


# Outcomes of pre-emptive and rescue use of percutaneous left ventricular assist device in patients with structural heart disease undergoing catheter ablation of ventricular tachycardia

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## Abstract

**Purpose** Patient selection and timing of percutaneous left ventricular assist device (pLVAD) insertion for maximal benefit during ventricular tachycardia (VT) ablation is not well defined. We aimed to assess the outcomes of pre-emptive and rescue use of pLVAD during VT ablation in patients with ischemic and non-ischemic cardiomyopathy.

**Methods** Between January 2009 and October 2011, 93 patients underwent VT ablation. Three groups were compared: (1) Rescue group ( $n = 12$ )—patients who required emergent pLVAD insertion due to hemodynamic collapse during VT ablation, (2) Pre-emptive group ( $n = 24$ )—patients who had pre-ablation pLVAD insertion, and (3) Non-pLVAD group ( $n = 57$ )—patients who did not undergo pLVAD insertion. Procedural outcomes including 30-day mortality were compared.

**Results** Thirty-day mortality was higher in the Rescue group compared to the Pre-emptive group (58 vs. 4 %,  $p = 0.003$ ) and non-pLVAD (58 vs. 3 %,  $p = 0.001$ ) group. There was no significant difference in 30-day mortality or long-term freedom of VT between the pre-emptive and non-pLVAD groups.

**Conclusions** Despite rescue pLVAD insertion, hemodynamic collapse during VT ablation is associated with a persistently high 30-day mortality. Further studies are warranted to predict hemodynamic collapse and to refine the role of pLVAD in this setting.

**Keywords** Percutaneous left ventricular assist device · Ventricular tachycardia · Ablation · Cardiomyopathy

## Abbreviations

CABG	Coronary artery bypass grafting
ECMO	Extra corporeal mechanical oxygenation
LAP	Left atrial pressure
LVEDD	Left ventricular end diastolic dimension
LVEDP	Left ventricular end diastolic pressure
LVEF	Left ventricular ejection fraction
MAP	Mean arterial pressure
NYHA	New York Heart Association
VT	Ventricular tachycardia
pLVAD	Percutaneous left ventricular assist device

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## 1 Introduction

Catheter ablation of scar-related ventricular tachycardia (VT) has shown to be an effective therapy in patients with ischemic and non-ischemic cardiomyopathy [1, 2]. Although activation and entrainment mapping during VT can facilitate successful ablation, such strategies are limited as only 30 % of patients can hemodynamically tolerate VT [3]. Even with a substrate modification approach, patients remain vulnerable to hemodynamic compromise and/or mortality [4].

The use of percutaneous left ventricular assist devices (pLVAD) has been shown to extend the time span patients can remain in VT without affecting cerebral perfusion and other end organ damage [5]. Aside from allowing support to map VT, often pLVAD insertion is performed after development of hemodynamic collapse in an attempt to “rescue” patients from ongoing clinical deterioration. The question remains, however, whether pLVAD insertion in this scenario alters either short- or long-term outcomes. Our objective was to assess the outcomes of rescue use of pLVAD in patients with hemodynamic collapse during VT ablation.

## 2 Methods

### 2.1 Patient selection

From January 2009 to October 2011, 93 patients with LVEF  $\leq 40\%$  underwent VT ablation due to recurrent, drug refractory VT. All patients gave informed written consent.

Twelve patients (13 %) developed hemodynamic collapse—defined as pulseless electrical activity, refractory ventricular fibrillation/tachycardia, persistent hypotension (mean arterial pressure, MAP  $< 50$  mmHg) despite vasopressor(s), and/or acute pulmonary edema—during the procedure and underwent emergent pLVAD insertion (rescue pLVAD group).

A pre-emptive pLVAD group ( $n = 24$ ) underwent pLVAD insertion prior to ablation per the operator’s discretion.

A non-pLVAD group ( $n = 57$ ) was identified without the use of pLVAD during ablation. Among them, eight had intra-aortic balloon pump support placed at the start of the procedure at the operator’s discretion.

### 2.2 Electrophysiology study and ablation

General anesthesia was used in the majority ( $n = 64$ ) of cases. Trans-septal catheterization was performed in 88 out of the 93 cases (95 %). Epicardial access, by method described previously [6], was performed in six patients.

Heparin infusion was initiated after access to the left ventricle (LV) with a goal to maintain activated clotting time of 250–350 s.

In all patients, programmed electrical stimulation was performed using standard ventricular extrastimuli protocol to assess for initial inducibility and identification of a targeted VT for ablation. None of the patients were in VT at the beginning of the procedure. Activation mapping was performed if a VT was tolerated (MAP  $\geq 50$  mmHg). If VT could not be induced or tolerated, an electroanatomic map was generated (CARTO, Biosense Webster, Inc. or EnSite, St. Jude Medical) to identify regions of scar where tissue voltage was  $\leq 1.5$  mV. Catheter ablation was performed via an externally irrigated 3.5 mm ablation catheter (Navistar Thermocool, Biosense Webster,

Inc.). The strategy of a substrate-based approach for ablation included ablation of late potentials within scar, sites with pacemaps of induced or clinical VTs within scar, and borderzones of scar that were presumed exit sites of VT. After ablation, programmed electrical stimulation was performed to attempt re-induction of VT if the patient was hemodynamically stable.

In all patients, hemodynamic parameters (after induction of anesthesia) of baseline left atrial pressure (bLAP)  $\geq 20$  mmHg, left atrial pressure  $\geq 20$  mmHg with RV pacing at cycle length 600 ms or longer (pLAP), or a mean arterial pressure drop to less than 50 mmHg with RV pacing were assessed. Further, total procedural time, total time in VT, termination of VT during ablation, and VT requiring cardioversion and/or pace termination were also recorded.

### 2.3 Percutaneous left ventricular assist device implantation

Patients with pLVAD underwent insertion of either Impella<sup>TM</sup> (Abiomed, Inc., Danvers, MA) or TandemHeart<sup>TM</sup> (CardiacAssist, Inc., Pittsburgh, PA). Insertion of Impella<sup>TM</sup> was performed via a 13F femoral arterial sheath [7], and performance was set to a maximum level (2.0–2.5 L/min) throughout the procedure [Fig. 1a]. Insertion of TandemHeart<sup>TM</sup> was performed via a 21F trans-septal inflow sheath and either a 15F or 17F sheath in the femoral artery [8] [Fig. 1b].

After Impella<sup>TM</sup> removal, hemostasis was achieved via manual pressure to the femoral artery. For TandemHeart<sup>TM</sup> removal, a per-close arteriotomy technique was employed (PerClose ProGlide, Abbott Laboratories, Abbott Park, IL). Surgical closure was performed if any concern for vascular compromise existed.

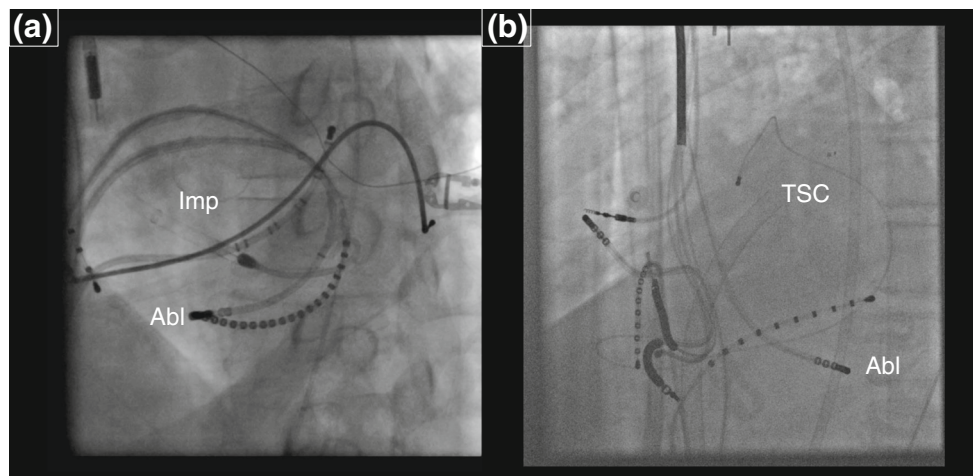
### 2.4 Statistical analysis

Data are expressed as the mean value  $\pm$  standard deviation (SD), unless otherwise indicated. For categorical variables, inter-group comparisons were performed using the chi-square test. The Fisher exact test was used if the expected cell count for a  $2 \times 2$  table was  $< 5$ . The two-tailed unpaired Student’s *t* test was used to analyze continuous variables, and the Mann–Whitney *U* test was used if the assumption of normality was not met. A *p* value  $< 0.05$  was considered statistically significant. Statistical analyses were performed using SAS software (SAS Inc., Cary, NC).

## 3 Results

A total of 93 patients underwent VT ablation. Twelve patients (13 %) underwent rescue pLVAD insertion, while 24 patients (26 %) underwent pre-emptive pLVAD insertion, and 57 patients

**Fig. 1** Insertion of percutaneous left ventricular assist device. Insertion of **a** Impella and **b** TandemHeart pLVAD during VT Ablation. *Abl*—Ablation catheter, *Imp*—Impella across aortic valve, *TSC*—trans-septal cannula of TandemHeart



(61 %) did not undergo pLVAD implant. In the overall cohort, average age was  $66 \pm 6$  years, 14 % were female, and LVEF was  $26.6 \pm 9$  %. The underlying substrate was ischemic cardiomyopathy in 51/93 (55 %) patients and 67/93 (72 %) were in NYHA heart failure Class II/III and 23/93 (25 %) in NYHA Class IV status. Two or more anti-arrhythmic drugs were used in 34/93 (37 %) patients and 80/93 (86 %) were on amiodarone.

### 3.1 Comparison between rescue pLVAD and non-pLVAD groups

#### 3.1.1 Patient characteristics

The baseline characteristics between the rescue pLVAD and non-pLVAD groups are shown in Table 1. There were no differences between the groups with regard to age, sex, LVEF, ischemic cardiomyopathy, left ventricular end-diastolic dimension (LVEDD), NYHA status, pre-procedure BNP levels, diabetes mellitus, hypertension, and renal insufficiency. All patients received ICD shocks except 3 in the non-pLVAD group, and there was no difference in the incidence of VT storm between the two groups.

Ninety-two percent of all patients were on beta-blocker therapy. Patients with rescue pLVAD insertion were more often on multiple anti-arrhythmic drugs compared to the non-pLVAD group (9/12 vs 14/57 patients,  $p = 0.002$ ).

Finally, PAINESD score to predict acute hemodynamic collapse during VT ablation as described by Santangeli et al. [9] was also calculated. Patients in the rescue pLVAD group had a significantly higher score ( $17.8$  vs  $13.4$ ,  $p = 0.01$ ) when compared to the non-pLVAD group.

#### 3.1.2 Procedural and ablation outcomes

Outcome data between the non-pLVAD and rescue pLVAD groups are presented in Table 2. Procedure times, which included pLVAD insertion, were longer in patients with rescue

pLVAD compared to the non-pLVAD group ( $226 \pm 88$  vs  $182 \pm 77$  min,  $p = 0.01$ ). The non-pLVAD group spent less time in VT when compared to the rescue pLVAD group ( $9.3 \pm 8.5$  vs  $23.0 \pm 28.9$  min,  $p = 0.03$ ). Five patients in the non-pLVAD group underwent epicardial mapping while no patients underwent epicardial mapping in the rescue pLVAD group.

Hemodynamic parameters were also assessed between the groups. There was no difference in elevated baseline left atrial pressure or RV pacing-related left atrial pressure (pLAP); however, a higher percentage of patients in the rescue pLVAD group developed a mean arterial pressure  $<50$  mmHg with RV pacing (16/57 vs 9/12,  $p = 0.006$ ).

At the end of the procedure, there was no difference in inducibility of VT in those patients that were assessed at the end of the procedure (4/8 in the rescue group vs. 29/44 in the non-pLVAD group,  $p = 0.43$ ). However, there was significantly increased 30-day all-cause mortality in the rescue pLVAD group (7/12 or 58.3 %) compared to the non-pLVAD group (2/57 or 3.5 %,  $p = 0.001$ ). There was no significant difference in the freedom from VT at 3-month follow-up in surviving patients [31/55 pts (56 %) in the non-pLVAD group and 3/5 pts (60 %) in the rescue group,  $p = 0.34$ ].

### 3.2 Comparison of rescue pLVAD and pre-emptive pLVAD groups

Twenty-four patients underwent pre-emptive pLVAD insertion at the operator's discretion prior to ablation. Outcomes were compared to the rescue pLVAD group.

#### 3.2.1 Patient characteristics

The baseline characteristics between the rescue pLVAD and pre-emptive pLVAD groups are shown in Table 1. There were no statistical differences between the groups with regard to age, sex, LVEF, LVEDD, NYHA status, pre-procedure BNP

**Table 1** Baseline characteristics

Characteristics	Non-pLVAD (N = 57)	Rescue pLVAD (N = 12)	<i>p</i> value <sup>a</sup>	Pre-emptive pLVAD (N = 24)	<i>p</i> value <sup>b</sup>	<i>p</i> value <sup>c</sup>
Age (years, mean ± SD)	64.8 ± 9	68.8 ± 8	0.34	65.8 ± 14	0.47	0.40
Male	47/57 (82 %)	12/12 (100 %)	0.30	21/24 (88 %)	0.54	0.75
HTN	49/57 (86 %)	10/12 (83 %)	0.24	19/24 (79 %)	0.30	0.51
DM	33/57 (58 %)	8/12 (67 %)	0.11	17/24 (71 %)	0.80	0.32
Renal insufficiency	22/57 (39 %)	5/12 (42 %)	0.80	8/24 (33 %)	0.64	0.80
COPD	14/57 (25 %)	5/12 (42 %)	0.29	9/24 (38 %)	1.00	0.28
Ischemic CMP	32/57 (56 %)	7/12 (58 %)	0.70	12/24 (50 %)	0.65	0.63
Prior CABG	12/57 (21 %)	3/12 (25 %)	0.50	5/24 (21 %)	0.79	1.00
NYHA Class I	2/57 (4 %)	1/12 (8 %)	0.44	0/24 (0 %)	0.33	1.00
NYHA Class II- III	42/57 (73 %)	8/12 (67 %)	0.30	17/24 (71 %)	0.81	0.79
NYHA Class IV	13/57 (23 %)	3/12 (25 %)	0.29	7/24 (29 %)	0.78	0.58
LVEF (%) (mean ± SD)	28 ± 5	24 ± 14	0.20	26 ± 9	0.67	0.77
LVEDD (cm)	6.3 ± 0.6	6.2 ± 1.0	0.66	6.7 ± 0.9	0.15	0.19
Prior ICD shock	54/57 (95 %)	12/12	1.00	24/24	1.00	0.98
VT storm	9/57 (16 %)	4/12 (33 %)	0.21	7/24 (29 %)	0.81	0.17
Beta-blocker	52/57 (92 %)	11/12 (92 %)	1.00	22/24 (92 %)	1.00	1.00
2+ AAD	14/57 (25 %)	9/12 (75 %)	0.002	11/24 (46 %)	0.16	0.06
Amiodarone	47/57 (82 %)	10/12 (83 %)	0.45	23/24 (96 %)	0.31	0.73
BNP (baseline)	975.6 ± 835	1072.3 ± 944	0.19	1154.1 ± 1422	0.82	0.37
PAINESD score	13.4 ± 5.4	17.8 ± 3.8	0.01	16.5 ± 5.4	0.47	0.02

PAINESD—risk score for acute hemodynamic collapse [9]: *P*—pulmonary disease, *A*—age, *I*—ischemic cardiomyopathy, *N*—NYHA Class III/IV, *E*—ejection fraction <25 %, *S*—VT storm presentation, *D*—diabetes

*HTN* hypertension, *DM* diabetes mellitus, *Renal insufficiency* creatinine >1.5 mg/dL, *COPD* chronic obstructive pulmonary disease, *LVEF* left ventricular ejection fraction, *CMP* cardiomyopathy, *CABG* coronary bypass surgery, *NYHA* New York Heart Association functional class, *AAD* anti-arrhythmic drug, *BNP* brain natriuretic peptide, *LVEDD* left ventricular end-diastolic diameter

<sup>a</sup> Comparison between Non-pLVAD and Rescue Groups

<sup>b</sup> Comparison between Rescue and Pre-emptive Groups

<sup>c</sup> Comparison between Non-pLVAD and Pre-emptive Groups

levels, number of anti-arrhythmic drugs, diabetes mellitus, hypertension, renal insufficiency, and ischemic cardiomyopathy. All patients received at least one ICD shock and there was no statistical difference in VT storm among the groups. Finally, there was no difference in the PAINESD score between the pre-emptive group and rescue pLVAD group (17.8 vs 16.5,  $p = 0.47$ )

### 3.2.2 Procedural and ablation outcomes

Outcomes of the pre-emptive and rescue pLVAD groups are presented in Table 2. There were no differences in procedure times or time in VT. There was a higher number of VT terminated during ablation in the pre-emptive pLVAD (median 2) group compared to the rescue pLVAD group (median 1).

Hemodynamic parameters among these groups were matched without any significant differences regarding bLAP, pLAP, or MAP drop with RV pacing.

At the end of the procedure, there was no significant difference in inducibility of VT in those assessed at the end of the procedure (rescue pLVAD 4/8 vs. pre-emptive group 11/18

patients,  $p = 0.62$ ). There was no difference in freedom from VT at 3-month follow-up between the surviving patients in the two groups (pre-emptive pLVAD 17/23 or 74 % vs. rescue pLVAD 3/5 or 60 % patients,  $p = 0.20$ ). Despite the similar baseline characteristics, there was an increased 30-day all-cause mortality in the rescue pLVAD group (58.3 %) when compared to the pre-emptive pLVAD group (4.2 %,  $p = 0.003$ ).

### 3.3 Comparison of non-pLVAD and pre-emptive pLVAD groups

Baseline characteristics between the two groups are compared in Table 1. Of note, there were no significant differences in any of the measured variables. Although not reaching statistical significance, there was a strong trend of a higher number of patients in the pre-emptive group on two or more anti-arrhythmic drugs. Finally, the PAINESD score was significantly higher in the pre-emptive group (16.5 ± 5.4 vs 13.4 ± 5.4,  $p = 0.02$ ).



**Table 2** Procedural characteristics and follow-up

Characteristics	Non-pLVAD (N = 57)	Rescue pLVAD (N = 12)	p value <sup>a</sup>	Pre-emptive pLVAD (N = 24)	p value <sup>b</sup>	p value <sup>c</sup>
General anesthesia	37/57 (65 %)	10/12 (83 %)	0.31	17/24 (71 %)	0.69	0.61
bLAP ≥20 mmHg	15 (28 %)	3 (25 %)	0.93	12 (50 %)	0.28	0.27
pLAP ≥20 mmHg	14 (25 %)	6 (50 %)	0.09	6 (25 %)	0.10	0.97
MAP <50 mmHg with RV pacing	16 (28 %)	9 (75 %)	0.006	13 (54 %)	0.29	0.03
Procedure time (min)	182 ± 77	226 ± 88	0.01	254 ± 102	0.57	0.007
Time in VT (min)	9.3 ± 8.5	23.0 ± 28.9	0.03	42.5 ± 36.1	0.35	0.02
No. of VT	2.1 ± 1.4	3.2 ± 1.6	0.44	2.8 ± 1.8	0.49	0.47
IABP	8 (14 %)	2 (17 %)	0.82	n/a		
Epicardial access	5/57 (9 %)	0/12 (0 %)	0.02	1/24 (4 %)	NS	0.48
VT cycle length (ms)	376 ± 12	393 ± 18	0.48	365 ± 22	0.23	0.44
RF term of VT (median)	0 (0–2)	1.0 (0–2)	0.04	2.0 (0–4)	0.03	0.01
CV/pace term of VT (median)	2 (0–4)	2 (0–2)	0.27	2.0 (0–3)	0.53	0.48
Non-inducible post- procedure	29/44 (66 %)	4/8 (50 %)	0.43	11/18 (61 %)	0.62	0.11
30-day mortality	2/57 (3.5 %)	7/12 (58.3 %)	0.001	1/24 (4 %)	0.003	0.94
3-month follow-up freedom from VT	31/55 (56 %)	3/5 (60 %)	0.34	17/23 (74 %)	0.20	0.23

bLAP baseline left atrial pressure, pLAP left atrial pressure with RV pacing, MAP mean arterial pressure, IABP intra-aortic balloon pump, RF radiofrequency ablation, CV/pace Term of VT VT termination with cardioversion or overdrive pacing

<sup>a</sup> Comparison between control and rescue groups

<sup>b</sup> Comparison between rescue and pre-emptive groups

<sup>c</sup> Comparison between non-pLVAD and pre-emptive groups

There were no significant differences in baseline and pacing-related LAP; however, a higher number of patients in the pre-emptive group had a drop in MAP with RV pacing compared to the non-pLVAD group (16/57 vs 13/24,  $p = 0.03$ )

Of note, there were no statistical differences between the pre-emptive pLVAD and non-pLVAD group with regard to 3-month freedom from VT (17/23 vs 31/55,  $p = 0.15$ ) and 30-day mortality (1/24 vs 2/57,  $p = 0.89$ ).

### 3.4 Impella™ vs TandemHeart™ insertion

Patient characteristics between both pLVAD types are shown in Table 3. In the rescue pLVAD group, 9/12 (75 %) patients underwent TandemHeart™ insertion, while the pre-emptive group had 6/24 (25 %) patients undergo TandemHeart™ insertion. There were no statistical differences between either groups with regard to gender, history of prior CABG, NYHA functional status, BNP, and incidences of hypertension, diabetes, mellitus, renal insufficiency, ischemic cardiomyopathy.

### 3.5 Complications

All patients that underwent rescue pLVAD insertion had evidence of hemodynamic collapse during the procedure. Nine of 12 patients in the rescue pLVAD group developed

cardiogenic shock as manifested by hypotension and/or pulmonary edema. Two patients in the rescue group had a cardiac arrest during the procedure (one pulseless electrical activity, one refractory ventricular fibrillation). One patient in the rescue group developed a pericardial effusion warranting a pericardiocentesis, and pLVAD was inserted for hemodynamic support due to refractory hypotension after pericardiocentesis. There were no procedural deaths in the rescue pLVAD group (defined as death that occurred during the procedure or <72 h afterwards); however, the 30-day all-cause mortality was 58.3 % in the rescue pLVAD group. Five out of seven patients died due to refractory heart failure post-procedure despite the use of pLVAD, one patient developed an intracerebral hemorrhage, and one patient died from sepsis.

In the pre-emptive pLVAD group, one patient developed a right external iliac dissection during coronary angiography warranting a peripheral intervention. Another patient in the pre-emptive pLVAD group developed pulseless electrical activity during the procedure and was upgraded to ECMO support. This patient survived the procedure with removal of all hemodynamic support. One patient died in the pre-emptive pLVAD group within 30 days due to refractory heart failure 20 days after ablation.

In the non-pLVAD group, there was one cardiac perforation managed with pericardiocentesis, and one retroperitoneal

**Table 3** Comparison of Impella™ and Tandem Heart™ patient characteristics

	Impella (N=21)	Tandem Heart (N=15)	p value
Age	63.6±14	70.3±7	0.08
Male	18 (86 %)	15 (100 %)	0.94
HTN	17 (81 %)	12 (80 %)	0.95
DM	14 (67 %)	11 (73 %)	0.68
Renal insufficiency	8 (38 %)	5 (33 %)	0.24
LVEF (%)	29.0±12.5	21.5±8.1	0.06
Ischemic CMP	11 (52 %)	10 (67 %)	0.40
Prior CABG	4 (19 %)	4 (27 %)	0.60
NYHA Class I	0 (0 %)	1 (7 %)	NS
NYHA Class II-III	14 (67 %)	11 (73 %)	0.68
NYHA Class IV	7 (33 %)	3 (20 %)	0.38
BNP	1380.7±2022	1182.9±1590	0.82
Insertion (days)	0.24±0.5	3.8±2.3	<0.001

Abbreviations as Table 1

hematoma managed conservatively with blood transfusion. Two patients died within 30 days of the procedure and both were related to refractory heart failure (>21 days post ablation).

One patient after epicardial access developed a left pleural effusion, which was conservatively managed without surgical intervention.

#### 4 Discussion

The major finding of this study is that patients undergoing VT ablation who develop hemodynamic collapse continue to have a high 30-day mortality despite the emergent insertion of a pLVAD. To our knowledge, this is the first study to specifically assess the outcomes of pLVAD insertion after development of acute hemodynamic collapse during VT ablation.

Percutaneous LVAD support has been used safely and effectively in high risk coronary intervention procedures [8, 10–12] and in patients with cardiogenic shock [13]. In the context of VT ablation, initial case reports indicated pLVAD could be used safely in patients with hemodynamically unstable ventricular tachycardia [14–16]. A report by Carbucicchio et al. [17] described the role of percutaneous cardiopulmonary support prior to VT ablation in 19 patients with hemodynamic failure and/or electrical storm. Despite the high risk patient population, there were no peri-procedural deaths and 67 % of patients had a reduction in VT burden with a 21 % mortality during a mean follow-up of 42 months [17]. Miller et al. have shown that pLVAD insertion allows for a longer time in VT, which leads to a greater number of VT termination during ablation in 22 patients with cardiomyopathy [5]. When

compared to patients without pLVAD, however, there were no differences in post-procedure inducibility of VT and patients had a similar VT reoccurrence rate at 3-month follow-up [5]. Furthermore, Bunch et al. [18] compared 13 patients with a pLVAD-assisted activation mapping approach versus 18 patients with a conventional substrate-based approach and found similar outcomes both regarding inducibility and VT recurrence at 9-month follow-up [18]. Finally, Reddy et al. [19] reported a multicenter experience in patients undergoing pLVAD insertion for VT ablation. Sixty-six patients underwent either intra-aortic balloon pump, Impella™, or TandemHeart™ insertion. Similar to prior reports, patients with in the non-IABP group had greater time mapping in VT with higher number of VT ablated. Despite these advantages, mortality and VT recurrence were not improved compared to the IABP group over a 12-month follow-up period. Consistent with these reports, patients in our cohort with a pre-emptive pLVAD insertion were able to tolerate VT longer which led to more termination of VT when compared to patients without pLVAD support, yet there was no significant difference in the freedom from VT at 3-month follow-up.

All the above data were in patients who had pLVAD placed at the onset of a VT ablation procedure per operator's discretion (pre-emptively). In clinical practice, however, many times pLVAD insertion is reserved for patients that develop acute hemodynamic collapse during ablation. The hope would be that immediate improvement in cardiac output would reverse the acute hemodynamic collapse. Our data indicate that the use of pLVAD in “rescuing” patients from severe hemodynamic compromise during VT ablation is still associated with poor outcomes even though it could provide increased cardiac output immediately upon insertion. A similar observation with the use of pLVAD in patients presenting with cardiogenic shock were made by Kar et al. [13]. Despite successful implantation of pLVAD in all patients with improved hemodynamic parameters, 40.2 % of patients in this series died within 30 days [13]. Of note, the majority (70 %) of the death was due to refractory heart failure that persisted after insertion of pLVAD despite hemodynamic improvement.

Ideally, if patient-specific variables could be identified to not only predict hemodynamic collapse, but also to predict which patients may benefit from pre-emptive pLVAD insertion, this could reduce peri-procedural morbidity and/or mortality. Recently, Santangeli et al. [9] reported on the incidence and predictors of acute hemodynamic collapse in a series of 193 patients undergoing VT ablation. In this series, 11 % patients developed hemodynamic collapse with 41 % undergoing mechanical support (IABP or pLVAD). Despite these interventions, there was

an increased 30-day, 6-month, and 1-year mortality [9]. The elevated mortality in the “rescue” group in our series is consistent with these findings. Predictors of hemodynamic collapse in this series included advanced age, higher NYHA status, ischemic cardiomyopathy, COPD, VT storm, and use of general anesthesia (PAINESD score). When excluding general anesthesia, a score between 9 and 14 points was associated with a 6 % risk of acute hemodynamic collapse, while that risk increased to 24 % in patients with a score  $\geq 15$ . Although the individual variables in our series did not specifically meet statistical significance between groups, patients in the rescue and pre-emptive pLVAD groups had higher PAINESD scores (mean 17.8 and 16.5, respectively) compared to the non-pLVAD group (mean 13.4). Further, despite similar PAINESD scores between the rescue and pre-emptive groups, there was a marked difference in mortality. This implies that this scoring system may provide operators not only a prediction regarding hemodynamic collapse, but perhaps could be used to determine if pre-emptive pLVAD may be of benefit for a particular patient. Further prospective studies are warranted to further assess this scoring system with regard to pre-emptive pLVAD insertion. Finally, the rescue group had longer procedure times and was on more anti-arrhythmic drugs compared to the non-pLVAD group. Both of these variables have been associated with an increased mortality [2, 20]. In addition to the above studied variables, in our cohort there was a trend towards a higher LVEDD in the pre-emptive group as well as a tendency for TandemHeart™ placement in patients with lower EF with longer insertion times. Future studies that assess patients with specific LV dimensions and/or EF may further identify which patients may benefit from pre-emptive pLVAD insertion.

Specific hemodynamic parameters were assessed under anesthesia in our patient population. In particular, both baseline and/or RV pacing-related left atrial pressure of  $\geq 20$  mmHg as well as a mean arterial pressure drop  $< 50$  mmHg with RV pacing was recorded. Interestingly, a statistically significant higher number of patients had a drop in MAP of  $< 50$  mmHg with RV pacing (cycle length 600ms or longer) in the rescue and pre-emptive pLVAD groups when compared to the non-pLVAD group. RV pacing can simulate how a patient may tolerate a slow VT and/or the drop in MAP with pacing may reflect unrecognized RV dysfunction. Future studies to further understand the implications of the drop in MAP with pacing in this patient population are warranted. Perhaps continuous pressure monitoring may help elucidate whether it is RV dysfunction (with associated rise in CVP) or LV dysfunction (LA pressure rise) or some other mechanism that contributes to this specific pacing response. Although retrospective data with a small number of patients, this parameter may provide a unique variable in predicting which patients may develop

hemodynamic collapse as well as who may benefit from pre-emptive pLVAD insertion.

The overall 30-day mortality in our cohort of 93 patients was 7.5 % and was primarily driven by the rescue pLVAD group. The 30-day mortality in the rescue pLVAD group was 58 % while only 4 % in the pre-emptive pLVAD group despite similar baseline clinical characteristics between the two groups prior to ablation. Prior multicenter ablation trials have noted procedural-related death from  $< 1$  to 3 % with 1-year mortality ranging from 15 to 25 % [1, 2, 21]. Nearly 25 % of all patients in our series had NYHA Class IV heart failure (29 % in the pre-emptive pLVAD group) prior to ablation. In comparison, Miller et al. [5] reported 39 % ( $n = 9$ ) of patients with NYHA Class III or IV symptoms, and Bunch et al. [18] had an average NYHA Class of 2.7 ( $n = 13$ ) in their series. Only one peri-procedural death was reported by Bunch et al. and none by Miller et al. All patients in these two reports had undergone pLVAD insertion pre-emptively prior to ablation. This may suggest that pre-emptive pLVAD insertion may be of benefit in certain high risk patient populations, regardless of the ablation strategy.

#### 4.1 Limitations

There are several limitations to our study that need to be acknowledged. There was no randomization of the groups, which could have introduced operator bias not only for pLVAD insertion but also the type (Impella vs TandemHeart) of device. Despite this limitation, there were no significant baseline differences between the rescue pLVAD and pre-emptive pLVAD groups. Also, there is no comparison of outcomes of patients with hemodynamic collapse who did not undergo pLVAD. Further, we did not include any measurement of end-organ perfusion such as cerebral oximetry. Patients in the rescue group were in VT longer during the procedure compared to control patients. Perhaps, cerebral oximetry would have provided earlier insight regarding hemodynamic deterioration. Further, all patients underwent insertion of Impella 2.5, while the Impella CP or 5.0 allow for a higher level of support. The use of these newer devices might have resulted in different outcomes. We also did not factor in the right ventricular (RV) function in the outcome assessment.

#### 5 Conclusions

The role of rescue pLVAD insertion for acute hemodynamic collapse during VT ablation in patients with cardiomyopathy is limited as it continues to be associated with a high 30-day mortality. Pre-emptive pLVAD insertion may benefit certain high risk patients. Further studies identifying patient selection and timing of pLVAD insertion are warranted.

## Compliance with ethical standard

**Conflict of interest** None

## References

- Tanner H, Hindricks G, Volkmer M, Furniss S, Kuhlkamp V, Lacroix D, et al. Catheter ablation of recurrent scar-related ventricular tachycardia using electroanatomical mapping and irrigated ablation technology: results of the prospective multicenter Euro-VT-study. *J Cardiovasc Electrophysiol*. 2010;21:47–53.
- Stevenson WG, Wilber DJ, Natale A, Jackman WM, Marchlinski FE, Talbert T, et al. Irrigated radiofrequency catheter ablation guided by electroanatomic mapping for recurrent ventricular tachycardia after myocardial infarction: the multicenter thermocool ventricular tachycardia ablation trial. *Circulation*. 2008;118:2773–82.
- Zeppenfeld K, Stevenson WG. Ablation of ventricular tachycardia in patients with structural heart disease. *Pacing Clin Electrophysiol*. 2008;31:358–74.
- Bohnen M, Stevenson WG, Tedrow UB, Michaud GF, John RM, Epstein LM, et al. Incidence and predictors of major complications from contemporary catheter ablation to treat cardiac arrhythmias. *Heart Rhythm*. 2011;8:1661–6.
- Miller MA, Dukkupati SR, Mitnacht AJ, Chinitz JS, Belliveau L, Koruth JS, et al. Activation and entrainment mapping of hemodynamically unstable ventricular tachycardia using a percutaneous left ventricular assist device. *J Am Coll Cardiol*. 2011;58:1363–71.
- Sosa E, Scanavacca M, d'Avila A, Pilleggi F. A new technique to perform epicardial mapping in the electrophysiology laboratory. *J Cardiovasc Electrophysiol*. 1996;7:531–6.
- Dixon SR, Henriques JP, Mauri L, Sjaauw K, Civitello A, Kar B, et al. A prospective feasibility trial investigating the use of the Impella 2.5 system in patients undergoing high-risk percutaneous coronary intervention (the PROTECT I trial): initial U.S. experience. *JACC Cardiovasc Interv*. 2009;2:91–6.
- Cook S, Windecker S. Percutaneous left ventricular assist devices during cardiogenic shock and high-risk percutaneous coronary interventions. *Curr Cardiol Rep*. 2009;11:369–76.
- Santangeli P, Muser D, Zado ES, Magnani S, Khetpal S, Hutchinson MD, et al. Acute hemodynamic decompensation during catheter ablation of scar-related ventricular tachycardia: incidence, predictors, and impact on mortality. *Circ Arrhythm Electrophysiol*. 2015;8:68–75.
- Maini B, Naidu SS, Mulukutla S, Kleiman N, Schreiber T, Wohns D, et al. Real-world use of the Impella 2.5 circulatory support system in complex high-risk percutaneous coronary intervention: the USpella Registry. *Catheter Cardiovasc Interv*. 2011.
- Basra SS, Loyalka P, Kar B. Current status of percutaneous ventricular assist devices for cardiogenic shock. *Curr Opin Cardiol*. 2011;26:548–54.
- Sjaauw KD, Konorza T, Erbel R, Danna PL, Viecca M, Minden HH, et al. Supported high-risk percutaneous coronary intervention with the Impella 2.5 device the Europella registry. *J Am Coll Cardiol*. 2009;54:2430–4.
- Kar B, Gregoric ID, Basra SS, Idelchik GM, Loyalka P. The percutaneous ventricular assist device in severe refractory cardiogenic shock. *J Am Coll Cardiol*. 2011;57:688–96.
- Friedman PA, Munger TM, Torres N, Rihal C. Percutaneous endocardial and epicardial ablation of hypotensive ventricular tachycardia with percutaneous left ventricular assist in the electrophysiology laboratory. *J Cardiovasc Electrophysiol*. 2007;18:106–9.
- Abuissa H, Roshan J, Lim B, Asirvatham SJ. Use of the Impella microaxial blood pump for ablation of hemodynamically unstable ventricular tachycardia. *J Cardiovasc Electrophysiol*. 2010;21:458–61.
- Zanobini M, Rossi F, Bertera A, Sandano S, Costa C, Fabrizi R, et al. Cardiopulmonary support during electrophysiological procedures for ventricular tachycardias not haemodynamically tolerated. *Perfusion*. 2003;18:79–82.
- Carbucicchio C, Della Bella P, Fassini G, Trevisi N, Riva S, Giraldo F, et al. Percutaneous cardiopulmonary support for catheter ablation of unstable ventricular arrhythmias in high-risk patients. *Herz*. 2009;34:545–52.
- Bunch TJ, Darby A, May HT, Ragosta M, Lim DS, Taylor AM, et al. Efficacy and safety of ventricular tachycardia ablation with mechanical circulatory support compared with substrate-based ablation techniques. *Europace*. 2012;14:709–14.
- Reddy YM, Chinitz L, Mansour M, Bunch TJ, Mahapatra S, Swarup V, et al. Percutaneous left ventricular assist devices in ventricular tachycardia ablation: multicenter experience. *Circ Arrhythm Electrophysiol*. 2014;7:244–50.
- Yu R, Ma S, Tung R, Stevens S, Macias C, Bradfield J, et al. Catheter ablation of scar-based ventricular tachycardia: relationship of procedure duration to outcomes and hospital mortality. *Heart Rhythm*. 2015;12:86–94.
- Tung R, Vaseghi M, Frankel DS, Vergara P, Di Biase L, Nagashima K, et al. Freedom from recurrent ventricular tachycardia after catheter ablation is associated with improved survival in patients with structural heart disease: an International VT Ablation Center Collaborative Group study. *Heart Rhythm*. 2015;12:1997–2007.