Heart, Lung and Circulation (2019) **28,** 134–145
1443-9506/04/$36.00
https://doi.org/10.1016/j.hlc.2018.10.006

REVIEW

Mechanical Circulatory Support During Catheter Ablation of Ventricular Tachycardia: Indications and Options

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Mapping of scar-related ventricular tachycardia (VT) in structural heart disease is fundamentally driven by identifying the critical isthmus of conduction that supports re-entry in and around myocardial scar. Mapping can be performed using activation and entrainment techniques during VT, or by substrate mapping performed in stable sinus or paced rhythm. Activation and entrainment mapping requires the patient to be in continuous VT, which may not be haemodynamically tolerated, or, if tolerated, may lead to adverse sequelae related to impaired end organ perfusion. Mechanical circulatory support (MCS) devices may facilitate haemodynamic stability and preserve end organ perfusion during sustained VT to permit mapping for long periods. Available options for haemodynamic support include an intra-aortic balloon pump (IABP), TandemHeart left atrial to femoral artery bypass system (CardiacAssist Inc., Pittsburgh, PA, USA), Impella left ventricle (LV) to aorta flow-assist system (Abiomed, Danvers, MA, USA), and extracorporeal membrane oxygenation (ECMO); the bypass and assist devices provide far better augmentation of cardiac output than IABP. MCS has potential key advantages including maintenance of vital organ perfusion, reduction of intra-cardiac filling pressures, reduction of LV volumes, wall stress, and myocardial consumption of oxygen, and improvement of coronary perfusion during prolonged periods of VT induction and/or mapping. Observational studies show MCS allows for longer duration of mapping, and increased likelihood of VT termination, without an increased risk of peri-procedural mortality or VT recurrence in follow-up, despite being used in a significantly sicker cohort of patients. However, MCS has increased risk of complications related to vascular access, bleeding, thromboembolic risk, mapping system interference, increase procedural complexity and increased cost. Acute haemodynamic decompensation occurs in ~11% of patients undergoing VT ablation, and is associated with increased mortality. Prospectively identifying patients at risk of acute haemodynamic decompensation in the peri-procedural period may allow prophylactic MCS. Although observational studies of MCS in patients at high risk of haemodynamic decompensation are encouraging, its benefit needs to be proven in randomised trials. This review will summarise the indication for MCS, forms of MCS, procedural outcomes, complications and utility of MCS during VT ablation.

Keywords Ventricular tachycardia • Mechanical circulatory support • Acute haemodynamic decompensation • Intra-aortic balloon pump • Extracorporeal membrane oxygenation • TandemHeart

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Introduction

Recurrent ventricular tachycardia resulting in shocks (or even repetitive anti-tachycardia pacing) from an implanted cardioverter defibrillator (ICD) is associated with poorer quality of life and increased mortality [1–3]. Catheter ablation has an established therapeutic role in the management of ventricular tachycardia (VT) [4]. Randomised controlled trials have shown that catheter ablation is superior to medical therapy in the treatment of post-myocardial infarction-related VT [5,6]. Ablation alleviates arrhythmia burden, reduces ICD shocks, improves quality of life, reduces the need for ongoing anti-arrhythmic drugs, and is cost-effective in the treatment of VT [1,7–10]. In the setting of intractable VT or electrical storm, ablation is a lifesaving intervention, resulting in the resolution of storm in 94% of patients in follow-up [11,12].

Ventricular tachycardia in structural heart disease is commonly related to re-entry in and around regions of scar. Poorly coupled surviving myocytes within scar are capable of slow and circuitous conduction, forming corridors or ''channels'' which support re-entry. Mapping VT is critically dependent on identifying the critical isthmus of conduction for one or more re-entrant VT circuits (Figure 1A, B). A VT circuit can be delineated by mapping the ventricle during sustained VT using activation and entrainment mapping techniques (Figure 1C, D). Entrainment mapping uses pacing to confirm the re-entrant mechanism of VT and identify critical and noncritical areas of a VT circuit, and often complements activation mapping (Figure 1D). However, VT is often unmappable due to haemodynamic instability, unreliable inducibility, or unstable morphology in response to attempts at entrainment manoeuvres. Therefore, contemporary VT mapping strategies are predominantly focussed on substrate mapping.

Substrate mapping is the process of identifying areas likely to support re-entrant VT based on anatomy and electrophysiological characteristics that can be determined during stable sinus or paced rhythm. This entails identification of regions of ventricular scar, based on tissue electrical voltage (Figure 1E) and identification of conducting channels,

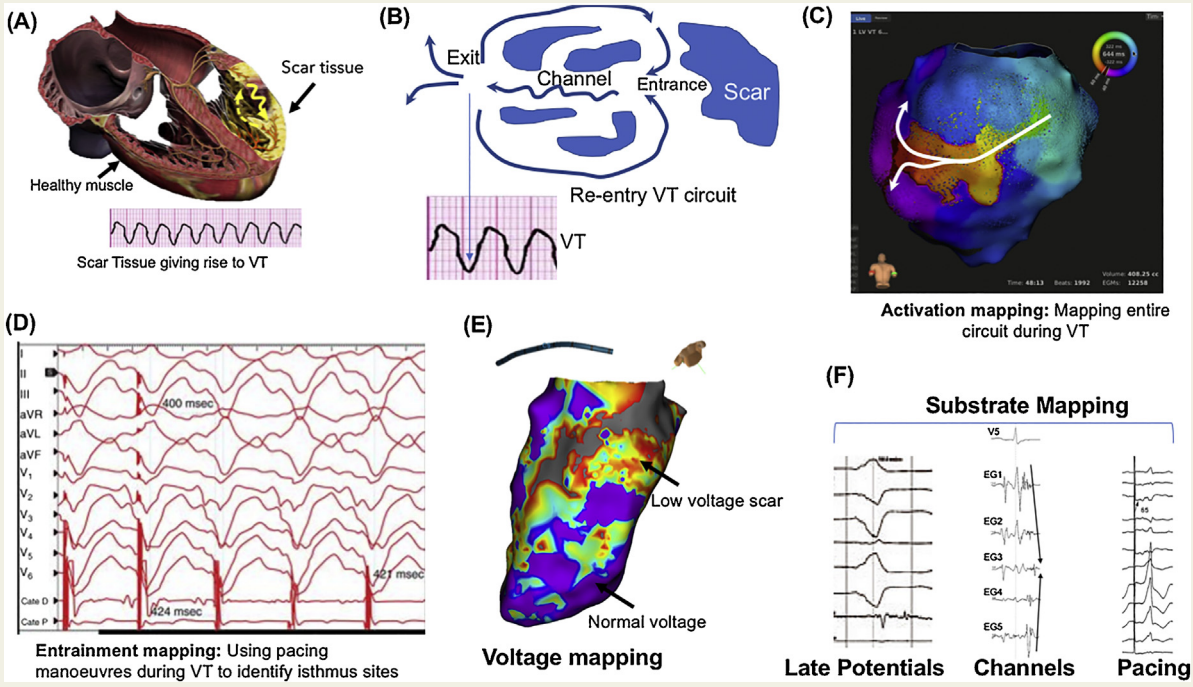

Figure 1 Principles of ventricular tachycardia (VT) mapping.
(A) VT in structural heart disease is invariably related to re-entry in and around scar; (B) re-entry is in and around fixed and/or functional barriers such as scar or valve annulus, with the entrance, the central common isthmus within a channel of slow conduction and exit forming key components of a circuit; (C) the entire VT circuit can be mapped whilst leaving the patient in VT, called activation mapping, however this requires the patient to maintain haemodynamic stability during VT; (D) the critical isthmus of a VT circuit can be determined by performing pacing manoeuvres during VT, called entrainment mapping, however this requires the patient to maintain haemodynamic stability during VT, at least for brief period, or tolerable repeated inductions of VT; (E) substrate mapping identifies components of the re-entry circuit in stable sinus or paced rhythm by identified low electrical voltage regions, consistent with scar (red, green, yellow regions) and healthy tissue (purple regions); (F) signals recorded during substrate mapping can be used to identify conducting channel]s or isthmus sites (late potentials, channels) and/or recording the response of the tissue to pacing to identify delayed conduction time to the formation of a surface QRS complex (pace mapping).

regions of slow conduction via fractionated electrograms, and use of pace-mapping to replicate electrocardiographic (ECG) morphologies of the targeted VT (Figure 1F). Although substrate mapping can facilitate ablation of multiple morphologies of VTs, and VTs that are unmappable due to haemodynamic instability, the localisation of a re-entry circuit is less precise, often requiring more extensive ablation which may result in prolonged procedure times, and collateral damage to structures such as the coronary arteries and phrenic nerve (during epicardial ablation). Substrate identification and targets are not as clear in non-ischaemic cardiomyopathy, comprising one third of the population presenting with VT, where fewer putative channels can be identified during sinus rhythm [13]. Endpoints of substrate ablation are also unclear, as procedural success is still ultimately defined by lack of inducible VT after ablation [14]. Outcomes, in the long term, however, are largely similar using predominantly substrate mapping vs. predominantly activation/entrainment mapping strategies alone [15].

As the majority of VTs in patients with structural heart disease are haemodynamically unstable, prolonged activation and comprehensive entrainment mapping is often not feasible [12,16]. Even a substrate mapping strategy can be complicated by recurrent VT events during catheter manipulation and require multiple cardioversions. Such repetitive brief episodes of unstable VT can have cumulative detrimental effects on end-organ perfusion, lactic acidosis, release of catecholamines and neurohormones and activation of systemic inflammatory cytokines. This may lead to depression of myocardial contractility and worsening end organ perfusion. A major sequelae is post-procedural decompensated heart failure, which is associated with increased mortality in follow-up [17–20]. Acute haemodynamic instability in the peri-procedural setting of VT ablation occurred in 11% of patients with structural heart disease undergoing VT ablation in one study, and was associated with an approximately five-fold increased risk of mortality in follow-up [21].

Intravenous vasopressor and inotropic agents can help support cardiac output during prolonged episodes of VT. However, the extent of support provided is usually insufficient in advanced structural heart disease [22]. In recent years, mechanical circulatory support (MCS) devices have been increasingly used during ablation of patients with advanced structural heart disease and/or co-morbidities with unstable VT, with the aim of facilitating delineation of the VT circuit, whilst maintaining the patient in VT. Another use of MCS devices is for high risk patients with severe advanced heart failure to support them through the procedure even when only a substrate-based ablation procedure is planned. MCS are able to maintain blood pressure and end-organ perfusion, may reduce the incidence of acute peri-procedural heart failure and enable a more rapid post-procedural recovery. The focus of this contemporary review is to discuss the indications for MCS during VT ablation, outline the advantages and limitations of current MCS devices, and summarise current evidence for MCS during VT ablation.

Indications for Mechanical Circulatory Support

Although MCS may be used as an emergent ''rescue'' therapy during VT ablation for patients who develop intra-procedural haemodynamic deterioration and cardiogenic shock refractory to vasopressors and inotropes, the contemporary challenge is to clearly define the clinical and arrhythmia variables that constitute ''high-risk'' so that appropriate candidates can be identified and pre-emptively allocated for MCS during VT ablation. Underuse of MCS during VT ablation risks failure to prevent avoidable peri-procedural morbidity and mortality, whereas an inappropriately low threshold for MCS predisposes to excessive cost expenditure and unnecessary exposure to the risk of device-related complications [19]. In a series of 193 patients undergoing VT ablation, Santangeli et al. noted that acute haemodynamic compromise occurs in 11% of patients. Notably, acute haemodynamic compromise did not occur during sustained VT activation mapping, but was observed during *stable sinus or paced rhythm* during substrate ablation. Univariate predictors of acute haemodynamic compromise included advanced age, ischaemic cardiomyopathy, more severe LV dysfunction and heart failure status, use of general anaesthesia, electrical storm at presentation, and comorbidities such as diabetes and obstructive pulmonary disease [21] Incorporating the relative impact of each of these variables, the authors produced a risk score (PAAINESD) that has been adopted in subsequent studies of VT ablation (Table 1) [17,23]. Prospective validation of this risk score has not been performed. Nevertheless, it represents a promising risk stratification tool to guide the selection of patients for MCS.

Table 1 PAAINESD score for predicting acute haemodynamic decompensation during VT ablation.

Variable	Score
Pulmonary disease [chronic obstructive]- COPD	5
Age >60 years	3
Anaesthesia (general)	4
Ischaemic cardiomyopathy	6
NYHA Class III or IV	6
Ejection fraction <25%	3
Storm (VT)	5
Diabetes Mellitus	3

Clinical variables found to be associated with acute haemodynamic decompensation in the study by Santangeli et al. [21]. The incidence of acute haemodynamic decompensation was found to significantly increase with increasing risk score (<10 points 2%; 10–16 points 6% and ≥17 points 24%). Removal of general anaesthesia variable does not change this relationship. Derived from Santangeli et al. [21].

Abbreviations: NYHA, New York Heart Association; VT, ventricular tachycardia.

Mechanical Circulatory Support Devices (MCS)

Devices (MCS) that have been used during VT ablation include: (i) intra-aortic balloon pump (IABP) counterpulsation, (ii) TandemHeart left atrial-to-femoral artery bypass (CardiacAssist Inc., Pittsburgh, PA, USA), (iii) Impella axial blood flow pump (Abiomed, Danvers, MA, USA), and (iv) extracorporeal membrane oxygenation (ECMO) or peripheral cardiopulmonary bypass (CPB). A comparison of the mechanism of action, advantages, limitations and contraindications of these devices is provided in Table 2. The basic mechanisms of these devices are summarised in Figure 2A–C.

Intra-Aortic Balloon Pump Counterpulsation (IABP)

Commonly used in the cardiac catheterisation lab, IABP carries the obvious advantage of familiarity of use and relatively smaller vascular access (7.5-8Fr). IABP insertion most commonly involves introduction of a balloon catheter through the femoral artery using a Seldinger technique. The balloon is positioned within the descending aorta such that the tip lies a few centimetres below the left subclavian artery and the proximal end is above the renal arteries. The balloon inflates during diastole to increase diastolic pressure and coronary perfusion. Deflation of the balloon during systole decreases afterload and left ventricular (LV) workload. However, IABPs provide very modest augmentation of cardiac output (0.5 L/min). Furthermore, as counterpulsation timing is dependent on ECG-based triggers that assume a stable and regular rhythm, their use is severely limited during rapid VT [19]. Complications relating to IABP are limb ischaemia, vascular injury or thromboembolic complications. Contraindications include significant aortic regurgitation, peripheral vascular disease and aortic aneurysms.

TandemHeart

The TandemHeart is a percutaneous left atrial-to-femoral artery bypass system that uses an external centrifugal pump (Figure 2A) [24]. A 21-French venous cannula is placed transseptally into the left atrium using fluoroscopic and/or

Table 2 Overview of mechanical circulatory support devices for haemodynamic support during ventricular tachycardia ablation.

Device	Mechanism	Advantages	Limitations	Contraindications
IABP	Diastolic support and systolic unloading via deflation and inflation of balloon within aorta	Relative ease of insertion Widespread availability Lower cost Smaller size vascular access (7.5–8 Fr)	Modest haemodynamic support (approximately 0.5 L/min) Function dependent on ECG triggers that assume regular stable rhythm–not ideal when in VT	Moderate to severe AI Severe PVD Aortic dissection or aneurysm
Impella 2.5/CP/5	Percutaneous left ventricle-to-ascending aorta axial pump	Single site of peripheral vascular access No need for trans-septal puncture Allow augmentation of cardiac output by 2.5 L/min (Impella 2.5), 3.5 (Impella CP) or 5 L/min (Impella 5)	Impella 2.5 and CP provides only partial LV support Requires large arterial cannula Electromagnetic interference with magnetic-based mapping systems May require trans-septal LV mapping	Moderate to severe AI or AS Severe PVD Ventricular septal defect Left ventricular thrombus Mechanical aortic valve Right ventricular failure
TandemHeart	Percutaneous left atrial-to-femoral artery bypass using external centrifugal pump	Can provide up to 5 L/min of cardiac output augmentation	Interference with trans-septal mapping usually mandates retrograde approach for VT mapping and ablation Requires large venous and arterial accesses	Moderate to severe AI Severe PVD Ventricular septal defect
ECMO	Peripheral CPB using external membrane oxygenator system	Provides complete biventricular support Useful in severe RV failure Allows augmentation of cardiac output >4.5 L/min	Increased complexity, requiring additional personnel and resources, large cannulae	Severe PVD Uncontrolled bleeding diathesis

Abbreviations: AI, aortic insufficiency; AS, aortic stenosis; CPB, cardiopulmonary bypass; ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; PVD, peripheral vascular disease; VT, ventricular tachycardia; ECG, electrocardiograph.

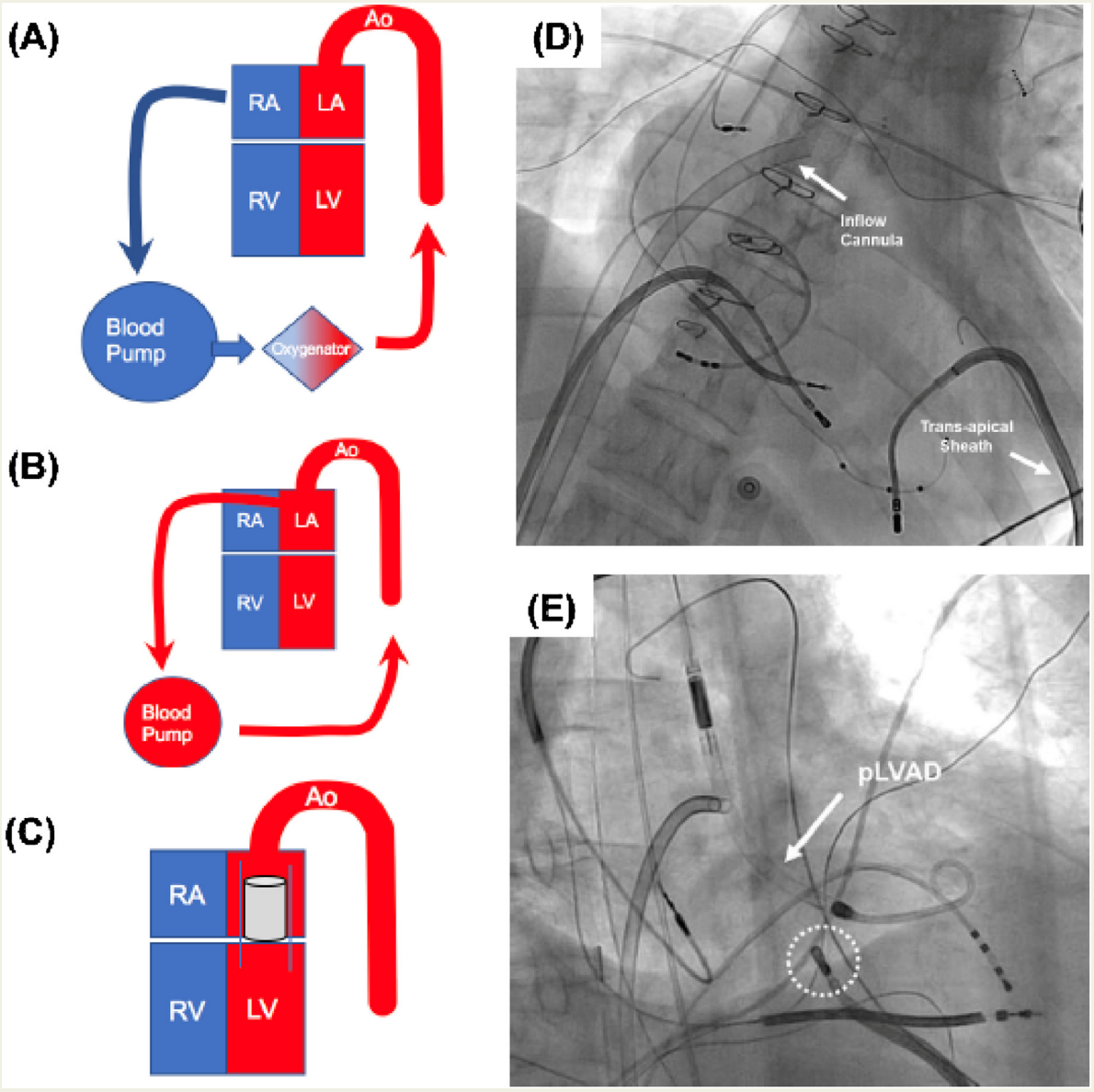

Figure 2 Mechanical Circulatory Support Devices.
Schematic showing the (A) extracorporeal membrane oxygenation system (right atrium-aorta; (B) TandemHeart system (left atrium); (C) and Impella system (left ventricle-aorta; (D) X-ray picture of the TandemHeart inflow cannula; (E) and a Impella system. Adapted D-E adapted from Palaniswamy et al.,[19] with permissions (license number 4444140041290). See text for details.
Abbreviations: Ao, aorta; LA, left atrium; LV, left ventricle; pLVAD, TandemHeart; RA, right atrium; RV, right ventricle.

intracardiac echocardiographic guidance, which forms the inflow cannula. A 17-French arterial cannula is placed into the femoral artery following arteriotomy tract dilation. The left atrial and femoral arterial ports are connected to a centrifugal pump that withdraws oxygenated blood from the left atrium and delivers up to 5 L/min to the arterial circulation. Intravenous heparin is required to keep an activated clotting time (ACT) ≥300 s. Use of the TandemHeart usually requires a retrograde aortic approach for subsequent VT mapping and ablation due to interference with trans-septal mapping caused by the venous cannula. Furthermore, device output may have to be reduced slightly to allow sufficient motion of the aortic valve to enable catheter passage across the aortic valve [25]. A disadvantage of the TandemHeart is the risk of major vascular complications due to the large venous and arterial accesses required. There are a number of potential complications related to the required large vascular access, which include cardiac tamponade (during left atrial cannula placement), bleeding (need for systemic anticoagulation), critical limb ischaemia (related to vascular port), sepsis, and residual atrial septal defects (due to large size of the trans-septal access port) [19].

Impella

The Impella 2.5 is an axial blood flow pump that is percutaneously inserted through a 13-French sheath in the femoral artery over a guide wire. The catheter is advanced in retrograde fashion across the aortic valve into the left ventricle, and appropriate location is then confirmed by fluoroscopy and pressure curve sensors built into the catheter [19]. The catheter sits in the left ventricle, generally 4 cm below the aortic annulus with the outlet just above the aortic root and pumps blood out to the ascending aorta, producing an output of up to 2.5 L/min (Figure 2). Augmentation of cardiac output can be increased to 3.5 L/min or 5 L/min with the use of the Impella CP or Impella 5.0, respectively. However, the Impella CP loads onto a larger (14-French) sheath and the Impella 5.0 requires surgical cut down for insertion. Intravenous heparin is given after establishing safe vascular access to maintain an ACT $\geq$250 s. The Impella device may cause electromagnetic interference (EMI) during VT ablation, if magnetic-based electroanatomic mapping systems are used. This is usually most pronounced when mapping the ventricular outflow tract, LV summit region due to the proximity of the motor to the magnetic sensor of the mapping catheter. It manifests in a temporary inability to acquire mapping points or distortion of the catheter position on the mapping system. In most cases, though, EMI is mild and resolved with reducing the revolutions per minute of the driving motor [22]. A trans-septal approach to the left ventricle may be preferred to avoid interference with the cannula, but EMI is just as likely when mapping the LV summit.

At comparable flow rates, the Impella is more efficient in reducing LV end-diastolic pressure and myocardial oxygen demand compared with the TandemHeart at comparable flow rates [26]. Possible complications of the Impella include vascular complications (injury, haematoma, retroperitoneal bleeding, pseudoaneurysm), valve injury (aortic), ventricular ectopy and arrhythmias from mechanical irritation caused by the inflow cannula, and thromboembolism [19].

Extra-Corporeal Membrane Oxygenation (ECMO)

Veno-arterial ECMO involves a 19–25 Fr venous cannula inserted in the right atrium via the femoral venous system and a 17–21 Fr arterial cannula placed in the aorta. Blood is extracted from the venous system (right atrium) using a centrifugal pump, and is passed through an external membrane oxygenator system and then its return into the arterial system (Figure 2) [24]. It can be performed percutaneously at the bedside using femoral or jugular vessels. Alternatively, surgical techniques can be used to facilitate direct cannulation of the right atrium and aorta. ECMO provides biventricular circulatory support and oxygenation thereby alleviating concerns about the potentially deleterious impact of prolonged VT on ventricular function, coronary perfusion, pulmonary perfusion, and reducing the need for additional vasopressor support. It is the MCS of choice in patients with severe right ventricular (RV) dysfunction as none of the other MCS adequately address RV output [25]. Limited reports of the Impella RP device show promise but tend to interfere with ablation in the right ventricle [27].

Clinical Experience

Due to the lack of prospective randomised controlled trials (RCT), data evaluating the role of MCS during VT ablation is limited to observational studies prone to confounding and bias. Nevertheless, the collective data from these studies provides important insights into the safety, feasibility and efficacy of MCS for VT ablation. Table 3 summarises the characteristics and findings of selected clinical series.

IABP

In a retrospective study of 12 patients receiving IABP, tolerability of VT was not improved relative to a control group receiving no MCS (n = 34) [28]. The use of an IABP did not permit greater induction or duration of VT, nor did it increase the number of VTs that were terminated. Accordingly, the rate of re-do VT ablation, recurrent ICD therapies and VT recurrence at follow-up (19 $\pm$ 12 months) was similar. To date, there have been no studies of IABP use that contradict these negative findings. The lack of benefit with IABP is likely explained by its modest augmentation of cardiac output and susceptibility to asynchronous counterpulsation in the setting of VT. Nevertheless, IABP has a role in patients with decompensated heart failure when VT control requires early ablation before optimisation of heart failure status. Even in substrate modification, it helps to maintain coronary perfusion and reduce afterload, and can potentially minimise the cumulative effect of repetitive haemodynamic insults of vasoplegic drugs and bursts of VT during the procedure.

Percutaneous Ventricular Assist Devices (PVAD)

Since the first published use of TandemHeart for haemodynamic support of unstable VT, several case series have reaffirmed its safety and feasibility in this role [25,29,30]. The major advantages of PVAD, when compared to no haemodynamic support and/or IABP is that patients maintain end-organ perfusion during VT for longer periods, require fewer terminations of VT (shocks or anti-tachycardia pacing) due to haemodynamic instability, resulting in a greater likelihood that VT is terminated during ablation. Furthermore, despite a sicker profile of patients in these studies, who undergo PVAD implantation and longer mapping times in VT, there are no deleterious effects on end-organ perfusion [19]. The studies, however, have not shown improvement in recurrence of VT in short- to mid-term follow-up.

In the study by Bunch et al., 13 patients receiving TandemHeart during VT ablation were compared with 18 propensity-matched controls [25]. A higher number of mean VTs were induced (3.2 vs. 1.6; p = 0.04) and ablated (2.2 vs. 1.5; p = 0.04) in the TandemHeart cohort without an increase in

Table 3 Summary of studies reporting outcomes following use of mechanical circulatory support devices during ventricular tachycardia ablation.

Study	Design	n	Device	Substrate	Follow-Up	Peri-Procedural Outcomes	Long-Term Outcomes
Aryana 2017 [36]	Retrospective analysis of US Medicare database	345	PVAD (n = 230) IABP (n = 115)	60% ischaemic; 40% non-ischaemic	≥12 months	Compared to IABP, PVAD associated with reduced mortality (6.5% vs. 19.1%), cardiogenic shock (9.1% vs. 23.5%), acute kidney injury (11.7% vs. 21.7%), 30-day re-hospitalisation (27.0% vs. 37.8%), and shorter hospital LOS (8.4 vs. 10.6 days).	PVAD group had similar re-do VT ablation rates at 1-year, compared with IABP (10.2% vs. 14.0%; p = 0.34).
Aryana 2014 [28]	Retrospective, single-centre	68	Impella 2.5/CP (n = 34, control (n = 34)	53% ischaemic; 47% non-ischaemic	19 ± 12 months	Compared to control, VT sustained longer with Impella (27.4 vs. 5.3 minutes) and higher number of VTs terminated (1.2 vs. 0.4), but similar acute procedural success (71% for both). Impella group had shorter hospital LOS (4.1 vs. 5.4 days) and lower incidence of composite endpoint [30-day re-hospitalisation, re-do VT ablation, recurrent ICD therapies and 3-month mortality] (12% vs. 35%).	Similar VT recurrence at follow-up for Impella (26%) and control (41%) groups (p = 0.31).
		12	IABP (n = 12)			No difference in composite endpoint [30-day re-hospitalisation, re-do VT ablation, recurrent ICD therapies and 3-month mortality] between IABP and control group (33% vs. 41%; p = 0.74).	Similar VT recurrence at follow-up for IABP (33%) and control (35%) groups (p = 1.00).
Baratto 2016 [39]	Retrospective, single-centre	64	ECMO (n = 64)[a]	45% ischaemic; 55% non-ischaemic	23 ± 13 months	No inducible VT achieved in 69%. In-hospital mortality was 1.5%. Major complications included acute kidney injury (6%), vascular injury (4%) and acute heart failure (8%).	At follow-up, VT recurred in 33%, overall mortality was 12%, and rate of transplantation was 9%. Transplant and LVAD-free survival was 69%.
Bunch 2012 [37]	Retrospective, multi-centre, propensity-matched	31	TandemHeart (n = 13), control (n = 18)	65% ischaemic; 35% non-ischaemic	9 ± 3 months	Compared to control, greater induction of VTs in TandemHeart group (3.2 vs. 1.6) but similar acute procedural success (77% vs. 67%; p = 0.69). No significant difference in peri-procedural complications.	No significant difference in survival free of ICD shocks/recurrence of sustained VT between TandemHeart (55%) and control (48%) (p = 0.96).
Enriquez 2018 [23]	Retrospective, single-centre	21	ECMO (n = 21)[b]	90% ischaemic; 10% non-ischaemic	10 days	Acute procedural success achieved in 83%. In-hospital mortality was 62%. Major complications included vascular injury (14%), cannula-site thrombosis (5%) and thromboembolism (5%).	At 6 months, overall survival and VT-free survival was 33% and 24%, respectively. Transplant and LVAD free survival was 19%.
Kusa 2017 [31]	Retrospective, single-centre, propensity-matched	194	Impella 2.5 (n = 80), Impella CP (n = 29), control (n = 85)[c]	57% ischaemic; 4% non-ischaemic	7.17 months	In PM analysis, procedure duration longer in Impella group (416 vs. 350 minutes, p = 0.008) but no significant difference in VT inducibility at procedure end (14% vs. 10%; p = 0.43), complications (11% vs. 3%; p = 0.18) or hospital LOS.	No significant difference between Impella and control group in PM analysis for mortality (5% vs. 8%; p = 0.50), transplantation (5% vs. 0%; p = 0.25) or recurrent VT (26% vs. 21%; p = 0.29)

Table 3. (continued).

Study	Design	n	Device	Substrate	Follow-Up	Peri-Procedural Outcomes	Long-Term Outcomes
Lu 2013 [38]	Prospective, single-centre	16	Impella 2.5 (n = 5), peripheral CPB (=5), CF-LVAD (n = 6)	NR	3 months	Acute procedural success similar across Impella, CPB and CF-LVAD groups (60% vs. 60% vs. 50%; p = 0.92). In Impella group, 2 patients had insufficient haemodynamic support, 1 had major bleeding and 1 had vascular access complication.	Overall, at 3 months, clinical success (>75% reduction of VT episodes) was 73%. Median time for VT recurrence was 55 days.
Mathuria 2017 [17]	Retrospective, single-centre	93	TandemHeart (n = 15), Impella (n = 21)[d], control (n = 57)	55% ischaemic; 45% non-ischaemic	3 months	30-day mortality of 58.3% for rescue LVAD, compared to 4.2% for pre-emptive LVAD (p = 0.003) and 3.5% for control group (p = 0.001). No significant difference in VT inducibility post-procedure between groups.	At 3 months, there was no significant difference in freedom from VT between rescue LVAD (60%), pre-emptive LVAD (74%) and control (56%) groups.
Miller 2013 [34]	Prospective, open label, non-randomised study	20	Impella 2.5 (n = 20)	35% ischaemic; 65% non-ischaemic	30 days	VT rendered non-inducible in 75%. Rates of 30-day mortality, re-hospitalisation and recurrent VT were 10%, 25% and 20%, respectively. Acute kidney injury occurred in 15%. Mean LOS was 5.2 days.	NR
Miller 2011 [22]	Retrospective, single-centre	23	Impella 2.5 (n = 10), IABP (n = 6), control (n = 7)	48% ischaemic; 52% non-ischaemic	30 days	In Impella group, VT maintained for significantly longer (66.7 vs. 27.5 minutes, p = 0.03) and higher rate of ≥1 VT termination during ablation (90% vs. 38%, p = 0.03). No difference in markers of end-organ hypoperfusion, including cerebral oximetry.	NR
Reddy 2014 [35]	Prospective, multi-centre registry	66	IABP (n = 22), Impella 2.5 (n = 25), TandemHeart (n = 19)	70% ischaemic; 30% non-ischaemic	12 ± 5 months	Compared to IABP, more unstable VTs were mapped with PVAD (1.05 vs. 0.32; p < 0.001) and greater number of VTs terminated (1.59 vs. 0.91; p = 0.007), but with similar procedural success (89% vs. 86%). No significant difference in peri-procedural complications or LOS.	Compared to IABP, Impella and TandemHeart cohort had similar VT recurrence (42% vs. 50%) and mortality (36% in both) at 1 year.

Abbreviations: CF, continuous flow; ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; ICD, implantable cardioverter-defibrillator; LOS, length of stay; LVAD, left ventricular assist device; NR, not reported; PM, propensity matched; PVAD, percutaneous ventricular assist device; VT, ventricular tachycardia.

[a]5 patients received ECMO as ''rescue'' intervention following development of intra-procedural haemodynamic compromise.

[b]All 21 patients received ECMO as rescue intervention following development of intra-procedural haemodynamic compromise.

[c]78 patients included in 1:1 propensity matched analysis.

[d]24 patients underwent LVAD implantation prior to ablation, 12 patients underwent ''rescue'' LVAD following development of intra-procedural hemodynamic compromise.

haemodynamic compromise. However, this did not translate to improved acute procedural success (77% vs. 67%; p = 0.69) or greater VT-free survival (55% vs. 48%; p = 0.96) at follow-up (9 ± 3 months).

Several studies have demonstrated similar findings for the Impella device–namely, an increased tolerability of VT for longer periods, enabling more detailed mapping and ablation using activation and entrainment mapping, more unstable VTs targeted and ablated during ablation, but without an associated increase in acute procedural success or VT-free survival, despite a much sicker cohort being subjected to MCS [22,28,31]. Despite extended periods of mapping, no end-organ damage was noted. A retrospective analysis of the US National Medicare Database and a prospective, multicentre registry also demonstrated the use of PVADs (TandemHeart or Impella) was not associated with a reduction in VT recurrence at 1 year [29,32]. In a recent study, Muser et al. reported outcomes of prophylactic use of PVAD (Impella 2.5 or CP) in 75 patients perceived to be at high risk of peri-procedural acute haemodynamic decompensation and compared them to 75 propensity-matched controls without prophylactic PVAD [33]. Peri-procedural acute haemodynamic decompensation occurred in a greater proportion of control patients without PVAD vs. with upfront PVAD (23% vs. 7%, respectively $p < 0.001$); moreover, 12% in the control group required rescue PVAD. Upfront PVAD, compared to control was associated with greater VT non-inducibility (81% vs. 62%, p = 0.02), and lower rate of death or transplant at 12 months (33% vs. 66%, $p < 0.01$) but no significant difference in cumulative incidence of recurrent VT, nor procedural complications (11% vs. 7%, p = 0.56). This study suggested that prophylactic PVAD may provide significant advantages in high risk patients, but needs evaluation in a randomised trial. Prophylactic implementation of PVAD is a desirable in the appropriately selected group as the outcomes of rescue PVAD are much worse [23].

It is possible the detailed mapping and ablation enabled by MCS may have long-term benefits that are being masked by the heterogeneity of substrates in current studies. The effect of MCS on VT recurrence may be disproportionately concentrated in patients with non-ischaemic cardiomyopathy, in whom substrate-based ablation is less effective due to the frequent absence of clear identifiable targets. This hypothesis is supported by the subgroup analysis in the study by Aryana et al., in patients with non-ischaemic cardiomyopathy, where the use of the Impella device was associated with reduced incidence of the composite endpoint (primarily driven by lower rates of recurrent ICD therapies and re-do VT ablation) [28]. Alternatively, the lack of improved long-term outcomes with use of MCS may reflect the selection bias inherent in the current observational data. In the majority of studies, patients were selected for PVAD based on clinical variables predicting haemodynamic instability, and these same variables may also portend poorer long-term prognosis.

With regards to complications, cardiac tamponade was reported in several studies using PVADs, with incidences ranging from 3–11% [22,28,34,35]. The vast majority of these cases occurred in patients undergoing epicardial mapping and ablation. This is a noteworthy finding as both the TandemHeart and Impella require systemic anti-coagulation, which may expedite the progression from pericardial bleeding to tamponade. Despite concerns regarding vascular injury, an analysis of 230 patients receiving PVAD demonstrated a reassuringly low incidence (1.7%) of vascular access site complications [36]. This likely reflects the growing use of specific vascular closure devices to pre-close arteriotomy tracts prior to upgrading to larger sheath sizes [22,28,35,37,38]. Encouragingly, no studies reported an increased incidence of stroke or systemic embolism with the use of either Impella or TandemHeart.

ECMO

Baratto et al. reported favourable outcomes with use of ECMO in 64 patients (45% ischaemic cardiomyopathy; mean left ventricular ejection fraction (LVEF) 27%) undergoing VT ablation [39]. There was only one in-hospital death (1.5%) secondary to acute heart failure, though three (5%) patients required emergency heart transplantation. Device-related complications were rare, comprising two cases of acute peripheral artery ischaemia that recovered uneventfully following revascularisation. At follow-up (mean 23 months), freedom from VT was 67% and overall survival was 88%. Conversely, poorer outcomes with ECMO were reported in another series of 21 patients (90% ischaemic cardiomyopathy; mean LVEF 21.1%) [23]. In this cohort, in-hospital mortality was 62%. Device-related complications included major vascular access site bleeding in three patients (14%), thrombosis of ECMO cannula in one patient (5%), and embolism of LV thrombus in one patient (5%). At 6 months, overall and VT-free survival were only 33% and 24%, respectively. It is important to note that all patients in this latter series received ECMO as ''rescue'' therapy *after* development of acute haemodynamic collapse, whereas ECMO use was pre-emptively planned in 92% of cohort in the Baratto et al. study [14]. A similar discrepancy in outcomes by indication is also seen in studies of PVADs, with one series demonstrating rise of peri-procedural mortality from 4.2% with pre-emptive PVAD use to 58.3% with ''rescue'' indication [17]. These findings again strongly emphasise the importance of thorough pre-procedural assessment to ensure high-risk, potentially unstable patients are identified and appropriately triaged to MCS prior to undergoing VT ablation. ECMO tends to cause marked stasis of blood in the LV as stroke volume can be low reflected in the reduced aortic valve opening. Hence, ECMO requires systemic heparinisation with a target activated clotting time generally >250 seconds.

Haemodynamic Monitoring During MCS

Haemodynamic monitoring, especially during continuous MCS, is challenging. Arterial blood pressure and pulse oximetry are suboptimal due to lack of pulsatile flow.

Centres with expertise in MCS propose use of cerebral oximetry as it does not require pulsatile flow for accurate measurement. Cerebral tissue oxygen saturations of >50–55% or even >60% are suggested based on data from patients undergoing carotid endarterectomy and cardiac surgery, and no validated cut-off exists for patients undergoing VT ablation. It may also guide successful weaning from MCS [19].

Table 4 Optimising patient outcomes during MCS in VT ablation.

Preparation	Details
Pre-procedural preparation	Coronary artery status 12 lead ECGs of spontaneous VT; ICD electrograms of spontaneous VTs (rate, CL, morphology) Substrate characterisation (MDCT, cMRI) Ventricular function and valvular assessment (echo, with contrast) to exclude AI, LV thrombus Vascular access assessment (CT femoral arteries and descending aorta for atherosclerotic occlusion ± femoral venous ultrasound to exclude occlusion) Anti-arrhythmic drug therapy (cessation of oral therapy; temporising intravenous short-acting therapy) Anaesthesia review
The team	• Interventional cardiologists • Cardiothoracic surgeons • Vascular surgeons • Cardiac anaesthestists • Perfusionists • Lab and theatre nurses • Mapping and EP techs • Intensive care unit team and bed
The equipment	• TOE ± ICE • 3D mapping • Mapping, ablation catheters plus spares • ECMO pump and long lines (or other MCS equipment) • Percutaneous and cut down equipment • Vascular clamps • Proglides • Cerebral oximetry monitoring • Pericardiocentesis equipment
The procedure	• Secure continuous power • Adequate lab space for ECMO/MCS • Trans-septal ± retrograde access (some advocate both) • Epicardial access usually pre ECMO/MCS especially in non-ischaemic cardiomyopathy with basolateral substrate/VT, ACM, repeat procedures after failed endocardial ablation, 12-lead ECG or imaging suggesting epicardial target • Invasive arterial SBP >90 mmHg, MAP >70 mmHg • Continuous pulse oximetry • Consider central venous pressure ± PA catheter, CI 2–2.4 L/min/m^2 • LV sheath for LV pressures • ECG • Urine output and hourly fluid/balance assessment • Blood gases and lactate • Antibiotics • Continuous cardiac imaging (TOE or ICE) • Continuous intravenous heparin ACT >250 seconds

Abbreviations: ACT, activated clotting time; AI, aortic incompetence; CI, cardiac index; CL, cycle length; ECMO, extracorporeal membrane oxygenation; EP, electrophysiology; ICD, implanted cardioverter defibrillator; ICE, intra-cardiac echo; LV, left ventricular; MAP, mean arterial pressure; MCS, mechanical circulatory support; MDCT, multi-detector CT; MRI, magnetic resonance imaging; PA, pulmonary artery; SBP, systolic blood pressure; TOE, transoesophageal; VT, ventricular tachycardia.

Integrated Care For Ventricular Tachycardia

Catheter ablation for VT is one of the most challenging procedures in cardiology. The complexity is even further enhanced by use of MCS. Key pre-procedural preparation is essential including defining anticoagulation status, VT and substrate characterisation by 12-lead ECG/ICD electrograms and cardiac imaging, planning vascular access, optimising haemodynamics and biventricular function, and anaesthesia assessment (Table 4). Throughput from multiple teams is required including procedural cardiologists, general cardiologists, advanced heart failure physicians, cardiac anaesthetists, perfusionists, cardiothoracic surgeons, skilled lab and theatre nurses, 3D mapping and electrophysiology technicians, sonographers and intensive care teams (Table 4). Long-term bail-out plans in case of persistent severe LV dysfunction and haemodynamic failure should be discussed and formulated prior to MCS-assisted VT ablation. Key equipment needs to be readily available for the procedure (Table 4). Work from Della Bella and colleagues exemplifies the value of this team in managing the sickest of all cardiology patients [40]. A coordinated team approach leads to peri-procedural optimisation of the patient. This is critical as acute outcomes during the VT ablation procedure have major prognostic implications, and successful ablation is likely associated with a reduction in cardiac mortality. Non-inducibility of all VTs yields much better outcomes than non-inducibility of the clinical VT alone (partial success) or procedural failure (persistent inducibility of the clinical VT).

Conclusions

Mechanical circulatory support (MCS) during VT ablation has key advantages including maintenance of vital organ perfusion, reduction of intra-cardiac filling pressures, reduction of LV volumes, wall stress, and myocardial consumption of oxygen, improvement of coronary perfusion and support of systemic circulation during prolonged periods of VT induction and/or mapping [41]. MCS facilitates mapping of a greater number of VTs, for longer periods using activation and entrainment mapping, which is otherwise not possible due to haemodynamic instability, and which increases the likelihood of termination during ablation. Despite the absence of major impact on VT recurrence, MCS allows therapy in a sicker cohort of patients without an adverse effect on mortality. However, complications related to vascular access, trans-septal access, anticoagulation, thromboembolism and the cost of MCS are chief impediments to its widespread use. Although there is no evidence that this translates to an improvement in long-term freedom from VT, findings may be obfuscated by the heterogeneity, confounders and biases in current observational studies. Prospective randomised controlled trials are required to clarify the clinical impact of MCS during VT ablation for specific patient groups, particularly those with non-ischaemic cardiomyopathy; and, also to assess the economic impact and cost-effectiveness of current MCS devices. Prospectively identifying patients at highest risk of acute peri-procedural decompensation is paramount in optimising the risk/benefit profile and guiding prophylactic MCS before VT ablation. The decision to use MCS during VT ablation should be based on careful consideration of individual clinical, electrophysiologic and procedural factors within a multidisciplinary setting.

Funding Sources

None.

Acknowledgements

None.

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