

# Continuous-Flow Left Ventricular Assist Device Support in Patients with Ischemic Versus Nonischemic Cardiomyopathy

Brendan P. Chou, BS<sup>1</sup>; Andre Critsinelis, MD<sup>1</sup>; Harveen K. Lamba, MD<sup>1</sup>; Gregory Long, BS<sup>1</sup>; Andrew B. Civitello, MD<sup>1,2</sup>; Reynolds M. Delgado, MD<sup>1,2</sup>; Subhasis Chatterjee, MD<sup>3</sup>

<sup>1</sup>Division of Cardiothoracic Transplantation and Circulatory Support, Baylor College of Medicine, Houston, Texas

<sup>2</sup>Department of Cardiopulmonary Transplantation and Center for Cardiac Support, Texas Heart Institute, Houston, Texas

<sup>3</sup>Division of Cardiothoracic Surgery, Baylor College of Medicine, Houston, Texas

*To determine whether the cause of cardiomyopathy affects outcomes in patients who undergo continuous-flow left ventricular assist device support, we compared postimplant adverse events and survival between patients with ischemic and nonischemic cardiomyopathy. The inclusion criteria for the ischemic group were a history of myocardial infarction or revascularization (coronary artery bypass grafting or percutaneous coronary intervention),  $\geq 75\%$  stenosis of the left main or proximal left anterior descending coronary artery, or  $\geq 75\%$  stenosis of  $\geq 2$  epicardial vessels.*

*From November 2003 through March 2016, 526 patients underwent device support: 256 (48.7%) in the ischemic group and 270 (51.3%) in the nonischemic group. The ischemic group was older (60.0 vs 50.0 yr), included more men than women (84.0% vs 72.6%), and had more comorbidities. More patients in the nonischemic group were able to have their devices explanted after left ventricular recovery (5.9% vs 2.0%;  $P=0.02$ ). More patients in the ischemic group had gastrointestinal bleeding (31.2% vs 22.6%;  $P=0.03$ ), particularly from arteriovenous malformations (20.7% vs 11.9%;  $P=0.006$ ) and ulcers (16.4% vs 9.3%;  $P=0.01$ ). Kaplan-Meier analysis revealed no difference in overall survival between groups ( $P=0.24$ ). Older age, previous sternotomy, higher total bilirubin level, and concomitant procedures during device implantation independently predicted death ( $P \leq 0.03$ ), whereas cause of heart failure did not ( $P=0.08$ ).*

*Despite the similarity in overall survival between groups, ischemic cardiomyopathy was associated with more frequent gastrointestinal bleeding. This information may help guide the care of patients with ischemic cardiomyopathy who receive continuous-flow left ventricular assist device support. (Tex Heart Inst J 2021;48(4):e207241)*

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Cardiomyopathy; heart-assist devices/adverse effects; heart failure/etiology; treatment outcome; survival

**Corresponding author:**

Harveen K. Lamba, MD, Department of Cardiopulmonary Transplantation and Center for Cardiac Support, Texas Heart Institute, 6770 Bertner Ave., DAC C355N, Houston, TX 77030

**E-mail:**

harveen.lamba@bcm.edu

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**C**ontinuous-flow left ventricular assist devices (LVADs) are used as bridge-to-transplant, bridge-to-recovery, or destination therapy in patients with end-stage heart failure. Since 2006, more than 17,000 continuous-flow LVADs have been implanted, according to the Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) database, and overall outcomes have improved.<sup>1</sup>

The causes of advanced heart failure vary greatly,<sup>2</sup> and how continuous-flow LVAD support affects outcomes has not been thoroughly explored. Some studies have shown that survival times are shorter in patients with ischemic cardiomyopathy (ICM) than in those with nonischemic cardiomyopathy (NICM); patients with ICM tend to be older and have more comorbidities, widespread atherosclerotic disease, multivessel coronary artery disease, and arrhythmias associated with sudden death.<sup>3-8</sup> Other studies have produced conflicting results regarding whether the cause of heart failure is an independent predictor of death after continuous-flow LVAD implantation.<sup>9-11</sup> Few investigators have explored the differences between the types and the incidence of postimplant adverse events based on the distinctly different causes of heart failure.

Therefore, the effect of heart failure cause on outcomes in patients who receive continuous-flow LVADs as bridge-to-transplant or destination therapy warrants

further investigation. In this study, we compared the overall survival and incidence of adverse events after continuous-flow LVAD implantation in patients with ICM versus NICM.

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## Patients and Methods

We performed a single-center retrospective review of all patients who underwent primary implantation of a continuous-flow LVAD—either the HeartMate II (Thoratec, an Abbott company) or the HeartWare HVAD (Medtronic)—from November 2003 through March 2016. Patients were divided into 2 groups on the basis of a standardized definition of ICM developed for clinical research.<sup>12</sup> The criteria for inclusion in the ICM group were a history of myocardial infarction, coronary revascularization (coronary artery bypass grafting or percutaneous coronary intervention),  $\geq 75\%$  stenosis of the left main or proximal left anterior descending coronary artery, or  $\geq 75\%$  stenosis of  $\geq 2$  epicardial vessels.<sup>12</sup> The NICM group included all other patients. The incidence of ICM in the study population was calculated for the periods from 2003 through 2009 and from 2010 through 2016. The Institutional Review Board of CHI St. Luke's Health—Baylor St. Luke's Medical Center approved the study (Protocol H38751). The requirement for informed consent was waived because the study was retrospective.

From each patient's medical record, we documented demographic information, preoperative characteristics including laboratory INTERMACS profile<sup>13</sup> and comorbidities, hemodynamic and echocardiographic data, operative details, postoperative adverse events, and survival outcomes. Severity of valve regurgitation was assessed by color-flow imaging in all cases and quantitatively graded on a 4-point scale (severe = 3, moderate = 2, mild = 1, and none = 0),<sup>14</sup> as documented in the medical record. Operative characteristics included device model (HeartMate II or HeartWare HVAD), cardiopulmonary bypass use and time, aortic cross-clamp use and time, and concomitant procedures performed during the index LVAD implantation. Concomitant procedures included coronary artery bypass grafting, patent foramen ovale closure, and valvular repair or replacement.

Outcome variables included the incidence of postoperative adverse events (including events per patient-year) and overall survival rates through 6 years of follow-up after LVAD implantation. Readmission was defined as a return to the hospital within 30 days of discharge from the index admission. Neurologic dysfunction was defined as a new neurologic deficit associated with abnormal neuroimaging findings and was categorized as either ischemic or hemorrhagic. Patients were considered to have gastrointestinal bleeding if they had one or more of the following: guaiac-positive stool, hematemesis, melena, active bleeding at the time of endoscopy

or colonoscopy, or blood in the stomach at the time of endoscopy or colonoscopy. Patients were considered to have an infection if they had one or more of the following: a driveline infection that required surgical treatment, a pump infection that required surgical treatment, or bacteremia (confirmed by positive blood cultures from 2 separate sites). Acute kidney injury within 7 days of LVAD implantation was defined according to the RIFLE (risk, injury, failure, loss of kidney function, and end-stage kidney disease) classification as an abrupt doubling of the serum creatinine level or a 50% reduction in the estimated glomerular filtration rate.<sup>15</sup>

## Statistical Analysis

Patient demographics, preoperative and operative characteristics, hemodynamic and echocardiographic data, and postoperative adverse events were compared between the 2 groups in a univariate analysis. Continuous variables were reported as mean  $\pm$  SD. Tests of normality were performed on each variable. Normally distributed variables were compared by using the Student *t* test; variables that were not normally distributed were compared by using the Mann-Whitney U test. Categorical variables were reported as number and percentage and were compared by using the Pearson  $\chi^2$  test (or the Fisher exact test if expected counts were not sufficiently large). We used  $\chi^2$  analysis to evaluate the difference in the number of patients who had postoperative adverse events.

Overall survival rates in the ICM and NICM groups were compared by using Kaplan-Meier analysis and the log-rank test, with long-term survival defined as 6 years after LVAD implantation. Cox proportional hazards analysis was used to identify independent predictors of mortality. Proportional hazards assumptions were tested by using Schoenfeld residuals. Preoperative and operative variables with *P* values  $< 0.20$  were included in the univariate analysis. Variables with a *P* value  $< 0.20$  in the univariate Cox analysis were included in our final multivariate model. Variables with a variance inflation factor  $> 10$  were removed to avoid introducing collinearity. Heart failure cause was forced into our model. *P* values  $< 0.05$  were considered statistically significant. All analyses were performed with SPSS, version 20 (SPSS, an IBM company).

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## Results

A total of 526 patients underwent primary implantation of a continuous-flow LVAD during the study period, including the HeartMate II in 403 (76.6%) and the HeartWare HVAD in 123 (23.4%). The ICM group included 256 patients (48.7%); the NICM group, 270 (51.3%) (Table I). From 2003 through 2009, 52 patients (43.7%) had ICM; from 2010 through 2016, 204 (50.1%) had ICM (*P*=0.22).

**TABLE I. Baseline Characteristics and Operative Details for the 526 Patients**

Variable	Ischemic Cardiomyopathy (n=256)	Nonischemic Cardiomyopathy (n=270)	P Value
Age (yr)	60.0 ± 9.8	50.0 ± 14.7	<0.001
Male	215 (84.0)	196 (72.6)	0.002
Body mass index*	27.4 ± 5.9	28.9 ± 7.3	0.27
Tobacco use	127 (49.6)	92 (34.1)	<0.001
Medical history			
Hypertension	177 (69.1)	142 (52.6)	<0.001
Diabetes	135 (52.7)	97 (35.9)	<0.001
Chronic obstructive pulmonary disease	42 (16.4)	32 (11.9)	0.13
Peripheral vascular disease	16 (6.3)	5 (1.9)	0.005
Stroke	32 (12.5)	41 (15.2)	0.36
Severe valvular regurgitation			
Mitral	46 (18.0)	62 (23.0)	0.16
Aortic	5 (2.0)	1 (0.4)	0.10
Tricuspid	24 (9.4)	35 (13.0)	0.19
Previous sternotomy	127 (49.6)	46 (17.0)	<0.001
Mechanical circulatory support**	138 (53.9)	131 (48.5)	0.22
INTERMACS profile	—	—	0.77
1	40 (15.6)	35 (13.0)	—
2	88 (34.4)	85 (31.5)	—
3	91 (35.5)	109 (40.4)	—
4	26 (10.2)	29 (10.7)	—
5+	11 (4.3)	12 (4.4)	—
Laboratory values			
Hemoglobin (g/dL)	11.4 ± 2.1	11.6 ± 2.2	0.26
White blood cell count (× 10 <sup>3</sup> /μL)	9.6 ± 4.3	9.1 ± 5.0	0.21
Platelets (× 10 <sup>3</sup> /μL)	208.0 ± 93.1	205.0 ± 94.1	0.71
Sodium (mEq/L)	135.2 ± 4.6	135.0 ± 4.5	0.52
Creatinine (mg/dL)	1.5 ± 0.8	1.4 ± 0.7	0.31
Blood urea nitrogen (mg/dL)	29.0 (19.8–40.0)	24.0 (18.0–34.0)	0.002
Aspartate aminotransferase (U/L)	37.5 (26.8–68.0)	36.5 (25–56.8)	0.009
Alanine aminotransferase (U/L)	30.0 (21.0–56.0)	30.0 (20.0–53.0)	0.12
Total bilirubin (mg/dL)	1.0 (0.6–1.6)	1.2 (0.7–1.9)	0.12
International normalized ratio	1.3 ± 0.6	1.2 ± 0.4	0.20
Hemodynamic and echocardiographic data			
Cardiac output (L/min/m <sup>2</sup> )	3.5 ± 1.6	3.3 ± 1.5	0.09
Central venous pressure (mmHg)	11.8 ± 7.3	11.7 ± 7.7	0.82
Left ventricular end-diastolic diameter (cm)	6.3 ± 1.0	6.8 ± 1.1	0.52
Mean pulmonary artery pressure (mmHg)	35.7 ± 10.5	35.7 ± 11.6	0.99
Pulmonary capillary wedge pressure (mmHg)	24.5 ± 10.0	25.1 ± 10.4	0.51
Operative details			
Device model			
HeartMate II	193 (75.4)	210 (77.8)	0.87
HeartWare HVAD	63 (24.6)	60 (22.2)	0.87
Cardiopulmonary bypass use	249 (97.3)	254 (94.1)	0.07
Cardiopulmonary bypass time (min)	91.6 ± 55.1	79.7 ± 51.8	0.09
Aortic cross-clamp use	24 (9.4)	26 (9.6)	0.92
Aortic cross-clamp time (min)	4.3 ± 17.3	5.2 ± 21.2	0.61
Concomitant procedures	96 (37.5)	84 (31.1)	0.12
Valve repair/replacement			
Aortic	12 (4.7)	10 (3.7)	0.87
Mitral	26 (10.2)	32 (11.9)	0.49
Tricuspid	18 (7.0)	21 (7.8)	0.87

INTERMACS = Interagency Registry for Mechanically Assisted Circulatory Support

\* Calculated as weight in kilograms divided by height in meters squared

\*\* Use of temporary ventricular assist device, intra-aortic balloon pump, Impella heart pump (Abiomed, Inc.), or extracorporeal membrane oxygenation

Data are shown as mean ± SD, as number and percentage, or as median and interquartile range. *P* < 0.05 was considered statistically significant.

The mean age of the ICM group was significantly greater than that of the NICM group, and the ICM group included a larger percentage of men, as well as smokers. Before LVAD implant, more of the ICM patients had hypertension, diabetes, peripheral vascular disease, and previous sternotomy. The ICM group also had significantly higher mean levels of blood urea nitrogen and aspartate aminotransferase. The groups were similar in terms of the percentage of patients who were bridged to transplant ( $P=0.45$ ), preoperative complete blood count, hemodynamic measurements, and distribution of INTERMACS profiles. Operatively, the groups were similar in terms of cardiopulmonary bypass use, cardiopulmonary bypass time, aortic cross-clamp use, and aortic cross-clamp time.

Of the 270 patients in the NICM group, 190 (70.4%) had idiopathic NICM (Table II). Among the remaining patients with NICM, the most common causes were viral myocarditis in 20 (7.4%), familial dilated cardiomyopathy in 16 (5.9%), valvular dysfunction in 12 (4.4%), and doxorubicin toxicity in 9 (3.3%).

The postoperative outcomes in both groups were largely similar. However, 16 patients (5.9%) in the NICM group recovered LV function to the extent that the LVAD could be explanted, compared with 5 patients (2.0%) in the ICM group ( $P=0.02$ ) (Table III). The ICM group had greater percentages of patients who were bridged to transplant (20.7% vs 17.0%), continued receiving LVAD support (32.8% vs 33.7%), or died (39.1% vs 35.2%).

In our analysis of postoperative adverse events, the 2 groups had similar rates of neurologic dysfunction, infection, acute kidney injury, and 30-day readmission (Table IV). Gastrointestinal bleeding was more frequent in the ICM group (31.3%) than in the NICM group (22.6%) ( $P=0.03$ ). To better characterize these events, we evaluated the site and cause of bleeding in each event (Table V). Notably, the ICM group had higher rates of bleeding in both the upper (28.1% vs 18.1%;  $P=0.007$ ) and lower (23.0% vs 13.3%;  $P=0.004$ ) gastrointestinal tracts. In particular, the ICM group had higher rates of gastrointestinal bleeding in the small intestine (4.7% vs 1.5%;  $P=0.03$ ) and colon (10.2% vs 5.6%;  $P=0.049$ ). The ICM group also had higher rates of gastrointestinal bleeding from arteriovenous malformations (20.7% vs 11.9%;  $P=0.006$ ) and ulcers (16.4% vs 9.3%;  $P=0.01$ ).

The ICM group also had a higher rate of diverticulosis (5.9% vs 3.3%), but this difference was not statistically significant ( $P=0.17$ ).

Finally, Kaplan-Meier analysis revealed that overall survival did not differ between the ICM and NICM groups ( $P=0.24$ , log-rank) (Fig. 1). Furthermore, multivariate Cox proportional hazards analysis revealed that the cause of heart failure was not an independent predictor of death ( $P=0.08$ ) (Table VI). Significant independent predictors included older age ( $P < 0.001$ ), total

**TABLE II. Causes of Nonischemic Cardiomyopathy in 270 Patients**

Cause	No. (%)
Idiopathic	190 (70.4)
Viral myocarditis	20 (7.4)
Familial dilated cardiomyopathy	16 (5.9)
Valvular dysfunction	12 (4.4)
Doxorubicin exposure	9 (3.3)
Left ventricular noncompaction	5 (1.9)
Peripartum cardiomyopathy	4 (1.5)
Alcohol use	4 (1.5)
Sarcoidosis	3 (1.1)
Hypertension	2 (0.7)
Amyloidosis	1 (0.4)
Chagas disease	1 (0.4)
Cocaine use	1 (0.4)
Congenital malformation	1 (0.4)
Scleroderma	1 (0.4)

**TABLE III. Postoperative Outcomes**

Outcome	ICM (n=256)	NICM (n=270)	P Value
Transplant	53 (20.7)	46 (17.0)	0.28
Continued LVAD support	84 (32.8)	91 (33.7)	0.83
LVAD explant	5 (2.0)	16 (5.9)	0.02
Death	100 (39.1)	95 (35.2)	0.36
Loss to follow-up	13 (5.1)	20 (7.4)	0.27

ICM = ischemic cardiomyopathy; LVAD = left ventricular assist device; NICM = nonischemic cardiomyopathy

Data are shown as number and percentage.  $P < 0.05$  (Pearson  $\chi^2$  test) was considered statistically significant.

bilirubin level ( $P=0.001$ ), previous sternotomy ( $P=0.03$ ), and concomitant procedures during LVAD implantation ( $P=0.048$ ).

## Discussion

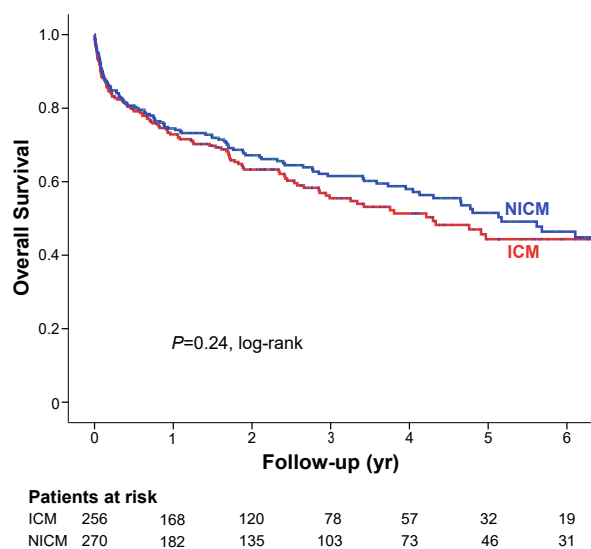
In this study, we found that the cause of heart failure did not independently predict long-term survival after continuous-flow LVAD implantation. However, we also found that gastrointestinal bleeding, particularly from arteriovenous malformations and ulcers, occurred significantly more often in the ICM group ( $P=0.03$ ). Multivariate Cox proportional hazards analysis showed that older age, previous sternotomy, total bilirubin level, and concomitant procedures during LVAD implantation were independent predictors of death. In addition, the mortality rate was higher for patients with ICM

**TABLE IV. Postoperative Adverse Events**

Adverse Event	Ischemic Cardiomyopathy (n=256)			Nonischemic Cardiomyopathy (n=270)			P Value
	No. of Patients (%)	No. of Events	Events/Pt-Yr	No. of Patients (%)	No. of Events	Events/Pt-Yr	
Neurologic dysfunction	73 (28.5)	101	0.237	68 (25.2)	91	0.169	0.43
Ischemic	43 (16.8)	55	0.129	39 (14.4)	44	0.082	0.46
Hemorrhagic	43 (16.8)	46	0.108	42 (15.6)	47	0.087	0.70
Infection	91 (35.5)	236	0.555	103 (38.1)	297	0.550	0.54
Pump	13 (5.1)	24	0.056	19 (7.0)	36	0.067	0.35
Driveline	21 (8.2)	42	0.099	34 (12.6)	73	0.135	0.10
Bacteremia	83 (32.4)	170	0.400	83 (30.7)	188	0.349	0.68
Gastrointestinal bleeding	80 (31.3)	131	0.308	61 (22.6)	85	0.158	0.03
Acute kidney injury	103 (40.2)	—	—	105 (38.9)	—	—	0.55
30-day readmission	54 (21.1)	—	—	46 (17.0)	—	—	0.24

Pt-Yr = patient-year

*P* < 0.05 ( $\chi^2$  test) was considered statistically significant.



**Fig. 1** Graph shows Kaplan-Meier estimates of overall survival after left ventricular assist device implantation in patients with either ischemic cardiomyopathy (ICM) or nonischemic cardiomyopathy (NICM).

(hazard ratio [HR]=1.32) (*P*=0.08). Larger studies of this difference are warranted.

The overall incidence of ICM in our study population (50.1%) was comparable to that among the 3,511 patients in the Nationwide Inpatient Sample database who had an LVAD implanted in the period from 2011 through 2014 (53.5%).<sup>9</sup> Our ICM group was older and included more men when compared with the NICM group. The patients with ICM also had fewer comorbidities (for example, tobacco use, peripheral vascular disease, diabetes mellitus, and hypertension). This finding

**TABLE V. Gastrointestinal Bleeding Characteristics**

Characteristic	ICM (n=256)	NICM (n=270)	P Value
Anatomic site			
Upper GI tract			
Esophagus	7 (2.7)	2 (0.7)	0.08
Stomach	48 (18.8)	35 (13.0)	0.07
Duodenum	15 (5.9)	7 (2.6)	0.06
NOS	2 (0.8)	5 (1.9)	0.28
Lower GI tract	59 (23.0)	36 (13.3)	0.004
Jejunum	10 (3.9)	9 (3.3)	0.72
Small intestine–NOS	12 (4.7)	4 (1.5)	0.03
Colon	26 (10.2)	15 (5.6)	0.049
Rectum	5 (2.0)	4 (1.5)	0.68
NOS	6 (2.3)	4 (1.5)	0.47
Cause			
AV malformation	53 (20.7)	32 (11.9)	0.006
Ulcer	42 (16.4)	25 (9.3)	0.01
Diverticulosis	15 (5.9)	9 (3.3)	0.17
Polyp	3 (1.2)	4 (1.5)	0.76
M-W syndrome	2 (0.8)	1 (0.4)	0.53
Unknown	15 (5.9)	16 (5.9)	0.97

AV = arteriovenous; GI = gastrointestinal; ICM = ischemic cardiomyopathy; M-W = Mallory-Weiss; NICM = nonischemic cardiomyopathy; NOS = not otherwise specified

Data are shown as number and percentage. *P* < 0.05 was considered statistically significant.

was expected because these comorbidities are risk factors for the development of ICM.<sup>16</sup> A greater percentage



**TABLE VI. Multivariate Cox Proportional Hazards Analysis of Mortality**

Variable	HR	95% CI	P Value
Age	1.03	1.01–1.04	<0.001
Total bilirubin	1.06	1.02–1.11	0.001
Previous sternotomy	1.30	1.04–1.87	0.03
Concomitant procedures	1.08	1.00–1.75	0.048
Heart failure cause	1.32	0.97–1.77	0.08
BUN	1.01	1.00–1.01	0.08
INR	1.15	0.88–1.49	0.31

BUN = blood urea nitrogen; HR = hazard ratio; ICM = ischemic cardiomyopathy; INR = international normalized ratio

$P < 0.05$  was considered statistically significant.

of patients with ICM had undergone a previous sternotomy, typically for coronary artery bypass grafting. (In the NICM group, previous sternotomy was typically for valvular repairs and replacements to treat valvular cardiomyopathy). Although viral and familial dilated cardiomyopathy and valvular disease were common causes of heart failure in our NICM cohort, by far the most frequent cause was idiopathic (70.4%). This was not unusual because the cause of NICM remains unknown in many cases<sup>17</sup>; regardless of the cause of chronic end-stage heart failure, invasive and costly diagnostic studies are usually not warranted in patients who are being considered for LVAD support because the results will not alter their care and management. The rarest causes in our NICM cohort were related to exposure to chemotherapeutic agents (for example, doxorubicin), peripartum cardiomyopathy, alcohol and cocaine use, viral infection or Chagas disease, and congenital disorders (for example, left ventricular noncompaction and congenital malformation).

A significantly greater percentage of NICM patients than ICM patients recovered LV function to the extent that they could have their LVADs explanted ( $P=0.02$ ). This finding concurs with an analysis by Goldstein and colleagues,<sup>18</sup> who concluded that LV recovery is more likely in young patients with NICM. Studies of myocardial response to LVAD therapy have shown that LV unloading may improve cardiomyocyte contractile function and normalize LV geometry and neurohormonal function.<sup>19–21</sup> Other studies suggest that reverse remodeling is less likely in patients with ICM, perhaps because of chronic and irreversible scarring after myocardial infarction, which may hamper recovery.<sup>18,22,23</sup> Of the NICM causes analyzed in this study, several are acute and may be more reversible and prone to reverse remodeling (although this may apply only to certain causes such as viral and postpartum cardiomyopathy). In contrast, some types of chronic NICM (those with hereditary or valvular causes, for example) may resist

reverse remodeling and recovery. Additional studies are warranted to understand fully the optimal conditions for reverse remodeling and the best timing for bridge-to-recovery strategies.

An unexpected and interesting finding was that postoperative gastrointestinal bleeding, particularly from arteriovenous malformations and ulcers, was more common in patients with ICM than in those with NICM. In our study, age at implantation may have contributed to this difference. This is consistent with the suggestion by Kawabori and associates<sup>24</sup> that age at implantation is the primary predictor of postoperative gastrointestinal bleeding. Of note, we also saw an increase over time in the proportion of LVAD recipients with ICM—from 43.7% (2003–2009) to 50.1% (2010–2016) ( $P=0.22$ ).

The association of gastrointestinal bleeding from arteriovenous malformations with nonpulsatile flow and anticoagulation has been established.<sup>24–26</sup> One theory is that the aortic stenosis effectively caused by axial flow prevents aortic valve opening and increases the shear stress caused by the pump, leading to acquired von Willebrand disease.<sup>27</sup> Other studies suggest that reduced pulsatility reduces intestinal mucosal perfusion, causing ischemia and formation of friable new vessels.<sup>28</sup> A recently proposed theory, related to the pathophysiology of arteriovenous malformation–related bleeding in continuous-flow LVAD recipients, is that angiodysplasia may be part of the natural pathophysiology of advanced heart failure. Patel and colleagues<sup>29</sup> reported a significantly higher prevalence of angiodysplasia ( $P=0.009$ ) in patients with advanced heart failure than in a control group. Clinically, our findings suggest that gastrointestinal bleeding may be encountered more frequently in LVAD recipients as the proportion of those who are older and have ICM grows. Consequently, our results warrant wider investigation to identify preventive strategies to reduce the incidence of such bleeding in this patient population.

### Limitations

Our study had several limitations. First, it was a non-randomized, retrospective, single-institution study and therefore inherently limited by its design. Second, the cause of the cardiomyopathy in most of our NICM patients was unknown, which may have confounded our results. Last, our classifications of heart failure causes in this study may not mirror the various classifications that have been used in other studies on this topic.

### Conclusions

We investigated postimplant complications and long-term survival in patients with ICM or NICM who underwent continuous-flow LVAD support. The cause of heart failure was not a significant independent predictor of survival after continuous-flow LVAD implantation

(HR=1.32;  $P=0.08$ ), and patients with ICM were more likely to have gastrointestinal bleeding, particularly from arteriovenous malformations and ulcers.

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