LVAD alone may not provide adequate circulatory support, we believed a strategy of planned biventricular assist device (BiVAD) implantation would be more effective. Unfortunately, in most published series, the mortality in patients requiring BiVAD as a bridge to transplantation has been reported to be >40% in contrast to the 25% mortality of patients bridged on an LVAD. This disparity in survival between BiVAD and LVAD, in addition to the perceived technical challenges to placement of a BiVAD, has led some to question the rationale for use of a BiVAD for morbid congestive heart failure (MCHF).

Most reports of using mechanical circulatory support as a bridge to transplantation contain very few patients who receive a BiVAD as an intention to treat, and those that do reflect a substantial number of patients who are at much higher risk, such as patients with postcardiotomy failure.
Although the consequences of prolonged inotropic support for a dysfunctional right ventricle in the setting of an LVAD are not well documented, such a strategy necessitates long-term inpatient management and has implications for rehabilitation and mobility as well as affecting the filling characteristics of the LVAD. In the past 10 years, we have used the Thoratec BiVAD (Thoratec Corp) strategically in patients with significant right ventricular dysfunction as well as in patients in whom we felt the risk for right ventricular failure would be high peroperatively on the basis of our early experience with using a single ventricle strategy. Our hypothesis was that through optimizing right ventricular flow and meticulous postoperative management protocols, we could achieve an acceptable morbidity and mortality in a morbidly ill patient population.

Methods

Data
Demographic and clinical outcome data, including adverse events and information regarding pump performance and device malfunction, were collected prospectively on all VAD recipients at the time of device implantation or on listing for transplantation. Patient data were prospectively collected into the Web-based Transplant Patient Management System (TPMS). This database was designed to function as a clinical database and a research registry and operates under protocols for these purposes that are approved by the University of Pittsburgh institutional review board for the use of patient management, quality assurance reports, and clinical research. It interfaces between multiple electronic databases within the University of Pittsburgh Medical Center and automatically ensures that all laboratory work, tests, and procedures related to the assist device are recorded in the database. In addition to all qualitative data on laboratory values including hematology, blood chemistry, and microbiology, qualitative reports, progress notes, device-related complications, and demographic data are entered directly by the ventricular assist device (VAD) coordinators, bioengineers, and physicians. The values reported are those that were drawn immediately before device implant and did not reflect the worst-case scenario so that every effort was made using maximal medical therapy to normalize the laboratory values before implantation of the device. Integrity of the database and quality assurance of the data are maintained by one of the investigators (J.R.B.) who performs the aggregate reports for this study as the honest broker in a deidentified format. Access to the TPMS database is password protected and conforms to Health Insurance Portability and Accountability Act (HIPPA) requirements.

Patients
The prospectively collected data from the TPMS was evaluated retrospectively in patients receiving biventricular support from January 1990 to December 2003. Patients were assigned on the basis of the date of BiVAD implantation into 2 cohorts: group A implanted from 1990 to 1999 (n=29), and group B implanted from 2000 to 2003 (n=44). Patients were included if they were heart transplant candidates and underwent Thoratec BiVAD support as the intention to treat as a bridge to heart transplantation. Patients were excluded from the analysis if the RVAD was placed >24 hours after implantation of the LVAD. Patients were also excluded from the study if they received a different type of RVAD from their LVAD because the anticoagulation protocols for each device were different. Therefore, this report dealt with only those patients for whom a preplanned BiVAD was the method for circulatory support.

Device
The Thoratec VAD system was used for biventricular support in all patients. This device consists of 3 components: (1) a blood pump, which has a 65-mL stroke volume and can deliver pulsatile flows of 1.3 to 7.1 L/min; (2) cannulae, which connect the blood pump to the heart; and (3) a drive console that powers the blood pump pneumatically. The VADs were placed in a paracorporeal position on the anterior abdominal wall. The LVAD cannulation was performed via left ventricular apex (inflow) with return to ascending aorta (outflow). We exclusively used left ventricular apical cannulation even in cases of a fragile left ventricle attributable to acute myocardial infarction. The RVAD was cannulated via the right atrium or, rarely, the right ventricle (inflow) and the pulmonary artery (outflow). Right atrial cannulation was performed in 90% (Figure 1). During implantation, transesophageal echocardiography was used to assess for a patent foramen ovale, which was closed if present. During the group A era of patients, the postoperative anticoagulation protocol consisted of immediate postoperative anticoagulation, with heparin and rapid conversion to Coumadin as soon as the patient was transferred out of the intensive care unit with the aim of removing intravenous lines as soon as possible. The goal was to achieve a target international normalized ratio (INR) of 2.5 as soon as possible. Postoperative anticoagulation in the group B era patients was started with 40% Dextran at 25 mL per hour for 6 hours after admission to the intensive care unit if bleeding was <100 mL per hour. Subsequently, heparin was started when postoperative bleeding from the chest tubes was <50 mL per hour over 3 consecutive hours. The goal for the partial thromboplastin time (PTT) was 40 to 51 seconds for at least the first 72 hours or until the risk of bleeding from more aggressive anticoagulation was felt to be acceptable. Heparin was then increased to maintain a PTT of 42 to 62 seconds. Coumadin was introduced at postoperative day 10 to keep the INR between 2.5 and 3.5. Heparin was discontinued after obtaining an INR of 2.5. The philosophy of anticoagulation in this latter group was to maintain heparin until the patient demonstrated a low risk for bleeding complications and after there had been a period of stable gastrointestinal tract function and diet. This usually was found to occur ~10 to 14 days after implant. A daily dose of 325 mg of aspirin was also started ~48 hours after implant. After discharge, the INR was assessed at a minimum of 2× per week for stable patients. To prevent infection from the exit site and kinking of the conduits, the VADs were restrained with an elastic dressing after implantation.

In the group A patients, driveline dressing changes occurred on a daily basis with the use of antibiotic or betadine rinses. This persisted...
even after discharge. Driveline dressings in the group B patients were performed with Hibiclens (chlorhexidine gluconate) once per week unless drainage was present. After discharge, home care nurses or family members were primarily responsible for the driveline dressing changes. We cultured the exit site at the first sign of unexpected or purulent-appearing drainage and actively involved the transplant infectious disease team for any bloodstream, pump pocket, or driveline infection. Therefore, we changed our dressing protocols to reduce the irritation and exposure of the driveline exit site by reducing the exposure of the wounds to once per week instead of daily.

Analysis

Data were analyzed using the commercially available statistics packages Statview (version 5.0; SAS Institute Inc.) and Statistica (StatSoft Inc). Survival is expressed in 30-day and actuarial format using Kaplan–Meier statistics, and significance is expressed as $P < 0.05$ with Mantel–Cox statistics or by using parametric or nonparametric comparisons where appropriate. Freedom from driveline and bloodstream infection and freedom from neurological events are expressed actuarially.

Results

There were 228 patients who received a VAD at University of Pittsburgh Medical Center during the study period. A total of 73 patients (32%) underwent placement of a biventricular support as the intention to treat for severe congestive heart failure. This number does not include 3 patients who received an intention-to-treat LVAD followed by an emergency RVAD 24 hours after their LVAD, all of whom died (2 on device before transplant and 1 within 24 hours after transplantation). In addition, it does not include 5 patients who received a hybrid BiVAD: 3 patients received a Novacor LVAS and Thoratec RVAD, and 2 patients received HeartMate LVAS (Thoratec Corp) and Thoratec RVAD. All Novacor-Thoratec hybrid patients died either on device before transplantation (2) or within 30 days after transplantation (1), and 1 HeartMate-Thoratec hybrid patient died on device before transplantation, whereas 1 survived after transplantation. All 73 patients who met criteria for this review received the Thoratec VAD system. Patients were then categorized according to the era in which they had their BiVAD implanted, from 1990 to 1999 (group A) or from 2000 to 2003 (group B). The latter time period was one in which standardized postimplant protocols for driveline and anticoagulation were established, the details of which are discussed below. The clinical characteristics and preoperative laboratory values of the entire cohort and each implantation era are shown in Table 1. There were 46 males (63%) and 27 females (37%), with ages ranging from 7 to 66 years (mean $43.5 \pm 16.1$). Mean body surface area was $1.89 \pm 0.27$ m$^2$ (range 1.11 to 2.33). Intravenous inotropic agents were used in all patients before BiVAD implantation. An intra-aortic balloon pump was placed before BiVAD implantation in 77% (mean duration 4.1 days; range 1 to 24 days). In 12% of the patients, the implantation of the BiVAD was preceded by extracorporeal membrane oxygenation or a temporary RVAD. Overall, moderate hepatic and renal dysfunction was present with a creatinine of $1.6 \pm 0.9$ mg/dL and a total bilirubin of $1.5 \pm 0.9$ mg/dL. Preoperative hemodynamic data collected from 54 patients are shown in Table 2. Mean right atrial pressure was $16 \pm 0.9$ mm Hg with a mean pulmonary artery pressure of $28 \pm 0.9$ mm Hg. The average preimplant cardiac index on full medical therapy was $2.0 \pm 0.9$ L/min per m$^2$. There were no statistical differences in

<table>
<thead>
<tr>
<th>TABLE 1. Demographics</th>
<th>Overall (n=73)</th>
<th>Group A (n=29)</th>
<th>Group B (n=44)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>43.5±16.1</td>
<td>46.2±11.1</td>
<td>42.2±8.2</td>
<td>NS</td>
</tr>
<tr>
<td>Gender (% male)</td>
<td>63.0</td>
<td>58.6</td>
<td>65.9</td>
<td>NS</td>
</tr>
<tr>
<td>BSA (m$^2$)</td>
<td>1.89±0.27</td>
<td>1.80±0.26</td>
<td>1.96±0.26</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>52.1</td>
<td>44.8</td>
<td>56.8</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes (%)</td>
<td>34.2</td>
<td>34.5</td>
<td>34.1</td>
<td>NS</td>
</tr>
<tr>
<td>Dialysis (%)</td>
<td>2.7</td>
<td>3.4</td>
<td>2.3</td>
<td>NS</td>
</tr>
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<td>Inotropic support (%)</td>
<td>100</td>
<td>100</td>
<td>100</td>
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</tr>
<tr>
<td>IABP support (%)</td>
<td>76.7</td>
<td>75.9</td>
<td>77.3</td>
<td>NS</td>
</tr>
<tr>
<td>Etiology of CHF</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic (%)</td>
<td>31.5</td>
<td>37.9</td>
<td>27.3</td>
<td>NS</td>
</tr>
<tr>
<td>Idiopathic (%)</td>
<td>19.2</td>
<td>13.8</td>
<td>22.7</td>
<td>NS</td>
</tr>
<tr>
<td>Inflammatory (%)</td>
<td>11.0</td>
<td>10.3</td>
<td>11.4</td>
<td>NS</td>
</tr>
<tr>
<td>Postcardiomyopathy (%)</td>
<td>11.0</td>
<td>10.3</td>
<td>11.4</td>
<td>NS</td>
</tr>
<tr>
<td>Acute myocardial infarction (%)</td>
<td>8.2</td>
<td>3.4</td>
<td>11.4</td>
<td>NS</td>
</tr>
<tr>
<td>Others (%)</td>
<td>19.1</td>
<td>24.3</td>
<td>15.8</td>
<td>NS</td>
</tr>
<tr>
<td>Preoperative laboratory values</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BUN (mg/dL)</td>
<td>33.7±22</td>
<td>35.7±19.8</td>
<td>32.4±24.6</td>
<td>NS</td>
</tr>
<tr>
<td>Creatinine (mg/dL)</td>
<td>1.6±0.8</td>
<td>1.7±0.7</td>
<td>1.5±0.9</td>
<td>NS</td>
</tr>
<tr>
<td>Total bilirubin (mg/dL)</td>
<td>1.5±0.9</td>
<td>1.5±1.0</td>
<td>1.5±0.9</td>
<td>NS</td>
</tr>
</tbody>
</table>

BSA indicates body surface area; CHF, congestive heart failure; IABP, intra-aortic balloon pump.
Severe right ventricular dysfunction—characterized CVP

Intractable ventricular arrhythmia or persistent ventricular fibrillation

Acute cardiogenic shock with multiorgan dysfunction with coagulopathy

These patients were, in effect, “destination” BiVAD patients.

fied that would limit successful survival after transplantation.

which major psychosocial or compliance issues were identi-

be performed safely or because of unusual circumstances in

a level of clinical stability that would allow transplantation to

patients died while supported because of a failure to achieve

tually listed for cardiac transplantation. Of those not listed, 5

in 3 (13%).

patients (13%), respiratory failure in 2 (8%), and other causes

mic, and cerebrovascular accident) in 5 (22%), sepsis in 3

10 (44%), neurological event (embolic, hemorrhagic, ische-

end of the study period, mortality after device placement was

and 3 patients (6%) remained supported with a BiVAD at the

recovery (mean support of 88 days; range 47 to 191 days),

received a cardiac transplant (after a mean support of 86 days;

Of the 73 patients, 50 (68%) patients, of whom 42 (84%)

ventricular failure, or perioperative bleeding.

there was an increased risk for multiorgan dysfunction, right

TABLE 3. Indications for BiVAD Implantation

Acute cardiogenic shock with multiorgan dysfunction with coagulopathy

Intractable ventricular arrhythmia or persistent ventricular fibrillation

Severe right ventricular dysfunction—characterized CVP >18 mm Hg or mean PAP <25 mm Hg or diastolic PAP <15 mm Hg on inotropic support and IABP

Giant cell myocarditis

Acute biventricular myocardial infarction with or without ventricular septal defect

Acute biventricular postcardiotomy failure

LVAD flow <2.0 L/min/m² and CVP >18mm Hg after LVAD implantation

CVP indicates central venous pressure; IABP, intra-aortic balloon pump; PAP, pulmonary artery pressure.

TABLE 2. Preoperative Hemodynamics

<table>
<thead>
<tr>
<th></th>
<th>Overall Data (n=54)</th>
<th>Group A (n=22)</th>
<th>Group B (n=32)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RAP (mm Hg)</td>
<td>16.3±7.0</td>
<td>17.6±7.6</td>
<td>15.4±6.7</td>
<td>NS</td>
</tr>
<tr>
<td>sRVP (mm Hg)</td>
<td>53.2±13.4</td>
<td>53.1±11.6</td>
<td>53.2±14.6</td>
<td>NS</td>
</tr>
<tr>
<td>dRVP (mm Hg)</td>
<td>17.8±6.9</td>
<td>17.0±6.7</td>
<td>18.4±7.2</td>
<td>NS</td>
</tr>
<tr>
<td>sPAP (mm Hg)</td>
<td>51.9±14.6</td>
<td>52.5±12.8</td>
<td>51.4±15.9</td>
<td>NS</td>
</tr>
<tr>
<td>dPAP (mm Hg)</td>
<td>27.3±8.2</td>
<td>27.3±7.9</td>
<td>27.4±8.6</td>
<td>NS</td>
</tr>
<tr>
<td>mPAP (mm Hg)</td>
<td>36.7±9.8</td>
<td>37.9±9.6</td>
<td>35.8±10.0</td>
<td>NS</td>
</tr>
<tr>
<td>PCWP (mm Hg)</td>
<td>27.5±7.9</td>
<td>28.7±7.7</td>
<td>26.6±8.0</td>
<td>NS</td>
</tr>
<tr>
<td>CI (L/min/m²)</td>
<td>1.94±0.61</td>
<td>2.01±0.72</td>
<td>1.89±0.52</td>
<td>NS</td>
</tr>
</tbody>
</table>

CI indicates cardiac index; PAP, pulmonary artery pressure; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure; RVP, right ventricular pressure.

Another 3 patients showed significant recovery of biventricular function from peripartum or inflammatory cardiomyopathy to the extent that they could eventually be successfully weaned from support and have the devices explanted. Once listed, 9 (14%) of the 65 patients were subsequently delisted, 7 because of the development of terminal comorbidities, and 2 additional patients were weaned after their initial listing.

Overall 30-day postimplant mortality was 15% (Figure 2A). We examined the mortality in a subset of 59 patients who did not have postcardiotomy failure or acute myocardial infarction with cardiogenic shock because these have been shown previously to be very high-risk populations.4–7 This was confirmed in our study, with our 14 postcardiotomy patients having a 30-day postimplant mortality of 21% and a transplant success rate of only 36%. With these patients excluded, the therapeutic success rate in group A was 72%, and in group B, the success rate improved to 76%.

Mortality 30 days after transplant for those surviving to transplantation was only 4% in the entire cohort, but there was a substantial decrease in the 30-day post-transplant mortality from 6.9% in group A to 2.3% in group B patients (Figure 2B).

Actuarial survival rate after VAD implantation was 53% at 12 months (Figure 3A). Five-year actuarial survival rates after heart transplantation calculated with Kaplan–Meier method was 58% (Figure 3B).

Complications

The need for re-exploration because of bleeding occurred in 25 patients (34%): 17 required only 1 re-exploration, 6 required 2 re-explorations, and 2 required 3 re-explorations. The 4-month

Figure 2. Thirty-day mortality after BiVAD implantation (A) and 30-day mortality after heart transplantation (B). AMI indicates acute myocardial infarction; PC, postcardiotomy. In group A, patients implanted from 1990 to 1999 (n=29); in group B, patients implanted from 2000 to 2003 (n=44).
actuarial freedom from driveline infection was 10% in the 29 group A patients, whereas it was 70% in the 44 group B patients (Figure 4). The 4-month actuarial freedom from bloodstream infection in group A was 54% and improved to 79% in group B (Figure 4). The 4-month actuarial freedom neurological events improved from 48% in group A to 80% in group B (Figure 4). Although there were no major device malfunctions, 3 patients required elective change-out of the RVAD pump: 2 because of thrombus formation (postoperative days 48 and 321) and 1 patient because of a cracked housing after extended long-term use (postoperative day 584).

**Patients Discharged From Hospital**

Twenty-one patients (48%) of group B were successfully discharged from the hospital: 8 patients (38%) to an outpatient residence, 7 (33%) to a skilled nursing facility, and 6 (29%) to home, including 1 patient discharged home via a skilled nursing facility. The major barriers to discharging patients directly to home were: a period during which the portable controller of Thoratec VAD system was unavailable, thus requiring discharge to an outpatient skilled nursing facility on console; the requirement for inpatient rehabilitation; and inadequate family support, especially with regard to the inability to provide adequate wound care. Readmission occurred in 20 patients (61%), with approximately half requiring ≥1 admission and half ≥2 admissions. Reasons for readmission included infection in 20%, bleeding in 10%, neurological events in 6%, device malfunction in 6%, arrhythmia in 6%, hepatic dysfunction in 6%, pulmonary dysfunction in 6%, renal dysfunction in 3%, and other causes in the remaining 37%.

**Discussion**

The rationale for use of a BiVAD for MCHF is still controversial. Patients requiring BiVAD are generally more severely ill in the preoperative period, and the survival of patients through heart transplantation is significantly better in patients who received a LVAD (74%) than in those who had a BiVAD (58%). With twice as many cannulae and pumps, the higher incidences of thrombotic, infectious, cannulation, and mechanical problems with a BiVAD versus an LVAD

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**Figure 3.** Actuarial survival rate after BiVAD implantation (A) and actuarial survival rate after heart transplantation (B).

**Figure 4.** Comparison of postoperative complications (driveline infection, neurological events, and bloodstream infection) between groups A and B. In group A, patients implanted from 1990 to 1999 (n=29); in group B, patients implanted from 2000 to 2003 (n=44).
alone are not unexpected. However, Pennington et al showed no difference in mortality or morbidity in between BiVAD in 16 patients and LVAD in 23 patients.9 Farrar et al reported there was no significant difference in post-transplantation survival through hospital discharge (BiVAD 81%; LVAD 89%). However, the survival to transplantation of the BiVAD patients was only 58% compared with 74% for LVAD patients. In a more current report from 2003, in a smaller series of patients with profound cardiogenic shock supported with a BiVAD, 59% of patients were bridged successfully to transplantation, with a 90% post-transplant survival rate.10 Our data showed similarly acceptable results, with an overall survival rate of 68%. Furthermore, when the high-risk postcardiotomy patients are excluded, the overall survival rate in the more recent group B era increased to 76%. Our strategy of implanting BiVADs proactively rather than as a bail-out in the setting of LVAD-associated cardiogenic shock demonstrated a reasonable survival to transplantation and a very acceptable survival rate after transplantation.

Patient selection for BiVAD support is crucial to obtaining successful outcomes, but the criteria for predicting the need for a BiVAD are not well established. A previous report from our institution has shown that clinical factors that reflect preimplantation degree of illness and perioperative factors (lower mixed venous oxygen saturation, a greater level of inotropic need, impaired mental status, and a lower ratio of right ventricular fraction to inotropic need) that result in impairment of pulmonary blood flow or reduced perfusion of right ventricle after LVAD implantation were considered to be more predictive of the need for additional right ventricular assist than preimplantation measures of right ventricular function or hemodynamic variables.11 Farrar demonstrated patients requiring BiVAD were more severely ill, as demonstrated by a higher serum creatinine, a greater proportion of ventilator dependence before VAD, and emergent implantation.8 Other authors concluded the need for circulatory support, female gender, nonischemic etiology,12 low pulmonary artery pressure, and low right ventricular stroke work index13 were significant predictors for RVAD use after LVAD insertion. We have chosen to place a BiVAD in patients with right ventricular dysfunction as evidenced by low pulmonary pressures (<25 mm Hg mean or a diastolic pressure <15 mm Hg) and high central venous pressure (>18 mm Hg). In addition, we placed a BiVAD in patients with severe biventricular dysfunction in the setting of myocardial infarction, in the presence of severe pulmonary edema, or in whom there is a severe coagulopathy with risk for substantial perioperative bleeding, particularly in the setting of multiorgan failure. These factors have been shown to lead to post-LVAD right ventricular failure even when the preimplant hemodynamics appear to be satisfactory for LVAD alone support.

Data from Farrar et al and our own work has revealed that the septal shift, which accompanies the implantation of an LVAD, results in mild to moderate right ventricular dysfunction in most patients receiving an LVAD. However, the profound right ventricular afterload reduction, which accompanies the reduction of an elevated left atrial pressure in patients with chronic congestive heart failure, is ultimately more beneficial to the function of the right ventricle than the negative impact of septal shift. Therefore, in most cases, even with echocardiographically documented severe right ventricular dysfunction, an RVAD is not required. On the other hand, if complications occur that raise the parenchymal or vascular pulmonary pressure, such as with pre-existing pulmonary edema or microvascular obstruction that accompanies severe bleeding and subsequent massive blood product transfusion, then there is little afterload reduction for the right ventricle, the negative effects of septal shift will predominate, and right ventricular failure becomes pronounced. Therefore, it appears that the most important factor in avoiding right ventricular failure after LVAD implantation is protection of the lungs. It was our prejudice to implant a BiVAD in those instances in which we expected pulmonary insults including bleeding requiring massive blood product transfusion and ongoing severe preimplant pulmonary edema. This was in addition to the indications outlined previously that included factors affecting right ventricular coronary perfusion, severe preoperative right ventricular dysfunction, or severe intractable ventricular arrhythmias. It is our belief that the morbidity of delayed placement of an RVAD or attempting to support a patient on an LVAD with high doses of inotropic support leads ultimately to multiorgan failure. For these reasons, when presented with a high-risk patient population as outlined in Table 2, it is our preference to implant a BiVAD strategically as the first device.

The 1- and 5-year survival rates after heart transplantation in patients after BiVAD implantation were 76% and 58%, respectively. This compares favorably to data from the registry of the International Society for Heart and Lung Transplantation (ISHLT) 21st Official Adult Heart Transplant Report—2004, in which 1- and 5-year survival rates were ~80% and 65%, respectively.2 Although our 5-year survival rate was inferior to that of ISHLT, our 15 patients who died after heart transplantation had more risk factors than the patients in the ISHLT report (Table 4).

The most common complications after VAD implantation are bleeding, infection, and thromboembolic events. With the increase of the mean support duration because of the shortage of donor organs, patients are exposed to an increasing probability of infectious and thromboembolic complications during VAD support. Bleeding is the most common perioperative complication and is especially important in those with pre-existing hepatic dysfunction secondary to cardiogenic shock combined with preimplant anticoagulation with Coumadin. The reported prevalence of bleeding in patients with Thoratec VAD is 31% to 60%.814 We developed strategies to decrease intraoperative bleeding by using aprotinin or Amicar before surgery. We also used meticulous preclotting of the outflow grafts with blood- and calcium-activated thrombin spray and apply calcium product bio-glu to the apex cannulation site. The safety provided by the device allows us to withhold anticoagulation for ≥8 to 12 hours after device implantation, thereby reducing the risk of perioperative bleeding.

Driveline infection is still a continuing problem for VAD patients, especially with extended periods of use. Ankersmit et al pointed out that VAD implantation causes an aberrant state of T-cell activation, heightened susceptibility of CD4 T cells to activation-induced cell death, progressive defects in cellular immunity, and increased risk of opportunistic infection.15 In the REMATCH trial, the probability of infection of the LVAD was 28% within 3 months after implantation.16 Although these infections occur primarily in the VAD driveline and pocket, it is nevertheless common to develop to fatal sepsis. Because driv-
elined infection rates are high even in an intracorporeal LVAD with only the 1 driveline passing through the skin, it would be expected that the Thoratec BiVAD would have a higher infectious risk because it has 4 percutaneous cannulae. McBride et al demonstrated driveline infection prevalence was 15% in bridge to transplant patients with a mean support duration of 40.7 days. To address the issue of driveline infections, we changed to a strategy of decreasing the frequency of wound exposure and thus have decreased the frequency of dressing changes from daily to weekly as long as there is no significant drainage present. Wound care was done with betadine (povidone–iodine 10%) solution initially but switched to Technicare because of an increase in the rate of infection. However, after we switched to Technicare, we experienced an outbreak of pseudomonas infection, and now diluted Hibiscens is the preferred agent during dressing changes. We also asked the patients to not shower as frequently. With the institution of these changes to the dressing protocol, we improved the 4-month actuarial freedom from driveline infection rate from 10% in group A to 70% in group B.

The rate of thromboembolic events in bridge to recovery and bridge to transplantation patients is 9% and 19%, respectively. The 4-month actuarial freedom from neurological complications in our series improved from 48% in group A to 80% in group B. This result parallels the reduction in infection rates and also reflects careful patient and family training on how to manage drivelines. We recently introduced use of the thromboelastogram (TEG) to monitor platelet function. We anticipate the precise platelet function control based on TEG will further prevent long-term thromboembolic complications.

From the beginning of introduction of the Thoratec VAD, we initiated a program of discharge from hospital with VAD support. The benefits of discharging patients to home couples quality of life improvements and economic advantages for patients on VAD. DeRose et al were able to discharge 19 of 32 patients (59%) on LVAD from the hospital on mean postoperative day 41 ± 4 (range 17 to 68) for an outpatient support time of 108 ± 30 days (range 2 to 466). In group B, 21 of 44 patients (48%) were discharged after the patients and families received education for proper VAD maintenance. Use of the newer implantable configurations of system (Thoratec implantable VAD [IVAD]) will likely further increase the proportion of patients who can be discharged directly to home because of the simplicity of its wound care compared with the BiVAD. However, the readmission rate is still high (61%). Our outpatient patient support team includes a group of clinical bioengineers (Vital Engineering), experienced nurses who respond to patient and family concerns and visit patients in the surrounding regional extended care, skilled nursing facilities trained to care for patients on VADs, and an extensive patient and family teaching program that ensures confidence in managing these patients at home. For this field to expand, it is mandatory to decrease readmission rate for patients on VADs in the future. Hopefully, use of the IVAD will remove the cumbersome paracorporeal arrangement and further enhance morbidity-free survival of a BiVAD.

### Limitations

This study is a single-center retrospective study of prospectively gathered information. Not all data from every patient were available or could not be obtained because of the critical illness of the patients studied. The BiVAD was placed at the discretion of the surgeon after consulting with the transplant cardiologists. There were not definite criteria used to determine which patients received BiVAD versus LVAD; rather, the decision was made on the preponderance of the clinical data.

### TABLE 4. Risk Factors for 5-Year Mortality

<table>
<thead>
<tr>
<th>Variable factors</th>
<th>Data From This Series (n=15, %)</th>
<th>ISHLT Data (%)</th>
<th>ISHLT (odds ratio)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Repeat transplant</td>
<td>6.7</td>
<td>2.0</td>
<td>1.93</td>
</tr>
<tr>
<td>Dialysis</td>
<td>6.7</td>
<td>No data</td>
<td>1.80</td>
</tr>
<tr>
<td>Intra-aortic balloon pump</td>
<td>73.3</td>
<td>5.9</td>
<td>1.66</td>
</tr>
<tr>
<td>Ventilator</td>
<td>13.3</td>
<td>3.0</td>
<td>1.62</td>
</tr>
<tr>
<td>Diagnosis congenital heart disease</td>
<td>6.7</td>
<td>2.7</td>
<td>1.51</td>
</tr>
<tr>
<td>Infection requiring intravenous drug therapy within 2 weeks of transplant</td>
<td>13.3</td>
<td>No data</td>
<td>1.37</td>
</tr>
<tr>
<td>Diagnosis coronary artery disease</td>
<td>53.3</td>
<td>44.7</td>
<td>1.29</td>
</tr>
<tr>
<td>Diagnosis other (excluding cardiomyopathy)</td>
<td>13.3</td>
<td>1.9</td>
<td>1.19</td>
</tr>
<tr>
<td>Diabetes</td>
<td>40.0</td>
<td>19.3</td>
<td>1.19</td>
</tr>
<tr>
<td>Intravenous inotropes</td>
<td>100</td>
<td>47.2</td>
<td>0.87</td>
</tr>
<tr>
<td>Sternotomy</td>
<td>100</td>
<td>No data</td>
<td>0.85</td>
</tr>
<tr>
<td>Continuous factors</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recipient age (years)</td>
<td>51.0</td>
<td>41.5</td>
<td></td>
</tr>
<tr>
<td>Ischemia time (minutes)</td>
<td>215±80</td>
<td>186±60</td>
<td></td>
</tr>
</tbody>
</table>

**Notes:**
- **ISHLT Data** refers to the International Society for Heart and Lung Transplantation data.
- **ISHLT (odds ratio)** indicates the odds ratio calculated from ISHLT data.
This was not meant to be a study of the differences in characteristics between patients who had LVAD and BiVAD but rather an assessment of the outcomes of our particular strategy of placing a BiVAD and our subsequent improvements in the care of these patients. Our efforts at outpatient management benefit from a robust support system of physicians, nurses, engineers, and other support personnel experienced with the care of patients on ventricular support. It also acknowledged that there is the potential that many of our reasons for placing patients on biventricular support are related to perioperative right ventricular dysfunction, and that if a low-cost removable alternative for temporary right heart support existed, we may have been able to place a traditional implantable LVAD. The temporary RVAD could then be removed at a later date. We did not systematically try to wean patients from the RVAD once the BiVAD had been implanted.

Conclusion

Support with BiVAD offers an acceptable rate of survival to cardiac transplantation. Furthermore, the use of a BiVAD itself does not confer an increased morbidity or mortality, and overall outcomes with this device are comparable to that of implantable LVADs if used strategically in severe congestive heart failure. With the institution of meticulous wound care, morbidity has been significantly reduced, and management as an outpatient is achievable; however, readmissions are still frequent. The precise factors that determine the need for biventricular versus univentricular support are still ill defined but remain a major focus for future research.

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Biventricular Assist Device Utilization for Patients with Morbid Congestive Heart Failure: A Justifiable Strategy
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