Original Article

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Right Ventricular Support after Implantation of a NOVACOR Left Ventricular Assist Device

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Abstract

The objective was to determine which factors might help to predict the need for mechanical right ventricular support following insertion of a left ventricular assist device (LVAD). A retrospective analysis was performed on 24 patients with cardiomyopathies who had insertion of a LVAD as a bridge to heart transplantation at Presbyterian University Hospital during the period 1987 to 1991. Group 1 consisted of 18 patients who could be adequately supported with the LVAD alone, while Group 2 consisted of 6 patients who required additional support with a right ventricular assist device. Group 2 exhibited longer periods of hypotension on cardiopulmonary bypass, increased inotropic support and decreased right ventricular ejection fraction at time of chest closure post-LVAD.

Introduction

Heart transplantation is the only option for select patients with end-stage heart failure but the supply of donor hearts is limited and as many as 30% of potential recipients die before a heart becomes available (1). Staged cardiac transplantation is the use of a ventricular assist device (VAD) or total artificial heart (TAH) as a "bridge to heart transplantation" until a donor heart becomes available. The results with the TAH in staged cardiac transplantation have been poor, and complications have included bleeding, renal failure, infection, and multi-organ failure with ultimate hospital discharge of less than 50% (2). Results are better with the use of a VAD, particularly with univentricular support (2).

At Presbyterian University Hospital, a trial of univentricular support with the Novacor type Left Ventricular Assist Device (LVAD) in staged heart transplantation was started in 1987 (3). It was the intent to support all patients with the LVAD regardless of pre-existing right ventricular dysfunction and while this was accomplished in most patients, several required mechanical support for refractory right ventricular failure post-LVAD. While it was not possible to identify these patients pre-LVAD, certain events occurred while on CPB, and soon after LVAD implantation, which would appear to predict the need for a right ventricular assist device (RVAD).

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Table 1: Patient Demographic Data

	Group 1	Group 2	p Value
Age (yr)	45 ± 10	34 ± 16	p < 0.05
Weight (kg)	69 ± 13	79 ± 19	NS
BSA (m^2)	1.81 ± 0.20	1.97 ± 0.29	NS
Days on LVAD	64 ± 75	48 ± 58	NS

Table 1
Patient Demographic Data
Data expressed as mean ± SD
BSA = body surface area
NS = not significant

Table 2: Hemodynamics at Chest Closure

	Group 1	Group 2	p Value
MAP	81 ± 10	75 ± 8	NS
MPAP	25 ± 7	28 ± 10	NS
RAP	15 ± 4	16 ± 8	NS
CI	2.96 ± 0.57	2.70 ± 0.50	NS
PVR	239 ± 139	254 ± 112	NS
SVR	987 ± 292	939 ± 327	NS
RVEF (%)	23 ± 8	14 ± 5	p < 0.05

Table 2
Hemodynamics at Chest Closure
MAP = mean arterial pressure
MPAP = mean pulmonary arterial pressure
RAP = right atrial pressure
CI = cardiac index
PVR = pulmonary vascular resistance
SVR = systemic vascular resistance
RVEF = right ventricular ejection fraction
Data expressed as mean ± SD
NS = not significant

Materials and Methods

The Novacor LVAD[a] is an electrically powered left ventricular assist device intended for long-term circulatory support for patients with end-stage heart disease. Its present design is for staged cardiac transplantation but the goal is to increase its applicability to a totally implantable system by late 1992 (4).

Protocols for collection of hemodynamic data were approved by the Institutional Review Board of the University of Pittsburgh Medical Center. Twenty-seven patients with cardiomyopathy had a Novacor LVAD placed as a bridge to heart transplantation at Presbyterian University Hospital between July 1987 and December 1991. Three patients had perioperative complications leading to death and were excluded from analysis. Group 1 consists of 18 patients adequately supported with the LVAD alone. Group 2 consists of 6 patients who developed right ventricular failure, typically soon after chest closure but within eight hours post-LVAD, and required a RVAD. A BioMedicus centrifugal pump[b] was used for the RVAD resulting in "hybrid" biventricular assist support, i.e., the electrical Novacor LVAD and a centrifugal RVAD. Right ventricular failure was defined as right atrial pressure (RAP) >20 mmHg and cardiac index (CI) <2.5 L/min/m^2.

Table 3: Cardiopulmonary Bypass Variables

	Group 1	Group 2	p Value
CPB Time (min)	70 ± 15	76 ± 9	NS
Mean Flow Index (l/min/m^2)	2.6 ± 0.2	2.7 ± 0.2	NS
SVR			
Initial	908 ± 253	1105 ± 621	NS
30 mins	1046 ± 273	884 ± 224	NS
60 mins	951 ± 213	794 ± 254	NS
Hematocrit (%)	21 ± 2	21 ± 3	NS
TM 50 (min)	12 ± 20	33 ± 29	p = 0.06
CPBTM50 (%)	16 ± 23	42 ± 36	p = 0.05

Table 3
Cardiopulmonary Bypass Variables
CPB Time = duration of CPB
Mean flow index = average flow on CPB
SVR = systemic vascular resistance
TM50 = minutes pressure < 50 mm Hg
CPBTM50 = minutes pressure < 50 mm Hg/duration of CPB
Data expressed as mean ± SD

Table 4: Inotropic Support at Chest Closure

	Group 1	Group 2	p Value
Dobutamine	20 ± 4	20 ± 2	NS
Epinephrine	0.02 ± 0.06	0.17 ± 0.16	p = 0.004
Norepinephrine	0.04 ± 0.10	0.28 ± 0.28	p = 0.004

Table 4
Inotropic Support at Chest Closure
Data expressed in μg/kg/min, as mean ± SD
NS = not significant

a Baxter Healthcare Corporation, Oakland, CA
b BioMedicus, Medtronic BioMedicus, Inc., Eden Prairie, MN

Data Collection

All patients undergoing LVAD insertion had hemodynamic profiles collected during the perioperative period which included systemic arterial pressure, right atrial pressure, and pulmonary arterial pressure (PAP; pressures are in mmHg). Cardiac index (L/min/m^2) and right ventricular ejection fraction (RVEF) were obtained with a right ventricular ejection fraction catheter[a] and systemic vascular resistance (SVR) and pulmonary vascular resistances (PVR) were calculated (dynes-sec/cm^5). Levels of inotropic, vasoconstrictor, and vasodilator support during the procedure were recorded.

The CPB records were reviewed for duration of CPB, flows on CPB, lowest hematocrit on CPB and systemic vascular resistance at onset, and at 30 and 60 minutes on CPB. Stockard (5) developed tm50, which is defined as the torr-minutes of perfusion below 50 torr, and is represented by the area between the 50 torr line on the pressure record and the MAP tracing when it is below 50 torr. It has been used as a scoring system for the relation between hypotension on CPB and subsequent cerebral dysfunction. Since this was a retrospective analysis of handwritten anesthesia records, the index was modified to measure only the total number of minutes that the pressure was less than 50 mm Hg on CPB and was termed TM50. To normalize the degree of hypotension to the length of CPB, CPBTM50, defined as TM50 divided by CPB duration, was calculated.

Statistical Analysis

Comparison of variables between groups was by analysis of variance with independent t-testing. A p value less than 0.05 was considered statistically significant. Data is presented as mean ± standard deviation.

Results

Patient demographic data are presented in Table 1. The significant difference in age between groups is unclear to us.

Hemodynamic data at chest closure is shown in Table 2. The only difference between the groups was in RVEF. Group 2 had a markedly reduced RVEF with a RAP and MPAP similar to Group 1.

CPB variables are shown in Table 3. Groups 1 and 2 did not differ in duration of CPB, CPB flow, CPB hematocrit, or SVR. The aim was to maintain CPB mean arterial pressure (MAP) above 50 mm Hg with bypass flows of 1.8 to 2.5 l/min/m^2 and a hematocrit between 18 and 22%. When a MAP of 50 mm Hg could not be maintained with these flows and hematocrit, vasoconstrictor agents, either phenylephrine or norepinephrine, were infused. While there was a non-significant difference (p=0.06) in TM50 between Group 1 and Group 2, there was a significant difference in CPBTM50, with Group 2 having 42% of CPB with a pressure below 50 mmHg.

Inotropic support at chest closure is shown in Table 4. Dobutamine was the primary inotropic agent of support for the right ventricle post-LVAD. When an infusion of 20 µg/kg/min of dobutamine was reached, additional right ventricular inotropy was obtained with epinephrine and/or norepinephrine. Norepinephrine was often added to maintain vascular resistance and perfusion pressure when cardiac output was adequate. At chest closure there were significant differences in the amount of inotropic support between the groups. Group 2 required several times the amounts of epinephrine and norepinephrine than Group 1 to maintain a similar cardiac index and systemic vascular resistance (see Table 2).

The patients in Group 2 had the RVAD in place for an average of 4 days. Of the 18 patients in Group 1, 15 patients (83%) had successful heart transplants and were discharged from the hospital while 3 patients suffered multi-organ failure or neurologic events which precluded heart transplantation. Of the 6 patients in Group 2, 3 patients (50%) had heart transplants and left the hospital while the remaining 3 patients died of multi-organ failure.

Discussion

The question of univentricular versus biventricular assist device support depends upon patient selection (6). The reported experience with RVAD support following LVAD insertion ranges from 0% (7) to 25% in the present study. The discrepancies are due to the selection of patients at different times in the progression of their cardiac decompensation.

Causes of right ventricular dysfunction after LVAD insertion include right ventricle (RV) conformational changes with the LV decompressed by the LVAD, insult to the RV during implantation, persistent pulmonary arterial hypertension, and inability to maintain a perfusion pressure to the RV.

It was not possible to predict which of our patients would require a RVAD before the LVAD was placed. Early indicators came with the relative inability to maintain a perfusion pressure on CPB. The causes of this hypotension were not examined in this study but are likely humoral and may represent endotoxemia due to poor perfusion to the liver and gastrointestinal tract. The persistent vasodilation after CPB necessitates the use of vasoconstrictor support such as norepinephrine to maintain a perfusion pressure for the right ventricle post-LVAD.

We support the previous conclusions (2), that the use of univentricular support results in a better outcome then the use of "hybrid" biventricular support.

In summary, by retrospective analysis, the triad of hypotension on CPB, epinephrine and norepinephrine requirement and decreased RVEF at time of chest closure, would appear to predict the need for RVAD after LVAD.

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