Characteristics of Ventricular Tachycardia Ablation in Patients With Continuous Flow Left Ventricular Assist Devices

Frederic Sacher, MD, PhD; Tobias Reichlin, MD; Erica S. Zado, PA-C; Michael E. Field, MD; Juan F. Viles-Gonzalez, MD; Petr Peichl, MD, PhD; Kenneth A. Ellenbogen, MD; Philippe Maury, MD; Srinivas R. Dukkipati, MD; Francois Picard, MD; Josef Kautzner, MD, PhD; Laurent Barandon, MD, PhD; Jayanthi N. Koneru, MD; Philippe Ritter, MD; Saagar Mahida, MBChB; Joachim Calderon, MD; Nicolas Derval, MD; Arnaud Denis, MD; Hubert Cochet, MD, PhD; Richard K. Shepard, MD; Jerome Corre, MD; James O. Coffey, MD; Fermin Garcia, MD; Meleze Hocini, MD; Usha Tedrow, MD; Michel Haissaguerre, MD; Andre d’Avila, MD; William G. Stevenson, MD; Francis E. Marchlinski, MD; Pierre Jais, MD

Background—Left ventricular assist devices (LVADs) are increasingly used as a bridge to cardiac transplantation or as destination therapy. Patients with LVADs are at high risk for ventricular arrhythmias. This study describes ventricular arrhythmia characteristics and ablation in patients implanted with a Heart Mate II device.

Methods and Results—All patients with a Heart Mate II device who underwent ventricular arrhythmia catheter ablation at 9 tertiary centers were included. Thirty-four patients (30 male, age 58±10 years) underwent 39 ablation procedures. The underlying cardiomyopathy pathogenesis was ischemic in 21 and nonischemic in 13 patients with a mean left ventricular ejection fraction of 17%±5% before LVAD implantation. One hundred and ten ventricular tachycardias (VTs; cycle lengths, 230–740 ms, arrhythmic storm n=28) and 2 ventricular fibrillation triggers were targeted (25 transseptal, 14 retrograde aortic approaches). Nine patients required VT ablation <1 month after LVAD implantation because of intractable VT. Only 10/110 (9%) of the targeted VTs were related to the Heart Mate II cannula. During follow-up, 7 patients were transplanted and 10 died. Of the remaining 17 patients, 13 were arrhythmia-free at 25±15 months. In 1 patient with VT recurrence, change of turbine speed from 9400 to 9000 rpm extinguished VT.

Conclusions—Catheter ablation of VT among LVAD recipients is feasible and reasonably safe even soon after LVAD implantation. Intrinsic myocardial scar, rather than the apical cannula, seems to be the dominant substrate.

Key Words: ablation ■ ventricular arrhythmia ■ ventricular assist device ■ ventricular tachycardia

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Ablation Procedure
Patients were referred for ablation because of refractory recurrent VAs. Transesophageal or intracardiac echocardiography was performed if a retrograde aortic approach was planned to rule out thrombus in the aortic root or in patients with AF to rule out left atrial appendage thrombus. The procedure was performed in patients fully anticoagulated either having a therapeutic International Normalized Ratio (between 2 and 3) or receiving unfractionated heparin with an activated clotted time >250/300 depending on centers. Femoral venous and arterial access was obtained for vascular access and hemodynamic monitoring (Figure 2). A 3-dimensional electro-anatomic mapping system (Carto 3, Biosense Webster or Velocity, St Jude Medical) was used to perform substrate mapping and to annotate points of interest. Endocardial voltage maps were created, and scar was defined as an area with voltage <1.5 mV. Except in cases of incessant arrhythmias, programmed stimulation was performed to induce and map VT. A macro-reentrant mechanism was diagnosed based on activation pattern and entrainment mapping, whereas a VT was considered focal or caused by micro-reentry when activation was concentric. VT was defined as related to the cannula when activation showed either an origin in the vicinity of the cannula with centrifugal activation or an activation consistent with a circuit turning around the cannula.

The ablation strategy was primarily targeting the clinical/induced arrhythmia via activation and entrainment mapping. Whether additional substrate modification was performed was dependent on the preference of the individual operators.

Irrigated tip ablation catheters were used for all procedures.

Statistical Analysis
Categorical variables were described as numbers with corresponding percentages and compared by using the χ² test. Continuous variables were described as means±SD or median [25th, 75th percentile] when not normally distributed. Comparisons were performed using the Student’s t test or the Wilcoxon rank test when not normally distributed. Statistical analyses were performed by using SPSS Software. All P values were 2-tailed with statistical significance set at 0.05. All confidence intervals were calculated at the 95% confidence interval.

Results
Population
Thirty-four patients (30 male, mean age 58±10 years old) with HM2 LVAD underwent VT/VF ablation in 9 electrophysiological centers between 2009 and 2014 (Table). The indication for LVAD implantation was progressive heart failure (n=21, 62%) or acute cardiogenic shock (n=13, 38%) with the underlying etiologies for the cardiomyopathy either ischemic (n=21, 62%) or nonischemic (n=13, 38%). In 25 (74%) patients, the LVAD implantation was considered a bridge to cardiac transplant. Mean turbine speed was 8968±491 rpm (range, 7800–9800).

An ICD was present in 32 patients (94%), including 2 which were implanted after LVAD placement. ICDs were implanted for secondary prevention in 13 (41%) patients. Patients were implanted with a single chamber (n=11), a dual chamber (n=4), or a cardiac resynchronization therapy (n=17) device. Two patients did not have an ICD at the time of their ablation. Of note, 3 patients required ICD replacement (Atlas, St Jude Medical n=1; Paradym, Sorin Group n=2) after LVAD implantation because of interference that prevented device interrogation.

The mean left ventricular ejection fraction was 17%±5% with a mean LV end diastolic diameter before LVAD of 71±9 mm that shortened to 57±11 mm (P<0.001) at least 1 month after LVAD implantation.
Ventricular Arrhythmia Characteristics in Patients Undergoing Catheter Ablation

Before LVAD implantation, 8 patients had experienced arrhythmic storm and 19 patients had at least one VT/VF episode, whereas 7 had no prior history of VA. After LVAD implantation, VA occurred in all 34 patients after a median of 11 [2–58] days (min–max, 0–510 days); 23 patients experienced arrhythmic storm post LVAD implantation. All of them had at least 1 VA episode before LVAD implantation. Nine patients required VT ablation <1 month after LVAD implantation because of intractable VT. These patients more often had arrhythmic storm episodes before LVAD implantation (5/9; 56%). On the contrary, patients without VA before LVAD implantation experienced VA later after implantation (60 days [27–210] versus 7 days [2–25]; P=0.02). Symptoms during VA were mainly related to ICD shocks (n=19) and, less commonly, syncope/lightheadedness (n=7) or asthenia/palpitation (n=8).

Mapping and Ablation

Five patients had a history of VT ablation before LVAD implantation. After implantation, the 34 patients underwent 39 new ablation procedures. A transseptal approach was used in 25 procedures (74%) and a retrograde-aortic approach in the remaining 14 (36%). Pericardial access was not attempted in any patient. An electro-anatomic mapping system (CARTO 3, Biosense Webster n= 37, or Velocity, St Jude Medical n=1) was used in all but one case (Figure 3) to perform a substrate map in baseline rhythm. In 2 other patients, interference between LVAD and the CARTO system prevented mapping the entire LV (transient loss of catheter visualization in some areas). In 6 patients, no endocardial scar could be identified. In the remainder, the mean scar surface was 65±50 cm² (median, 57 cm² [23–72]) with a maximum of 239 cm² (93% of the LV surface; Figure 3A) and a mean total LV surface of 202±39 cm². Scar locations were limited to the basal area in 4 (all patients with nonischemic cardiomyopathy), but involving the apex (insertion site of the cannula) in 11. They were also located at the anterior n=13, septal n=10, inferior n=8, and lateral n=7 LV wall. Of note, >1 segment was involved in some patients.

One hundred and ten VTs (median 2 [1–4.5] per procedure) with a cycle length from 230 to 740 ms and 2 VF

Table. Clinical Characteristics of the Population at LVAD Implantation

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>n=34</th>
</tr>
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<tbody>
<tr>
<td>Sex, male (n, %)</td>
<td>30 (88%)</td>
</tr>
<tr>
<td>Age, y</td>
<td>58±10</td>
</tr>
<tr>
<td>Ischemic CMP</td>
<td>21 (62%)</td>
</tr>
<tr>
<td>VT/VF pre-LVAD</td>
<td>27 (79%)</td>
</tr>
<tr>
<td>Arrhythmic storm pre-LVAD</td>
<td>8 (24%)</td>
</tr>
<tr>
<td>Atrial arrhythmia</td>
<td>14 (41%)</td>
</tr>
<tr>
<td>Beta-blocker therapy</td>
<td>24 (71%)</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>28 (82%)</td>
</tr>
<tr>
<td>LVEF pre-LVAD</td>
<td>17%±5%</td>
</tr>
<tr>
<td>LVEDD (mm) pre-LVAD</td>
<td>71±9 mm</td>
</tr>
<tr>
<td>Normal RV function</td>
<td>21 (62%)</td>
</tr>
<tr>
<td>ICD</td>
<td>32 (94%)</td>
</tr>
<tr>
<td>CRT</td>
<td>17/32 (53%)</td>
</tr>
</tbody>
</table>

CMP indicates cardiomyopathy; CRT, cardiac resynchronization therapy; ICD, implantable cardioverter-defibrillator; LVAD, left ventricular assist device; LVEDD, left ventricular end diastolic diameter; LVEF, left ventricular ejection fraction; RV, right ventricle; VF, ventricular fibrillation; and VT, ventricular tachycardia.
triggers were targeted during these 39 procedures. Of note, in 2 patients referred for VT ablation, no VA could be induced during the procedure.

The suspected VT mechanisms based on the electrophysiology study were macro-reentry (n=105, 95%), focal or microreentry (n=4, 4%), and bundle branch reentry (n=1, 1%). A possible epicardial VT origin was suspected for 6 VTs (5 patients with nonischemic cardiomyopathy and 1 with ischemic cardiomyopathy). Only 10/110 (9%) of the targeted VAs were related to the HM2 cannula site based on activation and entrainment mapping (Figure 4). In 9 of these patients, the VT mechanism was macro-reentry around the cannula insertion site. The preexisting substrate was anterior scar through which the cannula had been inserted. In the remaining patient, the VT was related directly to the cannula which touched the LV endocardium.

In these 10 patients in whom VT was related to the cannula, the first episode of VT occurred at a median of 38 days [3.5–187] after HM2 placement. In 5 of these patients with VT related to the cannula, VT occurred within 1 month of LVAD placement, in one at 2 months, and in the remaining 4, after 5 months. No particular 12-lead ECG VT morphology could be identified for VTs that were related to the cannula.

In the remaining 24 patients in whom VA was not related to the cannula, the first episode of VA occurred at a median of 8 days [2–30] after LVAD placement. In 5 of these patients without VA related to the cannula, VT occurred within 1 month of LVAD placement, in one at 2 months, and in the remaining 4, after 5 months. No particular 12-lead ECG VT morphology could be identified for VTs that were related to the cannula.

In the remaining 24 patients in whom VA was not related to the cannula, the first episode of VA occurred at a median of 8 days [2–30] after LVAD placement (P=0.07 compared with VT related to the cannula). VA started mainly during the first month (n=19), but continued to appear regularly thereafter. For 5 of the 7 patients without any VA episodes before HM2 implantation, VT began to occur >1.5 months after LVAD implantation.

Acute success (absence of VT inducibility at the end of the case or successful elimination of a culprit PVC in the case of a VF trigger) was present in 30/39 procedures (77%). Median RF duration was 24 minutes [10–35.5] ranging from 2 to 118 minutes. In 14 procedures, only VT/VF triggers were targeted, whereas additional substrate ablation was performed in the remaining 25 ablations.

Complications
One patient with VT ablation for arrhythmic storm developed cardiogenic shock with acidosis. One patient experienced a transient ischemic attack and another a stroke 8 days after ablation. Red blood cell transfusion (2 U) was required after groin hematoma in 1.

Outcome
During follow-up, 7 patients underwent cardiac transplantation, including one with VT recurrence before transplant, and 10 patients died (septic shock n=2, massive stroke n=2, intracranial hemorrhage n=1, hemolysis n=1, LVAD deactivation n=1, terminal heart failure n=1, sudden death n=1, and during redo emergency surgery as a result of cable failure n=1). Of the remaining 17 patients, 13 were arrhythmia-free at 25±15 months (median 24 months) after their last ablation procedure. In 1 patient with VT recurrence, a change of turbine speed from 9400 to 9000 rpm extinguished VT.
Discussion

The predominant substrate for VA in patients referred for ablation after HM2 implantation in our series is their underlying ventricular scar with a macro-reentrant mechanism rather than mechanical induction from the left ventricular cannula. Although some studies speculated that VT in LVAD patients could be linked to the cannula, others have reported findings similar to ours. Cantillon et al. found that 75% of VTs originated from the intrinsic scar versus 14% from the cannula area. They also found some VTs caused by micro-reentrant mechanisms (7%) and bundle branch reentry (3.5%). In our study, all patients with VT originating from the cannula area had the cannula inserted within an antero-apical scar and 9/10 had macro-reentrant VT.

Patients having VA early after LVAD implantation (<1 month) all had a history of VA before LVAD implantation. It has been reported previously that the risk of VA after LVAD was extremely low in the absence of VA before LVAD implantation (4% versus 46%). In our series, 7/34 (21%) did not have VA before LVAD implantation. In these patients, VA occurred later compared with patients with a history of VA (60 days [27–210] versus 7 days [2–25]; P=0.02). Interestingly, in patients with VT originating near the cannula, only half experienced VA within 1 month after LVAD implantation. Even if VT seems to occur later in this population (median: 38 days) compared with patients with VT from other locations (median: 8 days), this difference was not statistically significant (P=0.07). However early VT occurrence (<48 hours after LVAD placement) was more frequent for VT not related to the cannula. Mechanisms of early appearance of VT post LVAD implantation are not fully understood. Fluid and electrolyte shifts as well as autonomic nervous system imbalance may certainly play a role in the arrhythmia onset, especially as β-blockers are often withheld during the postoperative period. Further, LVAD implantation leads to more specific changes, such as ventricular unloading with changes in parietal stretch that may alter the electric properties of the tissue (refractory periods or conduction times), particularly in the scar area. However, no significant difference was observed in our study in terms of LV end-diastolic diameter reduction in patients with early versus late occurrence of VA after LVAD implantation.

Another possible mechanism is suction applied to an adjacent ventricular wall from the cannula, which occurred in at least one of our patients in whom a change of turbine speed from 9400 to 9000 rpm extinguished VT. The surgeon should be aware of this complication to prevent it. It is probably important to orient the cannula toward the infero-lateral part of the LV because in the closed chest, the diaphragm will push the device and may orient it toward the septum.

In patients undergoing LVAD implantation with a history of VA, the optimal VA management is unknown. Occurrence of VA post LVAD implantation, particularly in the early phase, is deleterious by increasing ICU length of stay and drug requirement. In the patients with a high risk of developing VA post LVAD implantation, prophylactic ablation may be an option, but the risk to benefit ratio of this approach is unknown. Based on the data from our series and others, performing ablation before LVAD implantation will likely result in overtreatment with associated procedural risk in this sick population. Another option is to perform substrate ablation during LVAD implantation as proposed by Mulloy et al. This approach requires precise knowledge of the area of scar, potentially obtained preoperatively by cardiac MRI or computed tomography scan, for example, to plan the approach and minimize the cross-clamp and perfusion times. In their experience, cryoablation at the time of LVAD implantation resulted in a dramatically shorter ICU length of stay (165 versus 441 hours; P=0.01) and postoperative hospital length of stay (26 versus 57 days; P=0.03). The final option is to perform only catheter ablation in the case of recurrent VA post LVAD implantation, as was the case in our series.

Patients generally received an HM device because they could not be immediately transplanted because of clinical status or absence of a donor heart or that they received the LVAD as destination therapy because of contraindication to transplantation. When feasible, cardiac transplantation is likely a better strategy than catheter ablation of VT because it eliminates the substrate both for the VT and end-stage heart failure. Catheter ablation may not improve survival but improves quality of life by reducing ICD therapies.

Practical Consideration for VT Ablation in LVAD Patients

Given the absence of a pulsatile peripheral pulse with a continuous flow device (Figure 2), the automatic sphygmomanometer may be misleading. It is crucial to invasively monitor arterial blood pressure. In the case of retrograde aortic access, it is important to recognize that there is no or little flow going across the aortic valve, and it may then be difficult to cross the valve with the ablation catheter because of the absence of aortic valve opening. This can be facilitated by transiently decreasing the LVAD flow. Moreover, there is risk of dislodging any thrombus that can be formed in the aortic root even in patients who are fully anticoagulated; therefore, it is reasonable to perform transesophageal echocardiography before attempting retrograde aortic access to the LV. For transseptal access, a steerable sheath is extremely helpful.

The risk of catheter entrapment in the cannula is extremely low. Several times during these procedures, catheters went into the initial part of the cannula without any adverse event. The catheters were never aspirated by the cannula. However, they should not be advanced too far in, particularly never beyond the initial portion of the cannula where the turbine is located.

As previously reported, 3 patients had LVAD interference that prevented ICD interrogation and programming (Atlas, St Jude Medical n=1; Paradigm, Sorin Group n=2). LVADs can also produce interference with the 3-dimensional electro-anatomic mapping systems, which we did encounter in 2 patients, resulting in areas (inferior apical LV wall close to the turbine) where the ablation catheter could not be visualized during mapping.

Limitations

This is an observational study with a limited number of patients included because of the specific entity studied. Because of the study design (observational and multicenter), ablation strategy
was not uniform in all patients, but reflects current strategies in different centers.

**Conclusions**

In this study, all patients developing intractable VT soon after LVAD implantation have a history of VT before HM II implantation. Despite a high mortality rate among LVAD recipients, catheter ablation of VT is effective and relatively safe even within 1 month post implantation. Intrinsic myocardial scar, rather than the apical cannula, seems to be the dominant substrate.

**Disclosures**

F. Sacher has received lecture honorarium from Biosense Webster and is consultant for St Jude Medical and Sorin Group. K. Ellenbogen is consultant and received research grant and fellowship support from Biosense Webster. J. Kautzner is a member of Scientific Advisory Board of Biosense Webster, Medtronic, and St Jude Medical. He received lecture honorarium from Biosense Webster, Boston Scientific, Medtronic, and St Jude Medical. J. Kautzner is a member of Scientific Advisory Board of Biosense Webster. P. Jais has received fellowship support and research grant from Biosense Webster. F. Marchlinski has received lecture honorarium and is part of the Scientific Advisory Board of Biosense Webster. M. Haissaguerre has received lecture honorarium and is part of the Scientific Advisory Board of Biosense Webster. P. Jais has received lecture honorarium and is part of the Scientific Advisory Board of Biosense Webster and St Jude Medical. The other authors report no conflicts.

**References**


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