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Effects of Left Ventricular Assist Device Therapy on Ventricular Arrhythmias

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OBJECTIVES In a retrospective study, we sought to evaluate the effect of left ventricular assist device (LVAD) therapy on ventricular tachyarrhythmias in patients with advanced congestive heart failure.

BACKGROUND Despite the increasing use of LVAD as a bridge to cardiac transplantation, our knowledge regarding its effect on ventricular arrhythmias is currently limited to small series. Little is known about the prevalence, predictors, and clinical consequences of ventricular arrhythmias in LVAD recipients.

METHODS We reviewed the pre- and post-LVAD course of the last 100 consecutive adult patients to receive a HeartMate LVAD (Thoratec Laboratories Corp., Pleasanton, California) at our institution. All ventricular arrhythmias sustained for at least 30 s or requiring defibrillation were analyzed. All documented pre- and post-LVAD sustained ventricular arrhythmias were classified either as monomorphic ventricular tachycardia (MVT) or polymorphic ventricular tachycardia (PVT)/ventricular fibrillation (VF).

RESULTS Our population had an average age of 51 years, had predominately ischemic cardiomyopathy (63%), and a mean left ventricular ejection fraction of 20 ± 10%. New-onset MVT was observed in 18 patients who did not have MVT before LVAD placement. After LVAD, new-onset MVT was 4.5 times more likely than elimination of previously present MVT ($p = 0.001$), whereas the effect of LVAD on incidence of PVT/VF was not significant. In a multivariate Cox proportional hazards regression analysis, serum electrolyte abnormality was an independent predictor of post-LVAD ventricular arrhythmias. Preoperative MVT did not predict postoperative MVT.

CONCLUSIONS After LVAD placement, there is a significant rise in the incidence of de novo MVT. By contrast, the incidence of PVT/VF was unaffected by LVAD placement. (J Am Coll Cardiol 2005;45:1428–34) © 2005 by the American College of Cardiology Foundation

As the prevalence of advanced heart failure continues to increase, the left ventricular assist device (LVAD) has become an excellent bridge to cardiac transplantation and to possible other future alternative therapies (1,2). Our understanding of post-LVAD ventricular arrhythmias is currently limited to clinical data from a few small case series (3,4), and little is known about the prevalence, predictors, and clinical significance of post-LVAD ventricular arrhythmias. Even fewer data are available with regard to the effects of LVAD therapy on the prevalence and the electrophysiologic characteristics of pre-existing ventricular arrhythmias. In this retrospective study, we investigated the prevalence, predictors, electrocardiography, and clinical outcomes of ventricular arrhythmias in LVAD recipients.

METHODS

Subjects. We reviewed the last 100 consecutive available charts of patients to receive a HeartMate LVAD (Thoratec Laboratories Corp., Pleasanton, California) at the Columbia University Medical Center between July 1997 and March 2001. All but two patients received a LVAD for medically unresponsive advanced heart failure, whereas two patients received LVADs for uncontrollable ventricular tachyarrhythmias. All LVADs were implanted by the cardiothoracic surgical staff at our institution and placed at a left ventricular (LV) apical site as previously described (5). The majority of the patients received a LVAD as a bridge to cardiac transplantation. Eighteen of the 100 patients had biventricular assist devices placed at other institutions for stabilization, until transfer to our institution for biventricular assist device explantation followed by HeartMate LVAD implantation. Another 12 patients required right ventricular assist device (RVAD) placement at our institution for severe right heart failure after LVAD implantation.

Ventricular arrhythmia analysis. All available past medical records, physician and nurse notes, telemetry tracings, retrieved implantable cardioverter-defibrillator (ICD) tracings, and 12-lead electrocardiograms (ECGs), both preoperatively and postoperatively, generated during the admission before LVAD implantation and during any subsequent admissions, were reviewed. All available preoperative and postoperative ventricular tachyarrhythmias lasting for at least 30 seconds or requiring transthoracic defibrillation or an ICD shock for termination were analyzed. Ventricular arrhythmias were classified as monomorphic ventricular tachycardia (MVT), using the constancy of cycle length from one beat to the next, and the reproducibility of ECG or rhythm strip QRS morphology or ICD local signal

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Abbreviations and Acronyms

ECG = electrocardiogram
ICD = implantable cardioverter-defibrillator
LV = left ventricular
LVAD = left ventricular assist device
LVEF = left ventricular ejection fraction
MVT = monomorphic ventricular tachycardia
PVT = polymorphic ventricular tachycardia
RVAD = right ventricular assist device
VF = ventricular fibrillation

morphology from one beat to the next, based on separate review by two electrophysiologists, each blinded to the classification of the other. Sustained ventricular arrhythmias not meeting the criteria for MVT were classified as polymorphic ventricular tachycardia (PVT), or ventricular fibrillation (VF). No distinction was made between sustained PVT and VF, (henceforth, PVT/VF) for analysis of the results.

Of the 100 consecutive patients initially selected into our study, medical chart reviews identified 9 patients whose problem lists included ventricular tachyarrhythmia but for whom no actual documentation of any ventricular arrhythmia was available. These cases were not included in the analysis because without proper documentation, these arrhythmias could not be classified. Moreover, the distinction between MVT and PVT/VF was one of the main points of our analysis. The remaining 91 patients either did not have a history of any ventricular tachyarrhythmia, or had documented MVT, or PVT/VF, or both.

Available 12-lead ECGs were also analyzed in a blinded fashion by two separate cardiac electrophysiologists, for estimation of the approximate site of origin of MVTs, using a previously published algorithm (6). There was a 95% agreement rate between the two cardiac electrophysiologists. In cases of disagreement, a third, blinded electrophysiologist was asked to review the data for a final decision.

Several clinical and hemodynamic data, including blood test results and postoperative LVAD flows, were also analyzed. For laboratory data, the last recorded values within the 24 h before the ventricular tachyarrhythmia and the first recorded values within 24 h after the ventricular tachyarrhythmia were used. Serum electrolyte abnormalities analyzed as correlates of ventricular tachyarrhythmias included hypokalemia, hyperkalemia, hypomagnesemia, and hypocalcemia. These were defined as serum potassium concentration <3.5 mEq/l, >5.2 mEq/l, serum magnesium concentration <1.5 mEq/l, and corrected serum calcium concentration <8.0 mEq/l, respectively, in accordance with the normal ranges of our hospital laboratory.

Statistical analysis. Proportions from different subgroups were compared by chi-square test. For comparisons within small groups in which the total data points were <40, a Fisher exact test was used. In comparing the prevalence of arrhythmias before and after LVAD therapy, McNemar's chi-square test was used. Continuous variables were compared using a Student t test. To investigate the association of postoperative ventricular arrhythmia and all-cause mortality, a Cox proportional hazards survival analysis was used. Once the transplant was complete, the patients were censored from survival analysis. A Cox proportional hazards analysis was also constructed to demonstrate the strength and independence of predictors of postoperative ventricular arrhythmias. Based on the number of ventricular arrhythmia events observed after LVAD, we selected and tested five clinical factors for MVT and four for PVT/VF, as potential predictors of post-LVAD ventricular arrhythmia. In this analysis, time to event was defined as time to onset of ventricular arrhythmia after LVAD implantation. The patients who underwent cardiac transplantation or died before a ventricular tachyarrhythmia event were censured from this analysis. A p value <0.05 was considered statistically significant.

RESULTS

Clinical characteristics. Of the initial 100 patients whose charts were reviewed, our population consisted of 91 patients who had either no pre-LVAD ventricular arrhythmias or documented pre-LVAD ventricular arrhythmias. Their clinical characteristics are shown in Table 1. Only the two patients whose indication for LVAD was uncontrollable ventricular arrhythmias were not receiving intravenous inotropic therapy before LVAD.

Arrhythmia prevalence. One hundred eighteen episodes of documented sustained clinical ventricular arrhythmia occurred in 30 patients (3.9 episodes per patient) from 3 years to 1 day before LVAD implantation. One hundred seventy-nine episodes of sustained ventricular arrhythmia occurred in 32 patients (5.6 episodes per patient) from 1 day to 126 days after LVAD placement. Of the 30 patients with ventricular arrhythmia before LVAD, 9 had documented MVT, 23 had documented PVT/VF, and 2 had both (Table 2). Of the 32 patients with post-LVAD ventricular arrhyth-

Table 1. The Clinical Characteristics of the Patient Population

Characteristics	Total N = 91 (%)
Age (yrs)	51 ± 10
Male	74 (81)
Ischemic heart disease	57 (63)
LVEF (%)	20 ± 10
Patients on amiodarone therapy before LVAD	38 (42)
Patients with cardiac arrest prior to LVAD	28 (30)
Patients with MVT or PVT/VF before LVAD	30 (33)
Patients with MVT or PVT/VF after LVAD	32 (35)
Patients with ICD implanted prior to LVAD	17 (19)
Patients with NYHA functional class IV before LVAD	81 (89)
Patients on inotropic support prior to LVAD	89 (98)
Patients on inotropic support after LVAD	91 (100)

ICD = implantable cardioverter-defibrillator; LVAD = left ventricular assist device; LVEF = left ventricular ejection fraction; MVT = monomorphic ventricular tachycardia; NYHA = New York Heart Association; PVT = polymorphic ventricular tachycardia; VF = ventricular fibrillation.