Extracorporeal Membrane Oxygenation for Hemodynamic Support of Ventricular Tachycardia Ablation

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Background—We report the experience in a cohort of consecutive patients receiving extracorporeal membrane oxygenation during catheter ablation of unstable ventricular tachycardia (VT) at our center.

Methods and Results—From 2010 to 2015, extracorporeal membrane oxygenation was initiated in 64 patients (average age: 63±15 years; left ventricular ejection fraction in 27±9%; cardiogenic shock in 23%, and electrical storm in 62% of patients) undergoing 74 unstable VT catheter ablation procedures. At least one VT was terminated in 81% of procedures with baseline inducible VT, and VT noninducibility was achieved in 69%. Acute heart failure occurred in 5 patients: 3 underwent emergency heart transplantation, 1 had left ventricular assist device (LVAD) implantation, and 1 patient eventually died because of subsequent mesenteric ischemia. All other patients were discharged alive. After a median follow-up of 21 months (13–28 months), VT recurrence was 33%; overall survival was 56 out of 64 patients (88%). Extracorporeal membrane oxygenation–supported ablation was the bridge to LVAD in 6.9% and to heart transplantation in 3.5% of patients. VT recurrence was related to ablation success (after 180 days of follow up: 19% when VT was noninducible, 42% if nonclinical VT was inducible, 75% when clinical VT was inducible, and 75% in untested patients, P<0.001). Incidence of all-cause death, heart transplantation, and LVAD was independently related to ablation outcome (at 180 days of follow-up: 9% when noninducibility was achieved, 50% in case of inducible VT, and 75% in untested patients, P<0.001). At multivariable analyses, noninducibility (hazard ratio 0.198; P=0.001) and left ventricular ejection fraction (hazard ratio 0.916; P=0.008) correlated with all-cause death, LVAD, and heart transplantation.

Conclusions—Ablation of unstable VTs can be safely supported by extracorporeal membrane oxygenation, which allows rhythm stabilization with low procedure mortality, bridging uncompensated patients to permanent LVAD or heart transplantation. Successful ablation is associated with better outcomes than unsuccessful ablation. (Circ Arrhythm Electrophysiol. 2016;9:e004492. DOI: 10.1161/CIRCEP.116.004492.)

Key Words: catheter ablation ■ cardiac arrhythmias ■ heart failure ■ ventricular tachycardia

Catheter ablation (CA) is an effective treatment for ventricular tachycardia (VT) in the setting of structural heart disease, preventing long-term arrhythmia and electrical storm (ES) recurrences and favorably affecting hospitalization and survival.1

In patients with poor systolic function and advanced heart failure, recurrent nontolerated VTs causing implantable cardioverter defibrillator shocks and prolonged incessant arrhythmias may cause further deterioration of target organ perfusion. Furthermore, multiple comorbidities may increase the risk of acute hemodynamic decompensation and periprocedural multiorgan failure2-4 and mortality triggered by myocardial stunning secondary to prolonged times under general anesthesia.

Current mapping techniques allow the accurate identification of the arrhythmia substrate and subsequent effective modification during sinus rhythm (SR),5,6 thus avoiding prolonged activation and entrainment mapping times during hypotensive VT. Substrate modification, however, may not be always effective. Reasons include an absence of defined SR targets, an absence of a well-defined scar as in the setting of idiopathic dilated cardiomyopathy,7 and residual VTs that may be induced even after an otherwise successful substrate modification procedure.8,9 The feasibility of mechanical circulatory
WHAT IS KNOWN
• Patients needing catheter ablation of ventricular tachycardia often have severe heart disease and multiple comorbidities that may increase the risk of peri-procedural hemodynamic decompensation, multi-organ failure and mortality, possibly related to myocardial stunning with ventricular tachycardia and long anesthesia times.
• Substrate ablation performed in sinus rhythm is not always feasible, and when mapping during unstable ventricular tachycardia is required, mechanical circulatory support can be considered, but the best strategy is not clear.

WHAT THE STUDY ADDS
• Extracorporeal membrane oxygenation was implemented in a series of patients according to a specific ventricular tachycardia–related triage.
• Extended intra- and peri-procedural extracorporeal membrane oxygenation support allows lengthy and complex ablation procedures with mapping to be completed with a low incidence of heart decompensation and acute death.

support in this setting is attractive, but no consensus on the best strategy has been reached.

The aim of our study is to report on the systematic use of extracorporeal membrane oxygenation (ECMO) to provide circulatory support in a consecutive cohort of high-risk patients undergoing CA of VT.

Methods
This study reports on a consecutive cohort of patients with structural heart disease and drug-refractory VT who underwent CA with ECMO at the San Raffaele University Hospital, Milan, between 2010 and 2015.

The study is in compliance with the declaration of Helsinki and was undertaken with the approval of the local ethical committee. All patients provided written informed consent.

Risk Assessment
Arrhythmia pattern was classified as paroxysmal, incessant VT, or ES (defined as the occurrence of ≥3 episodes of VT separated by ≥5 minutes during a 24-hour period). Hemodynamic state was evaluated in relation to both VT tolerance and during SR. Cardiogenic shock was defined as persistent hypotension (systolic blood pressure <90 mmHg and mean arterial pressure [MAP] 30 mmHg lower than baseline or <70 mmHg, with associated signs of end-organ hypoperfusion) with severe reduction in cardiac index (<1.5 and <8.3 L/min/m2 with pressor agents) and adequate or elevated filling pressure (eg, left ventricular end-diastolic pressure >18 mmHg or right ventricular end-diastolic pressure >15 mm Hg).15

Arrhythmia pattern, hemodynamic state, left ventricular systolic function, extent of coronary artery disease, and the presence of comorbidities (chronic kidney disease defined as serum creatinine ≥1.5 mg/dL and severe pulmonary disease based on the presence of PCO2 >50 mmHg) were used to classify patients into high and low risk, determining the timing of intervention and the need for the admission to the intensive care unit (ICU) versus the ventricular tachycardia unit, based on the systematic implementation of an algorithm as previously described.1

Selection of the Study Cohort
The experience with ECMO at our center was started as a bailout strategy in 2010. After preliminary encouraging results, we systematically expanded the use to other groups of patients in 2013, aiming to reduce bailout and expanding the overall series that may benefit from this approach.

Strategies for VT CA with ECMO included both preemptive and bailout indications:
1. Bailout indications
   1.1 Intra-procedural hemodynamic deterioration leading to cardiogenic shock.
   2. Preemptive indications
   2.1 Pre-procedural incessant VT/ES leading to cardiogenic shock, stabilized within the ICU before the ablation procedure.
   2.2 High-risk preoperative assessment and nontolerated VTs
   2.3 Previous ineffective substrate modification procedure performed during SR in patients with nontolerated VTs requiring a second ablation based on activation mapping and entrainment.

Extracorporeal Membrane Oxygenation
The ECMO circuit setup included a centrifugal pump and a coated polymethylpentene oxygenator (Revolution [Sorin, Italy], Centrimag [Levitronix, Waltham, MA], Rotaflow [Maquet, Germany]). All patients received peripheral cannulation.

Outlet cannulas ranged from 21 to 25 F, and inlet cannulas ranged from 15 to 19 F (Maquet; Jostra Medizintechnik AG, Hirrlingen, Germany). Pump flow was set to obtain a cardiac index between 2.0 and 2.4 L/min/m2.

Continuous monitoring of invasive arterial pressure, pulse oximetry, electrocardiography, urine output, and central venous pressure was performed. Blood gases were assayed at least every 2 hours. All patients received antibiotic prophylaxis. A perfusionist was continuously involved in the patients’ care.

During ECMO, heparin was administered to a target activated clotting time >250 seconds.16

After CA, in the ICU, unfractionated heparin or bivalirudin was administered targeting an activated partial thromboplastin time value between 45 and 50 seconds.16

Coagulation profile was monitored every 6 hours or when deemed necessary.

Ablation Procedure
Antiarrhythmic drug therapy was discontinued at least 5 half-lives before the procedure, when clinically appropriate. A first-line combined epicardial and endocardial mapping approach was undertaken in selected patients with nons ischemic etiologies; in patients with ischemic heart disease, the epicardial approach was undertaken after a previous endocardial failure. The epicardial puncture was performed before the institution of ECMO.

Patients underwent high-density electroanatomical mapping (defined as a 5-mm interpolation fill threshold in areas with a bipolar voltage <1.5 mV and 10 mm elsewhere) with the CARTO 3 (Biosense Webster, Diamond Bar, CA) or NavX Ensite Velocity (St. Jude Medical, Inc, Milwaukee, WI) systems. Simultaneous bipolar and unipolar maps were acquired to define areas of low voltage (respectively <1.5 and <8.3 mV) or scar (<0.5 and <5.0 mV). Absence of bipolar scar was defined as confluent scar involving <1 segment (6% of the total surface area).1 Late potentials were defined as local ventricular potentials occurring after the termination of the surface QRS.

Mapping and ablation times were collected for each procedure. Total procedure time was calculated from the entrance to the electrophysiology laboratory and then to the transfer to the ICU, including the time required for cannulation and general anesthesia.

Ablation Strategy
1. Activation mapping and substrate ablation:
1.1. Patients presenting with incessant VT at the beginning of the procedure underwent activation mapping-guided CA, and after VT termination, substrate modification was attempted during sinus or paced rhythm.

1.2. Patients in SR underwent substrate mapping followed by programmed ventricular stimulation and ablation guided by activation mapping during VT. After VT termination, CA continued in SR aiming at substrate modification.

2. Activation mapping ablation: In cases with inducible VTs and absence of targetable substrate during SR.

3. Substrate ablation: In patients in whom VT was not inducible, CA was performed in SR aiming for substrate modification. Substrate modification was performed aiming to abolish late potentials or, in absence of late potentials, fragmented abnormal electrograms within the QRS.

Activation mapping was performed as follows: during ongoing VT, point-by-point mapping was achieved by moving the catheter within the area of low voltage, starting from areas of maximum delay during SR. Local EGM activation time was measured with respect to earliest QRS onset on surface ECG, looking for diastolic electrical activity. The functional role of any diastolic potential was assessed by entrainment.17

End points of CA were VT termination during RF delivery guided by activation mapping, late potential abolition at remap during SR, and non-inducibility after CA with programmed ventricular stimulation ≤ 4 extrastimuli at the right ventricular apex and multiple left ventricular sites.

Post Procedure

After the procedure, patients were admitted to the ICU and managed according to institutional protocols for ECMO. As a rule, mechanical support was continued overnight, and weaning was performed after at least 24 hours.

Within the first 7 days after the procedure, peak values of daily arterial lactate, serum creatinine, and aspartate aminotransferase were sampled. Echocardiography was performed before the procedure and after seven days. Renal failure was defined according to the Acute Kidney Injury Network classification.18

Follow-Up

Clinical follow-up visits and implantable cardioverter defibrillator interrogations were scheduled at 1, 3, 6, and 12 months with additional telephone consultation every 6 months. VT recurrence rate is presented according to the last CA result.

**Statistical Analysis**

Continuous variables are presented as means (±SD) or medians (quartiles, Q1–Q3) and categorical variables as numbers and percentages. Comparisons between groups were undertaken using the *t* test or nonparametric tests (Wilcoxon rank-sum test) for continuous variables and Fisher exact or χ² tests for proportions as indicated. Paired observations were compared using the paired-to-paired Wilcoxon rank-sum test. Event-free survival was estimated according to CA end points by the Kaplan–Meier method using the log-rank test. Unadjusted Cox proportional hazards analyses assessed the relationship between background and procedural characteristics with respect to time to all-cause death. All baseline and procedural variables that were significantly (*P* <0.05) related to all-cause death were entered into a forward stepwise multivariable analysis. Differences were considered statistically significant at the 2-sided *P* <0.05 level. All analyses were undertaken using IBM SPSS statistics version 21 (IBM Corporation).

**Results**

A total of 74 VT CA procedures with ECMO support were performed in 64 out of 781 consecutive patients admitted at our unit from 2010 to 2015 for ablation of unstable, life-threatening, drug-refractory VTs. Fifty-nine out of 64 patients (92%) had a high-risk profile (Figure 1). The ECMO cohort includes patients with higher incidence of comorbidities, more advanced heart disease, presenting with a more complex arrhythmia pattern, and more frequently in cardiogenic shock, as compared with the overall VT series (Table 1).

**Study Cohort**

The baseline clinical characteristics of the study cohort (64 patients; age 63±15 years; 97% men) are shown in Table 1; 45% of patients had ischemic heart disease (23/29 had chronic occlusion of the infarct-related artery), 26% of patients had chronic obstructive pulmonary disease, and 67% of patients had chronic renal disease, mean left ventricular ejection fraction (LVEF) was 27±9%. Thirty-three patients (51% of the study series) were in New York Heart Association class III or IV. All patients had an implantable cardioverter defibrillator and a history of nontolerated VTs. Incessant VT was present on admission in 10 patients

![Figure 1](https://example.com/figure1.png)
Mechanical Circulatory Support

1. ECMO was used as a preemptive strategy in 59 out of 64 patients (92%).

1.1. In 14 patients (22%) who underwent hemodynamic stabilization of cardiogenic shock secondary to ES/ incessant VT in the ICU within a median time of 24 hours (5–48) before ablation;

1.2. In 31 patients (48%) because of nontolerated VTs induced at the electrophysiology study (Figure 2), in the setting of high-risk profile (incessant VT/ES in 42% of patients; respiratory disease in 25% of patients, renal disease in 29% of patients, LVEF <30% in 23% of patients, and ischemic heart disease with a known coronary vessel occlusion in 29% of patients).

1.3. In 14 patients (22%) because of failure of a previous unsuccessful substrate-based CA with persistent induction of unmappable nontolerated VTs (5 out of 14 at low risk);

2. ECMO was the bailout strategy in 5 out of 64 patients (8%) to overcome acute hemodynamic deterioration that developed intraprocedurally.

### Ablation Procedure

Overall, 74 procedures were required (ranging 1–3 per patient); a percutaneous epicardial approach was performed in 21 procedures (28%) and a surgical epicardial access was required in additional 6 procedures (8%).

One hundred and eighty-seven VTs (median 2 VTs/procedure; ranging 1–3 per patient) were induced in 68 procedures (92%). The MAP during VT was 75.8±11.8 mmHg with a target MAP >70 mmHg during VT being achieved with ECMO in 58 procedures (78% of inducible patients); MAP was between 60 and 70 mmHg in 14 procedures (19% of inducible patients); in the remaining 2 procedures (3%) with MAP <60 mmHg, adjunctive Impella 2.5 was implanted. Inotropes were administered during 63 out of 74 procedures (85%): epinephrine with a mean dosage of 0.1 µg/kg/min (0.03–0.3 µg/kg/min) and adjunctive norepinephrine in 35 procedures (47%) with a mean dosage of 0.06 µg/kg/min (0.05–0.1 µg/kg/min). A maximum ECMO flow of 1417.1±763.0 mL/min was provided. Minimum intraprocedural pH was 7.38±0.06. Intraprocedural electrophysiological variables are described in Table 2.

### Ablation Results

VT mapping and at least one termination of VT during CA was achieved in 48 out of 61 patients (79%) in 55 out of 68 procedures (81%) with inducible VT at baseline. Prevention of the inducibility of any VT was achieved in 51 procedures (69%). A previously documented VT remained persistently inducible after ablation in 5 (7%), nonpreviously documented VT in 8 (11%) and inducibility was not tested because of hemodynamic instability and long procedural times in 10 procedures (13%).

### Postoperative Course and Complications

ECMO was removed after a median time of 24.0 hours (24.0–48.0), and weaning was performed with the use of intra-aortic balloon pump after 7 procedures (9%). Median ICU stay was 3.0 days (2.0–5.0).

A transient postprocedural increase of median aspartate aminotransferases (79.0 U/L [49.0–114.0 U/L] from 29.0 U/L [20.0–45.5 U/L]; P=0.002) but not of median serum creatinine (1.1 mg/dL [1.0–1.3 mg/dL] preoperative versus 1.1 mg/dL...
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(1.0–1.4 mg/dL) postoperative; \( P = 0.252 \) was observed. Four patients (6%) experienced transient renal failure according to Acute Kidney Injury Network criteria (3 stage 1 and 1 stage 2). No changes in left and right ventricle systolic echocardiographic function were observed postprocedurally as compared with the preprocedural values (median LVEF: 25.0% [21.5–30.0] preoperative versus 25.0% [21.7–30.0] postoperative; \( P = 0.599 \) and median tricuspid annular plane systolic excursion: 20.0 mm [18.0–20.0 mm] preoperative versus 20.0 mm [18.0–21.0 mm] postoperative; \( P = 0.383 \); Table 3).

In the 5 patients in whom ECMO had been implemented as an intraprocedural bailout, faster VTs were observed (215±30 versus 293±61 ms, \( P = 0.005 \)), producing a lower MAP (67±4 versus 76±12 mm Hg; \( P = 0.005 \)), despite higher adrenaline dosage (0.19±0.06 versus 0.07±0.06 µg/kg/min; \( P = 0.012 \)), resulting into a lower intraprocedural minimum pH value (7.31±0.04 versus 7.39±0.06; \( P = 0.006 \)).

In 10 patients (16%) red blood cell and in 4 patients (6%) fresh frozen plasma transfusions were required. Acute peripheral artery ischemia occurred after the procedure in 2 patients, treated in one with Fogarty embolectomy and in the other with a reperfusion cannula positioning; both recovered uneventfully.

In-Hospital Outcome, Acute Heart Failure, and Mortality
Ten patients (16%) experienced in-hospital paroxysmal VT recurrence treated with drugs in 7 patients (11%) and with a second CA procedure in 3 patients (5%). Periprocedural acute heart failure occurred in 5 patients (8%), leading to death in 1 patient (1.5%) in the setting of subsequent mesenteric ischemia; 3 patients (5%) underwent emergency heart transplantation, and 1 patient (1.5%) received a permanent left ventricular assist device (LVAD). All other patients were discharged alive after a median hospitalization time of 18.5 days (14.0–31.2).

Follow-Up
After a mean follow-up of 23±13 months (6–72 months), 21 patients (33%) experienced VT recurrence requiring the reintroduction of amiodarone in 15 patients (23%), a second ECMO-supported CA in 5 patients (8%), and a third ECMO-supported CA in 1 patient (1.5%).

According to the inducibility of VT after CA, after 180 days of follow-up, VT recurrence was observed in 19% of patients with noninducible VT; 42% with nonclinical VT inducible; 75% with clinical VT inducible; 75% in whom acute outcome was not tested at the end of the procedure (\( P < 0.001 \); Figure 3).

Fifty-six patients were discharged alive. Overall mortality during follow-up was 8 patients (12%). Mortality was noncardiac in 4 patients (6%), related to end-stage heart failure in 3 patients (5%; no VT recurrences) and to VT in 1 patient (1.5%) after 4 months. Destination therapy for heart failure was required in 3 additional patients (5%), who underwent heart transplantation, and in 5 patients (8%), who required permanent LVAD implantation at 3±2 months.

Overall, LVAD was implanted in 6 patients (acutely in 1 patient and post discharge in 5 patients; 9%), and heart transplantation was performed in 6 patients (acutely in 3 patients and post discharge in 3 patients; 9%). Transplant and LVAD-free survival was 44 out of 64 (69%).

Figure 2. A, Electrophysiological study before implementing extracorporeal membrane oxygenation (ECMO) at ventricular tachycardia (VT) induction, mean arterial pressure drops under 50 mm Hg. B, VT mapping supported by ECMO: on ECMO support, mean arterial pressure rises to ≈100 mm Hg. C, Endocardial bipolar map of the left ventricle: basal anterior and anteroseptal scar (site of ablation).
All-cause death, heart transplantation, and LVAD implantation were less frequent in patients in whom VT was not inducible after ablation as compared with the patients in whom VT was inducible and with the untested patients (after 180 days of follow-up: 9% in patients with noninducible VT; 50% with inducible VT; and 75% in untested patients at the end of the ablation procedure; \( P < 0.001 \); Figure 4).

At unadjusted analysis, LVEF (hazard ratio, 0.891; \( P = 0.002 \)) and noninducibility at the end of the procedure (hazard ratio, 0.151; \( P < 0.001 \)) correlated with all-cause death, heart transplantation, and LVAD implantation. In the multivariable analyses, LVEF (hazard ratio, 0.916; \( P = 0.008 \)) and noninducibility (hazard ratio, 0.198; \( P = 0.001 \)) correlated with all-cause death, heart transplantation, and LVAD implantation (Table 4).

**Discussion**

In this study, we report the largest series of ECMO-supported CA of VT in high-risk patients. This initial experience supports the safety and efficacy of this modality of hemodynamic support to help patients overcome the unstable and clinically challenging preoperative and perioperative phase with a low incidence of postablation acute mortality (1.5%).

In 18.7% of these patients, ablation of unstable VTs refractory to antiarrhythmic drugs was critical to achieve rhythm stabilization, survive the acute phase, and allow the implementation of advanced heart failure treatment strategies (LVAD implantation and heart transplantation), whenever required.

On the contrary, the achievement of ablation acute success, in the long term, allowed clinical stabilization, through the prevention of all-cause death, LVAD implantation, and heart transplantation.

This goal seems to be particularly important in the higher-risk group, where failure to eliminate VTs is associated with

**Table 2. Procedural Data**

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Procedures (n=74)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT inducibility (baseline)</td>
<td>68 (91.9)</td>
</tr>
<tr>
<td>Fastest VT cycle, ms, mean±SD</td>
<td>289±60</td>
</tr>
<tr>
<td>Slowest VT cycle, ms, mean±SD</td>
<td>365±72</td>
</tr>
<tr>
<td>Number of induced VTs, sum</td>
<td>187</td>
</tr>
<tr>
<td>Number of induced VTs/procedure, median (Q1–Q3)</td>
<td>2.0 (1.0–3.0)</td>
</tr>
<tr>
<td>Number of terminated VTs, sum</td>
<td>126</td>
</tr>
<tr>
<td>No. of terminated VTs/procedure, median (Q1–Q3)</td>
<td>1.5 (1.0–2.25)</td>
</tr>
<tr>
<td>At least one VT termination during ablation, n (%)</td>
<td>55/68 (80.8)</td>
</tr>
<tr>
<td>Time on substrate mapping, min, median (Q1–Q3)</td>
<td>60.0 (35.0–82.5)</td>
</tr>
<tr>
<td>Time on activation mapping, min, median (Q1–Q3)</td>
<td>30.0 (14.75–51.0)</td>
</tr>
<tr>
<td>Ablation time, min, median</td>
<td>34.0 (20.7–57.5)</td>
</tr>
<tr>
<td>Ablation area, cm², median</td>
<td>25.0 (15.5–49.5)</td>
</tr>
<tr>
<td>Fluoroscopy time, min, mean (SD)</td>
<td>43.0 (30.0–53.0)</td>
</tr>
<tr>
<td>Overall procedural time, min, median (Q1–Q3)</td>
<td>300.0 (210.0–360.0)</td>
</tr>
<tr>
<td>Postablation inducibility, n (%)</td>
<td>13/64 (20.3)</td>
</tr>
<tr>
<td>PVS not performed</td>
<td>10 (13.5%)</td>
</tr>
<tr>
<td>VT noninducible</td>
<td>51 (68.9%)</td>
</tr>
<tr>
<td>VF inducible</td>
<td>9/51</td>
</tr>
<tr>
<td>Nondocumented VT inducible</td>
<td>8 (10.8%)</td>
</tr>
<tr>
<td>Previously documented VT inducible</td>
<td>5 (6.7%)</td>
</tr>
</tbody>
</table>

PVS indicates programmed ventricular stimulation; Q, quartile; and VT, ventricular tachycardia.

**Table 3. Hemodynamic Surrogates of End-Organ Perfusion, Postprocedure Organ Damage Indicators (Peak Within the First Week After the Ablation Procedure) and Postoperative Course**

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Procedures (n=74)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAP without ECMO (SR), mm Hg</td>
<td>83.9±11.2</td>
</tr>
<tr>
<td>MAP in ECMO (SR), mm Hg</td>
<td>86.9±9.8</td>
</tr>
<tr>
<td>MAP in ECMO (VT), mm Hg</td>
<td>75.8±11.8</td>
</tr>
<tr>
<td>Max ECMO flow, mL/min, mean±SD</td>
<td>4187.1±763.0</td>
</tr>
<tr>
<td>Intraprocedure arterial blood gases, minimum pH</td>
<td>7.38±0.06</td>
</tr>
<tr>
<td>Serum creatinine, mg/dL, median</td>
<td></td>
</tr>
<tr>
<td>Pre procedure</td>
<td>1.1 (1.0–1.3)</td>
</tr>
<tr>
<td>Post procedure</td>
<td>1.1 (1.0–1.4)</td>
</tr>
<tr>
<td>Aspartate aminotransferase, U/L, median (Q1–Q3)</td>
<td>29.0 (20.0–45.5)</td>
</tr>
<tr>
<td>Pre procedure</td>
<td>25.0 (21.5–30.0)</td>
</tr>
<tr>
<td>Post procedure</td>
<td>25.0 (21.7–30.0)</td>
</tr>
<tr>
<td>Left ventricular ejection fraction, %, median (Q1–Q3)</td>
<td>20.0 (18.0–20.0)</td>
</tr>
<tr>
<td>Pre procedure</td>
<td>20.0 (18.0–21.0)</td>
</tr>
<tr>
<td>Post procedure</td>
<td>20.0 (18.0–21.0)</td>
</tr>
<tr>
<td>Post procedure lactate, median (Q1–Q3)</td>
<td>4.4 (2.3–7.1)</td>
</tr>
<tr>
<td>Post procedure AKIN score, n (%)</td>
<td></td>
</tr>
<tr>
<td>Stage 1</td>
<td>3 (4.0)</td>
</tr>
<tr>
<td>Stage 2</td>
<td>1 (1.3)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>0</td>
</tr>
<tr>
<td>Time on ECMO, h, median (Q1–Q3)</td>
<td>24.0 (24.0–48.0)</td>
</tr>
<tr>
<td>ICU stay, d, median (Q1–Q3)</td>
<td>3.0 (2.0–5.0)</td>
</tr>
<tr>
<td>Hospital stay, d, median (Q1–Q3)</td>
<td>18.5 (14.0–31.2)</td>
</tr>
<tr>
<td>Postprocedural acute HF, n (%)</td>
<td>5 (7.8)</td>
</tr>
</tbody>
</table>

AKIN indicates Acute Kidney Injury Network; ECMO, extracorporeal membrane oxygenation; HF, heart failure; ICU, intensive care unit; MAP, mean arterial pressure; Q, quartile; TAPSE, tricuspid annular plane systolic excursion; VT, ventricular tachycardia.

*Comparison between pre- and postprocedure values (paired-to-paired Wilcoxon rank test).
increased cardiac mortality as compared with those in whom VT can be eliminated.1

Thus, the aim of an effective CA seems reasonable, provided that the risks of undertaking a complex and lengthy procedure are reduced by enforcing a high safety profile. From this perspective, our experience of high-risk CA supported by the ECMO compares favorably with other experiences where a systematic approach to hemodynamic support has not been undertaken or where hemodynamic support has been used only as an emergency strategy.3,4,13

Risk Stratification and Timing of Hemodynamic Support

Clinical presentations such as ES, increased duration and complexity of the procedure, advanced functional class, reduced ventricular function, and presence of comorbidities (renal failure, obstructive pulmonary disease) are associated with increased heart failure and cardiac mortality in the early stages after CA.1–4 This is related to prolonged times of reduced cardiac output and target organ hypoperfusion occurring before and during the procedure.

In the experience reported by Santangeli et al,4 the occurrence of acute decompensation during the procedure was extremely frequent in patients with the aforementioned clinical characteristics and was associated with excess mortality in the early postoperative period, at 6 months, and at 12 months. In this setting, general anesthesia seemed to play a further aggravating role. According to these authors, acute hemodynamic decompensation occurred most frequently in SR (63% of cases), during the substrate mapping phase, before induction of VT, requiring the institution of emergency hemodynamic support in 9 out of 22 patients and causing discontinuation of the procedure in 17 out of 22 patients.4 This finding may partially explain the lack of impact on the long-term outcome, with the use of hemodynamic support, observed in previous studies.10,12

This observation points to the need for counteracting prolonged low-output states, by providing earlier and sufficient hemodynamic support in high-risk patients. In this respect, the timing of institution of hemodynamic support seems to play a critical role. In our experience, the implementation of ECMO support on an emergency basis was related to a less favorable hemodynamic pattern, with faster VT rates and lower MAP values in spite of higher adrenaline doses, resulting in a greater extent of acidosis. It seems, therefore, indicated, when planning a supported procedure, to start the induction study when the ECMO is already operational. Furthermore, this may be even
more effective before general anesthesia and VT induction, to prevent the potential adverse consequences of hemodynamic deterioration. As suggested by the experience of others,4 and our own data,1 preoperative triage may be an important step to plan preemptive circulatory support and to prevent acute peri-procedural hemodynamic impairment.

There were no instances of procedure discontinuation in the present ECMO series, where a thorough ablation strategy, including both activation mapping and substrate modification, when indicated, could be accomplished. VT termination could be achieved in 80.8% of the inducible cases, achieving noninducibility of any VT in 68.9% of the procedures in the absence of hypoperfusion-related acidosis or significant procedural complications.

In keeping with the data from recent studies, a favorable long-term outcome was associated with the final acute result of noninducibility and successful modification of the arrhythmia substrate. Unlike other experiences,19 recurrent arrhythmic events during the hospital admission were not related to increased mortality and could be uniformly managed by anti-arrhythmic drugs and redo procedures.

Ablation Strategies for Unstable VTs

The advantages of a purely substrate-based approach to VT ablation among patients with stable VTs were recently demonstrated in a randomized trial.20 Comparable success rates with shorter procedural times and enhanced safety profiles were achieved by an extensive CA performed at sites of low voltage without attempting any VT induction. Late potentials abolition can be an effective end point of VT ablation and its prognostic value compares favorably with that achieved by postablation inducibility.5 Like other methods of substrate mapping, this technique overcomes the limitation of noninducibility or poor hemodynamic tolerance. Substrate mapping might be reasonably the first-line strategy in these compromised patients; however, one should be aware that it might not be uniformly feasible or successful and that a redo ablation with hemodynamic support might be required. On the contrary, the presence of late potentials itself may characterize a subgroup of patients with a more favorable prognosis after ablation.7,8 It should be realized that often, when a repeated ablation is required, the latter procedure happens to be the successful one, specifically when actions are undertaken to

Figure 4. Survival free from all-cause death, LVAD, and heart transplantation according to ventricular tachycardia (VT) inducibility after ablation. EPS indicates electrophysiological study.
overcome limitation of the first; however, in this setting, far different approaches were attempted. In this particular cohort, 14 patients underwent an ECMO-supported procedure after a failed substrate-guided ablation. In 5 of these first procedures, the goal of substrate mapping during SR had not been achieved because of the persistent mechanical induction of multiple pleomorphic nontolerated VTs, requiring multiple VT cardioversions that affected hemodynamic stability of patients. In an additional 5 cases, the extension of the scar was the reason for the incomplete abolition of the abnormal potentials in the remap; in the remaining 4, late or fragmented potentials were absent at all. That is why our second approach was scheduled to allow an adequate circulatory status during VT with the use of ECMO. Activation mapping during VT, in these cases, allowed the identification of diastolic activity and ablation successfully terminated arrhythmias, achieving noninducibility in 12 out of 14 patients (86%). ECMO support in patients with a previous failed substrate modification approach allowed a safe procedure leading to successful treatment of residual inducible recurring nontolerated arrhythmias.

**Alternative Strategies for Hemodynamic Support During VT Ablation**

No data are available to select the best strategy for hemodynamic support during VT ablation. Previous reports have focused on the use of different devices. Intra-aortic balloon pump assistance has substantial limitations during VT, related to the need for synchronization with the cardiac cycle, unsuitable during fast ventricular arrhythmias especially in patients with severely reduced left ventricular function. The Impella 2.5 and Tandem Heart devices are superior to intra-aortic balloon pump for the support of unstable VTs, allowing longer activation mapping times and VT termination rates. Tandem Heart-assisted VT CA guided by activation and entrainment mapping may be a feasible alternative to substrate mapping allowing comparable outcomes.

These devices have some limitations in the setting of VT CA in presence of mechanical aortic valves, LV thrombosis, and ventricular septal defects. They might also be affected by electromagnetic interferences (Impella 2.5) and may increase the difficulties of a concomitant transseptal approach for mapping and ablation (Tandem Heart). Furthermore, they neither provide right ventricular assistance nor respiratory support.

In agreement with the experimental work by Ostadal et al, ECMO proved uniformly effective in providing a cardiac output to tolerate any induced arrhythmia, without impairing the possibility of multiple accesses to the left ventricle, allowing extensive endo-epicardial mapping free from electromagnetic interferences. The increased complexity of the ECMO setup (need for a Perfusionist) is largely rewarded by a superior method of hemodynamic support and by improved safety during the procedure.

**Study Limitations**

The main limitation of the study is the lack of comparative data, as other support systems were not used in the laboratory because of the evidence provided.

The present article reports a single-center experience in a small cohort of consecutive patients; results should be confirmed in larger multicenter randomized studies.

**Conclusions**

After a specific VT-related triage, the implementation of ECMO hemodynamic support for VT ablation in high-risk patients is a safe strategy and allows prolonged times of VT activation mapping, leading to the achievement of successful VT ablation. The major advantage offered by the system is the extended intra- and periprocedural circulatory support that allows lengthy and complex ablation procedures to be completed with a low incidence of heart decompensation and acute death.

**Acknowledgments**

We would like to thank the Anesthesia and Intensive Care Unit staff and the Perfusionists Otello Turla and Elisabeta Fumagalli for their continuous support. We also would like to acknowledge the statistical contribution by Rosalba Lembo and Giovanni Affronti.

**Disclosures**

None.

**References**


### Table 4. Unadjusted and Multivariable Cox Regression Analysis of All-Cause Death, Heart Transplant, and LVAD

<table>
<thead>
<tr>
<th>Predictor</th>
<th>HR (95% CIs)</th>
<th>P Value</th>
<th>HR (95% CIs)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Noninducibility</td>
<td>0.151 (0.059–0.388)</td>
<td>&lt;0.001</td>
<td>0.198 (0.074–0.524)</td>
<td>0.001</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>0.891 (0.827–0.959)</td>
<td>0.002</td>
<td>0.916 (0.853–0.984)</td>
<td>0.008</td>
</tr>
<tr>
<td>Renal disease</td>
<td>2.186 (0.907–5.266)</td>
<td>0.081</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>2.139 (1.138–4.021)</td>
<td>0.068</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of VTs in the procedure</td>
<td>1.263 (0.879–1.813)</td>
<td>0.206</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Electrical storm</td>
<td>0.867 (0.333–2.257)</td>
<td>0.770</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CI indicates confidence interval; HR, hazard ratio; LVEF, left ventricular ejection fraction; and VT, ventricular tachycardia.


Extracorporeal Membrane Oxygenation for Hemodynamic Support of Ventricular Tachycardia Ablation

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