Scar Non-Excitability using Simultaneous Pacing for Substrate Ablation of Ventricular Tachycardia

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Short title: Simultaneous pacing and ablation of VT

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ABSTRACT

Objectives

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To describe an expedited strategy of simultaneous high-output pacing during radiofrequency ablation to achieve scar homogenisation and electrical inexcitability as an approach for substrate ablation for scar-related ventricular tachycardia (VT).

Background

Scar homogenisation with additional testing for electrical inexcitability are known endpoints for catheter ablation, but achieving both can be time consuming. We describe a strategy of simultaneous pacing during radiofrequency ablation to expedite this approach.

Methods and Results

Ten patients (age 74 ± 6 years; all men, LV ejection fraction of $33\% \pm 8\%$, ischaemic cardiomyopathy, 9; VT storm, 7) underwent scar homogenisation with electrical inexcitability to pacing (10mA, 9ms pulse width), as well as non-inducibility of any VT as an acute procedural endpoint. Thirty-four VTs were inducible in 10 patients with a total of 1127 ablation lesions applied. Median ablation lesions per patient was 97 lesions (interquartile range, IQR₂₅₋₇₅ 71-151) and the total ablation time was 49 minutes (IQR₂₅₋₇₅ 45-56 minutes) with average duration per lesion of 32.2 seconds (IQR₂₅₋₇₅ 25.8-37.8 seconds). Average power was 33 Watts (IQR₂₅₋₇₅ 32-38 Watts), average CF was 13g (IQR₂₅₋₇₅ 11.9-14.6g) with a median impedance drop of 9.6 ohms/lesion (IQR₂₅₋₇₅ 8.1-10.0 ohms). There were no ventricular fibrillation episodes using this strategy. The median procedure time was 246 minutes (IQR₂₅₋₇₅ 214-293 minutes). Acute procedural success was seen in 9 patients with 97% of VTs non-inducible.

Conclusion

Simultaneous ablation with high output pacing to achieve scar inexcitability, when combined with scar homogenisation and non inducibility of any VT may be an expeditious, safe and effective technique for catheter ablation.

KEYWORDS

Catheter ablation; Ventricular Tachycardia; Substrate; Pacing

ABBREVIATIONS

AAD-Antiarrhythmic drug(s)

CF-Contact force

FTI-Force time integral

ICD-Implantable cardioverter defibrillator

IQR-Interquartile range

LSI-lesion size index

VA-Ventricular arrhythmia

VT-Ventricular Tachycardia

INTRODUCTION

Complete substrate modification is associated with reduced ventricular arrhythmia (VA) recurrence compared to a standard ablation in patients with scar-related VT (1-3). The mechanism is likely related to elimination of all conducting channels capable of supporting re-entrant VTs. During scar-mediated VT ablation, a number of procedural endpoints have been described, beyond non-inducibility of VT. These include elimination of electrogram-based markers of substrate (late potential abolition, scar dechannelling, elimination of late abnormal ventricular activities, scar homogenisation) (2, 4-7). Ablation until loss of electrical inexcitability is a valid procedural endpoint in addition to other strategies (2, 8, 9). However, achieving all three endpoints of substrate elimination, scar homogenisation, and loss of

electrical inexcitability can be time consuming. In this study, we describe a novel technique of simultaneously performing ablation and high-output pacing until local tissue loss-of-capture, in addition to targeting non-inducibility of any VT, as an expeditious technique for scar-related VT ablation.

METHODS

Study population

Patients with scar-related, drug refractory VT undergoing catheter ablation at a single centre (Department of Cardiology, Westmead Hospital, Sydney, Australia) were included. Pre-procedural data, arrhythmia history, procedural characteristics, complications and follow-up were collected. All patients gave written informed consent for the procedure. This analysis was approved by the Western Sydney Local Health District Human Research Ethics Committee.

Mapping and radiofrequency ablation

Preparation

Procedures were performed under general anaesthesia (GA) as previously described (10). A decapolar catheter inserted into the CS and a quadripolar catheter was deployed to the RV apex. Intracardiac echocardiography (ICE) was routinely used for direct visualisation of catheter position (ViewFlex Xtra, Abbott Medical). Antiarrhythmic drug (AAD) therapy was withheld for 5 half-lives prior to the planned catheter ablation (except in the case of an emergent procedure). Systemic anticoagulation was administered after sheath insertion using intravenous unfractionated heparin to maintain an activated clotting time ≥ 350 seconds prior to left ventricular access, unless epicardial access was planned- in this situation, anticoagulation was commenced after safe epicardial access was established. Implanted cardioverter defibrillators (ICDs) were re-programmed to disable therapies prior to ablation.

The endocardial LV was accessed either transeptally (large curve Agilis, Abbott Medical) or anterogradely (SL1, Abbott Medical), or both. In cases of previously failed endocardial catheter ablation or if pre-procedural imaging strongly indicated intramural/epicardial substrate, epicardial access (via a percutaneous approach) was obtained. Coronary angiography was performed before epicardial ablation to avoid coronary injury and high output pacing (10 milliAmps [mA] and 9 milliseconds [ms] output) to exclude phrenic nerve capture.

VT Induction protocol

Programmed electrical stimulation (PES) was performed in all cases by pacing from a least 2 RV sites using a 400ms drive train with 4 extrastimuli beginning at 300ms, decrementing by 10ms down to ventricular refractoriness. Left ventricular (LV) stimulation was used if VA was non-inducible after RV stimulation. This was followed by burst RV pacing down to ventricular refractoriness from the RV apex. PES and burst RV pacing was then repeated from each site using highest tolerated dose of isoprenaline (2 microgram bolus and up to 40 micrograms/minute) with haemodynamic support to maintain perfusion pressure with inotropic support initiated at the start or during of the case. The same induction protocol was repeated post ablation.

Definitions of spontaneous and non-spontaneous VT

Spontaneous VT (or 'clinical') was defined as any inducible VT that has a 12-lead ECG morphology and rate (within 20ms) matching a VT that had documented to have occurred spontaneously before ablation; only the rate cut-off and intracardiac electrogram data from the implanted ICD (if present) were used when the 12-lead VT morphology was not available before ablation. Non-spontaneous (or 'non-clinical') VTs were inducible VTs that did not have

an identical rate (>20ms difference) or 12-lead ECG morphology to the spontaneous ('clinical VT') documented before ablation (10).

Electroanatomical mapping

3-dimensional (3D) electroanatomical mapping (EAM) of the RV or LV (or both) was performed using EnSite™ Precision (Abbott Medical). High-resolution multi-electrode mapping catheters were used (Advisor HD Grid or Livewire Duodeca 2-2-2 spacing, Abbott Medical). In the EAM system, bipolar electrograms were high pass-filtered at 20-30 Hz and low pass-filtered at 400 Hz. Bipolar electrograms were also pass-filtered from 30 to 500 Hz and digitally recorded using Cardiolab EP system (General Electric Healthcare, Chicago, Illinois).

An endocardial and/or epicardial 3D shell of chamber geometry was constructed for each ventricle and electrograms recorded during a sinus rhythm or paced voltage map. If atrio-ventricular (AV) conduction was preserved, coronary sinus pacing was performed at 600ms. If AV conduction was impaired, dual chamber pacing at 600ms was performed. If biventricular pacing (BiV) was present, dual chamber pacing at 600ms with LV and RV pacing was used. Activation maps of each VT were recorded if the VT was sustained and tolerated. Fill threshold (projection of colour from each mapping point) was set at 10 units for all maps. Conventional ventricular peak-to-peak bipolar voltage parameters were used (dense scar: <0.5 millivolts [mV], low voltage: 0.5mV-1.5mV, normal >1.5mV). Unipolar low-voltage was defined as electrogram amplitude <8.3mV (LV)(11) and <5.5mV (RV)(12). Image integration with pre-procedural contrast-enhanced magnetic resonance imaging or computer tomography scans were used, when available, to identify and reconstruct anatomy and were imported into the 3D mapping system. The chamber (left ventricle [LV] or right ventricle [RV] and space [endo/epicardial] map was based on the characteristics of the induced or spontaneous VA(s). The annulus was defined as a 1:1 ratio between atrial and

ventricular electrograms, as well as direct identification with ICE; and low voltage areas within 1cm of the annulus were excluded from measurements. ICE was used during mapping to confirm catheter apposition to the endocardial surface.

Ablation Protocol

A schema of the ablation approach is shown in Figure 1. Ablation was performed using an 8F bidirectional, irrigated catheter (TactiCath SE, Abbott Medical). The TactiCath SE catheter has 2-2-2 bipole pairs, a 3.5mm distal tip with tri-axial contact force sensors positioned behind the distal tip. A calibrated roller pump (CoolPoint, St. Jude Medical) connected to the ablation catheter delivered normal saline at 17 ml/min if power < 30W or 30ml/min if power > 30W (TactiCath SE) and 8 ml/min if power <30W. An Ampere RF generator (St. Jude Medical) was connected to deliver 550kHz unmodulated sine-wave RF energy pulses in a power-controlled mode. Continuous bipolar pacing from the TactiCath ablation catheter using a stand pacing rate of 600ms and capture threshold of 10mA @ 9ms pulse width maintaining $\geq 10g$ CF (13). Real-time visualisation of the catheter tip using ICE guided the ablation site, ensured contact and stability and was useful to confirm lesion formation. The baseline CF was calibrated while freely floating in the LV to a value of zero grams. The catheter was “zeroed” if there was no pace capture at a 10ms and 2mA output. Catheter orientation and contact was noted with ICE for all ablation lesions. Up to 50W of power was delivered with the aim of $\geq 10ohm$ impedance drop, until there was loss of capture at the ablation site.

Endpoints and success of ablation

Ablation was based on substrate and/or pace mapping alone. The specific approach targeted presumptive channels and exits as determined by paced QRS morphology similarity to the VT QRS morphology with a stimulus-to-QRS interval $>40\text{ms}$, abnormal fractionated potentials, double potentials, late potentials during sinus and paced rhythm, and late abnormal ventricular activities within areas of low voltage as defined above (10). Acute success was defined as non-inducibility of all inducible VTs using the same PES induction protocol with 4 extrastimuli, repeated with an isoprenaline infusion up to 40mcg/min (14).

Pace Capture Protocol

Bipolar pacing has demonstrated a greater predictive value over unipolar pacing in terms of loss of pace-capture and was used in our protocol (15). Compared to unipolar pacing, bipolar pacing uses stimulation poles at the distal catheter tip (anode and cathode) as both are typically in contact with myocardium. In this pacing mode, however, capture can occur either at the anode, cathode or both sites, reducing pace capture accuracy or spatial resolution (16, 17). Unipolar pacing stimulates myocardium adjacent to the distal electrode and ablation site. Unlike bipolar pacing where the electrode spacing is small electrical artefact is minimal, a disadvantage of unipolar pacing is significant electrical artefact during pacing. For each ablation site, pace capture before and during ablation was documented using bipolar pacing at a fixed output of 600ms with 10mA and a 9ms pulse width with a CF $\geq 10\text{g}$ (Figure 2 and 3). Pace capture was defined as present (1:1 or intermittent capture) or absent. Electrically inexcitable scar was defined as regions where there was failure of bipolar pacing capture at 600ms at 10mA and 9ms despite adequate contact force ($\geq 10\text{g}$). At each ablation site, high-output pacing was initiated and, if confirmed to be present by the operator, ablation was performed until loss-of capture at the same site. No pacing or ablation was

performed post ablation. If pacing dependent, the device was programmed DOO/VOO for the ablation after being programmed DDD/VVI for substrate mapping.

OUTCOMES

Acute procedural outcomes were reported as complete success (non-inducibility of any VT, spontaneous or non-spontaneous), partial success (elimination of at least 1 spontaneous VT), and failure (residual inducibility of spontaneous VT) (10). Follow-up outcomes included (1) survival free of any ventricular arrhythmia (VA); (2) arrhythmia control defined as any reduction in the number of VA episodes or number of AADs required for arrhythmia control during follow-up; and (3) overall survival.

FOLLOW-UP

Programming of the ICD or CRT was left to the discretion of the treating cardiologist; however, in general 2 zones were programmed. The first zone was set at a rate at least 20ms below the rate of the slowest VT (with or without ATP) with a second zone programmed at a minimum rate >188 beats per minute programmed to deliver a shock (with or without ATP). All patients were enrolled in a ICD remote monitoring program at Westmead Hospital. All ICD activations are recorded, logged and transmitted to the clinic staff, which prompted an in-office visit for detail evaluation of clinical and device data. Hospital records and outpatient clinic assessments were used to complete follow up.

STATISTICAL ANALYSIS

Statistical Package for the Social Sciences for Windows (SPSS) was used for analysis. Continuous variables were expressed at mean $\pm$ SD if normally distributed; median and interquartile range (IQR) 25% to 75% (Q25 – 75) or full ranges were used if the data was clearly skewed. A 2-tailed *P* value <0.05 was considered statistically significant.

RESULTS

Patient Demographics

Ten patients underwent catheter ablation using the described approach (mean age 73.7 $\pm$ 5.6 years; all men, mean LV ejection fraction of 33.1% $\pm$ 8.2%, Table 1). All procedures were performed under general anaesthesia and all patients had an ICD (6 patients with a single chamber system, 2 with a dual chamber system and 2 patients with a CRT-D device). None of the patients were pacing dependent.

Indication for VT ablation procedure was recurrent ICD shocks in 8 patients (80%), sustained VT in 2 patients (20%). 7 of the 8 patients with ICD shocks presented with electrical VT storm. Underlying aetiology of cardiomyopathy was ischaemic cardiomyopathy in 9 patients (90%) with the remaining patient having idiopathic dilated cardiomyopathy. Prior to catheter ablation all patients were prescribed >1 AAD with beta-blockers in 6 patients (60%) and amiodarone in 9 patients (90%). 1 patient was on sotalol and mexiletine, pre-procedurally. The majority of patients had not undergone a previous catheter ablation (8 patients). Two patients had previously undergone VT ablation (without an ablation to non-excitability approach) targeting the postero-medial papillary muscle in both cases (Supplemental table 1).

Electroanatomical Mapping Features

Endocardial mapping was performed in all cases using a high-density mapping catheter (HD Grid); 3 patients had biventricular EAM. Epicardial mapping was performed in 1 patient (following multiple re-do procedures). Mapping features (including mapping point density, scar surface area and distribution) are summarised in Table 2 and 3. Median number of mapping points was 5054 points (IQR₂₅₋₇₅ 3211-15,059 points). 7 patients had basal inferior LV scar on EAM, 1 antero-septal scar and 2 patients predominally had apical LV scar. Median total endocardial surface area was 132.7cm² (IQR₂₅₋₇₅ 115.1-205.6cm²), bipolar scar area was 65.2cm² (IQR₂₅₋₇₅ 50.3-94cm²), dense bipolar scar area was 25.6cm² (IQR₂₅₋₇₅ 14.5-50.5cm²) and unipolar scar area was 90.0cm² (IQR₂₅₋₇₅ 70.3-187.2 cm²). The median ablation area was 25.5cm² (IQR₂₅₋₇₅ 14.5-50.5cm²) which converted to a median percentage of bipolar scar ablated to be 46% (IQR₂₅₋₇₅ 27-66%).

VT characteristics

12-lead ECGs of the clinical VT were available for 8 patients (Table 1). The majority of patients had a RBBB V1 morphology and superior axis (5 patients, 63%). A total of 34 VTs were inducible for the 10 procedures (median of 4 VTs per procedure IQR_{25-75%} 2-6, Supplemental table 2). Mean CL of induced VT was 394±129ms (median 376ms; IQR₂₅₋₇₅ 295-482ms). In all cases, the ablation strategy was substrate-based targeting abnormal late and fractionated potentials rendering the low voltage tissue electrically unexcitable.

Catheter ablation and Acute Procedural Outcomes

Ablation parameters are summarised in Table 2 and 3. The total number of ablation lesions was 1127 with a median of 97 lesions (IQR₂₅₋₇₅ 71-151) and the total ablation time was 49 minutes (IQR₂₅₋₇₅ 45-56 minutes) with average duration per lesion of 32.2 seconds (IQR₂₅₋₇₅ 25.8-37.8 seconds). Median total energy was 112,826 joules (IQR₂₅₋₇₅ 100,618 – 135,693 joules) and average power was 33.4 Watts (IQR₂₅₋₇₅ 32.4-38.1 Watts). Median impedance drop was 9.6ohms per lesion (IQR₂₅₋₇₅ 8.1-10.0 ohms), average CF was 13g (IQR₂₅₋₇₅ 11.9-14.6g), FTI was 100.0g*s (IQR₂₅₋₇₅ 88.8-107.6g*s) and LSI was 5.2 (IQR₂₅₋₇₅ 4.8-5.5). An example of simultaneous pacing and ablation guided by CF and ICE and impedance parameters is shown in Figure 4.

Median procedure time was 246 minutes (IQR₂₅₋₇₅ 213.8-292.5 minutes). Acute procedural success was seen in 9 patients (90%) and partial success in the remaining patient (non-clinical VT was inducible post ablation and not targeted, Table 3). 33 VTs were non-inducible (97%) following catheter ablation.

Follow-up ICD data was available for all patients and summarised in Table 4. At a median follow-up of 182 days (IQR₂₅₋₇₅ 65-347 days), there was no VT recurrence or repeat ablation procedures in any patient. One patient died within 30 days following re-admission with multi-organ dysfunction *without* recurrent ventricular arrhythmias or a deterioration in left ventricular dysfunction. No patient had an increase in AAD therapy post ablation and 7 patients (70%) had a reduction in AAD therapy, all ceasing amiodarone treatment. In those patients followed up for > 6months, median LVEF was 45%.

Complications

There was no VF episodes during any pacing and simultaneous ablation session.

A major complication occurred in one patient with a retroperitoneal bleed in the context of high pre-procedural risk including peripheral vascular disease and previous iliac stents (despite prior ligation with vascular surgery and pre-planning). The patient required blood transfusion and was managed conservatively. There was no peri-procedural death or stroke in any patient.

DISCUSSION

In this study, we describe the efficacy and safety of simultaneous high-output pacing during radiofrequency ablation to achieve scar homogenisation and electrical inexcitability during substrate-based ablation for VT. The key findings of this study include:

- (1) In a series of patients with established (predominantly) severe ischaemic cardiomyopathy, extensive scar substrate and electrical VT storm presentation; a simultaneous pace and ablate to electrical non-excitability was technically feasible using a high-out, bipolar pacing configuration.
- (2) Conventional ablation parameters could be monitored during pacing; in particular, stable CF was recorded (and confirmed with real-time ICE imaging to confirm catheter apposition to the myocardium) and impedance reduction was consistently accurate without interference from bipolar pacing.
- (3) In >1100 ablation lesions (with a median lesion duration of 32 seconds), there were no immediate complications such as VF, peri-procedural stroke, cardiac tamponade or death.
- (4) Utilizing a simultaneous pace-and-ablate approach, 97% of VTs were no longer inducible using an extensive post ablation induction protocol.

- (5) Adapting a simultaneous pace-and-ablate approach does not prolong total procedural time or RF duration and is comparable to a complete substrate-based approach without this additional ablation lesion endpoint.
- (6) No VA recurrence occurred in any of the patients; with the majority of patients having a reduction in AAD therapy, ceasing amiodarone post ablation.

Substrate versus standard ablation approach

Substrate-based mapping guiding ventricular tachycardia (VT) ablation has become a widespread substitute either alone or combined with a standard technique in patients undergoing VT ablation aiming to transect putative VT exit sites with linear or clustered ablation lesions (18). As it is performed in sinus rhythm, its major advantage as compared to a standard approach is it bypasses the need to induce and sustain VT to facilitate complete mapping. As the majority of VT ablation procedures are limited by (a) VT non-inducibility and (b) haemodynamic intolerance limiting sole reliance on a standard approach, a substrate-based strategy can be performed in all cases.

In a recent meta-analysis including 396 patients (3), long-term VA recurrence and all-cause mortality was substantially lower (43% risk reduction) in the substrate-guided approach compared to the standard ablation group. The postulated superiority of substrate-based ablation is it may additionally target multiple, complex isthmii and circuits otherwise missed by standard ablation. This is further supported by the added finding that a complete substrate ablation (elimination of all scar-related abnormal signals) decreases VA recurrence as compared to an incomplete substrate ablation, regardless of the approach used.

Prior studies using a pace and ablate technique

Currently, definitions of both substrate and procedural endpoints are heterogeneous in substrate-based ablation. Descriptions of abnormal substrate endpoints and ablation approaches vary, creating heterogeneity in assessing which of the two approaches is most effective. As such, endpoints for substrate-based ablation are inconsistently reported. These include complete elimination of late potentials, change of late potential activation and failure to capture with high output pacing. In a prospective study, Di Biase et al., (4) demonstrated scar homogenisation achieved by rendering scar 'electrically unexcitable' (loss of pacing capture at an output of up to 20mA with pulse duration of 10ms) significantly increased freedom from ventricular arrhythmias (VA) as compared to a standard approach. Entire substrate isolation and core isolation have also previously been shown to be a useful strategy (19, 20). This is based on the premise that high-output pacing scar tissue should not contain any viable muscle channels that could participate in scar re-entry arrhythmias. In clinical practice, however, a complete substrate-based ablation is limited by procedural duration, which can be upwards of 5 hours and has been directly linked to increased hospital mortality (21). In the current study, the ability to simultaneously ablate scar and assess a clear endpoint is not to the detriment of procedural time, eliminating a current pitfall of a comprehensive ablation approach. Indeed, a median procedural time of 246 minutes and radiofrequency ablation time of 49 minutes is comparable to other studies adapting an extensive substrate-based approach (mean of 247 minutes in a recent study by Wolf et al. (22)).

An advantage of the Ensite (NavX) EAM system is the ability to be able to simultaneously pace during ablation (Supplemental video 1 and 2); whereas this feature has been disabled within the CARTO EAM system due to a theoretical risk of inducing VF due to current leakage occurring if the pacing channel is left open during RF (23). However, the

safety after bypassing the RF generator disconnect mechanism within the CARTO system has been recently evaluated. Barbhaiya et al. (24) demonstrated a simultaneous pace-and-ablate approach was safe and, importantly, complication rate was 1.2% with no cases of VF or symptomatic cerebrovascular accidents (due to micro-bubble formation) in a short-term follow-up. We also show no instances of VF-induced during pacing using the Ensite NavX system.

Radiofrequency lesion assessment

Multiple factors influence the ablation lesion development at the catheter tip. Power delivery, ablation duration, contact force (CF), tissue characteristics (thickness, fibrosis), catheter stability (thoracic and cardiac motion). Markers of true, transmural lesion formation are required to guide catheter ablation. Unlike non-irrigated catheters where the maximum catheter tip temperature correlates with irreversible lesion formation (25), temperature measurement at the catheter tip in openly irrigated catheters does not correlate to deep tissue temperature to be used as a surrogate of lesion formation (26, 27). Bipolar electrogram (EGM) amplitude diminution of 80% is associated with a transmural lesion (28) as is creation of a positive unipolar EGM during pulmonary vein isolation (PVI) (29). However, this marker is not typically used in clinical practice due to uncertainty of catheter stability and CF which can incorrectly change EGM amplitude and morphology without an adequate lesion. Reductions in impedance during ablation are also used to guide. However, an impedance-guided approach is also limited by catheter stability as any movement will alter the local impedance measurements as will respiratory variation, necessitating forced apnoea for a reliable real-time guide.

STUDY LIMITATIONS

The experience reported in this study is drawn from a single centre with a small sample size and follow-up limits the statistical interpretation and, as such, predominantly descriptive statistics are used in this study. Despite this limitation, we outline this novel ablation lesion endpoint to demonstrate our initial experience, effect on ablation parameters and safety. A comparison group where high-output pacing protocol was not performed would have been useful to draw conclusions with the study protocol. We did not repeat pacing following loss-of-capture at each ablation site or implement a waiting period at the completion of the ablations, as this may identify pacing re-capture due to a decrease in the local oedema effect following lesion creation. Furthermore, ideally all patients undergoing this protocol would have repeat non-invasive programmed electrical stimulation (NIPS) using our induction protocol 3 days following the procedure, as this has been shown to predict a group at risk of higher VT recurrence (30).

CONCLUSION

Preliminary work suggests that the simultaneous pace-and-ablate approach reaching endpoints of tissue non-excitability, scar homogenisation and non-inducibility of VT is feasible, safe and effective. There were no complications directly related to this strategy. Acute success rate was high with 97% of all VTs non-inducible and there were no recurrences during follow-up. Combining this substrate-based technique with high-density mapping and concurrent real-time ICE to guide catheter position and ensure tissue contact, may lead to improved catheter ablation outcomes for patients with scar-related VT. Prospective studies are required to assess the long-term efficacy of this technique in larger populations including translation to all substrate types.

CONFLICT OF INTERESTS

None

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ETHICAL APPROVAL

This analysis was approved by the Western Sydney Local Health District Human Research Ethics Committee.

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TABLES LEGENDS

Induced VT match clinical	N A	Yes (VT1)	No (clinical was non-inducible)	No (clinical was non-inducible)	No (clinical was non-inducible)	Yes (VT1)	Yes (VT3)	No	N A	N A
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Table 1. Baseline Characteristics.

		Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7	Patient 8	Patient 9	Patient 10
Baseline Characteristics	Age	74	68	74	76	75	66	74	87	72	71
	Male	Male	Male	Male	Male	Male	Male	Male	Male	Male	Male
Imaging	Left ventricular ejection fraction pre-ablation, %	35	30	27	16	31	33	37	40	35	47
	LVEDD pre-ablation	68	67	65	69	72	51	60	60	72	56
	Left ventricular ejection fraction post-ablation, %	50	30	52	NA	40	NA	NA	50	37	NA
	LVEDD post ablation	61	63	49	NA	70	NA	NA	60	70	NA

	Right ventricular function pre-ablation	Normal	Normal	Severely Impaired	Normal	Moderately Impaired	Severely Impaired	Mildly Impaired	Normal	Normal	Normal
	Right ventricular function post-ablation	Normal	Normal	Severely Impaired	NA	Mildly Impaired	NA	NA	Normal	Normal	NA
Regional Wall Motion Abnormalities (hypokinesis or akinesis)	Global				X	X					
	Segmental	X	X	X			X	X	X	X	X
	Septal		X				X	X	X	X	
	Inferior	X		X			X	X		X	X
	Lateral	X	X				X	X		X	X
	Anterior		X							X	
	MRI	No	No	No	No	No	No	No	No	No	Yes (pre-ablation – normal LV function EF 36%, mild-wall LG E basal and mid-septum and inferior walls; transmural LG E anterior)

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	ICD	Yes (Single chamber)	Yes (Single chamber)	Yes (CRT-D)	Yes (dual chamber ICD)	Yes (Single chamber)	Yes (Single chamber)	Yes (Single Chamber)	Yes (Single chamber)	Yes (Dual chamber)	Yes (CRT-D)
	Scar Aetiology	Ischaemic	Ischaemic	Ischaemic	Ischaemic	Ischaemic	Non-ischaemic	Ischaemic	Ischaemic	Ischaemic	Ischaemic
Indication for VT ablation	Recurrent ICD shocks	Yes	Yes (VT storm)	No	Yes (VT storm)	No	Yes (VT storm)	Yes (VT storm)	Yes (VT storm)	Yes (VT storm)	Yes (VT storm)
	Sustained VT	No	No	Yes	No	Yes	No	No	No	No	Yes
Anti-arrhythmic therapy (pre-ablation)	Amiodarone	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes
	Sotalol	No	No	No	No	No	No	No	No	Yes	No
	Mexiletine	No	No	No	Yes	No	No	No	No	Yes	No
	B-blocker	Yes	Yes	No	No	Yes	Yes	Yes	Yes	No	No
Clinical VT recorded	RBBB	X (positive precordial concordance)	X (precordial transition V4)		X (positive precordial concordance)		X (qR in V1 with pattern break in V2)	X (precordial transition in V4)	X (positive precordial concordance)	No t rec ord ed	Not rec ord ed
	LBBB			X (negative precordial concordance)		X (late precordial transition in V5)					
	Inferior axis		X			X					
	Superior axis	X		X	X		X	X	X		
	Horizontal axis										

Table 2: Summary of Procedural Characteristics.

<i>Parameter</i>	<i>Median/Mean</i>	<i>IQR₂₅₋₇₅/SD</i>
<i>Mapping points</i>	5054	3211-15,059
<i>Bipolar scar area (cm²)</i>	65.2	50.3-94.0
<i>Unipolar scar area (cm²)</i>	90	70.3-187.2
<i>Ablation area (cm²)</i>	25.5	14.5-50.5
<i>Bipolar scar ablated (%)</i>	46	27-66
<i>Ablation lesions</i>	97	71-151
<i>VTs per patient</i>	4	2-6
<i>Cycle length of VTs (ms)</i>	376	295-482
<i>Ablation lesion duration (s)</i>	32.2	25.8-37.8
<i>Total energy (J)</i>	112,826	100,618-135,693
<i>Power (W)</i>	33.4	32.4-38.1
<i>Temperature (°C)</i>	33.3	32.0-33.7
<i>Impedance drop (Ω)</i>	9.6	8.1-10.0
<i>Impedance drop (%)</i>	8.6	7.5-9.3
<i>Maximum Impedance (Ω)</i>	102.3	100.7-110.1
<i>Minimum Impedance (Ω)</i>	94.3	87.2-100.8
<i>Contact force (g)</i>	13.0	11.9-14.6
<i>Force-Time Integral (g*s)</i>	100.0	88.8-107.6
<i>Lesion Size Index</i>	5.2	4.8-5.5
<i>Total ablation time (mins)</i>	49	45-56
<i>Procedure time</i>	246	213.8-292.5

IQR = Interquartile range, g = grams, J = joules, LVEF = left ventricular ejection fraction, ms = milliseconds, SD = Standard Deviation, W = watts, Ω = ohms

Table 3. Scar Characteristics and Ablation Parameters.

	Scar characterisation														
	Region of scar	No. of mapping points	No. of ablation lesions	Ventricular surface area (cm ²)	Bipolar scar area (cm ²)	Bipolar dense scar area (cm ²)	Unipolar scar area (cm ²)	Ablation area (cm ²)	% scar ablated						
Patient 1	Endo: Basal-mid, inferior LV	2663	101	69.4	27.1	11.2	0	22.2	82 %						
Patient 2	Endo: Apical LV	4995	165	273.8	171.6	76.7	267.9	89.4	52 %						
Patient 3	Endo: Basal-mid, inferior LV	2532	47	137.7	64.8	27.8	130.9	12.7	19 %						
Patient 4	Endo: Basal antero-septum and apex	4855	180	206	172.6	66.7	206	56.3	32 %						
Patient 5	Endo: Basal inferior LV and antero-septal RVOT (focal)	5113	92	265	101	52.9	214	80	79 %						
Patient 6	Endo: Basal-mid inferior LV extending to septal and lateral wall; Epi: patchy scar over basal infero-septal region and apex	10199	216	Endo: 114.4; Epi: 397.7	Endo: 55.9; Epi: 151.2	Endo: 18.0; Epi: 30.1	Endo: 94.1	Endo: 11.9; Epi: 10.5	Endo: 21%; Epi 6%						
Patient 7	Endo: Basal inferior LV and basal anterolateral LV	2491	67	106.6	48.4	9.7	69.9	20	41 %						
Patient 8	Endo: inferior LV scar and aneurysm extending septally and laterally	16,679	80	127.7	73.0	23	85.9	28.9	40 %						
Patient 9	Endo: anterior with an aneurysm	38,437	68	204.4	65.7	48.9	71.6	32.9	50 %						
Patient 1	Endo: basal inferior	29,653	111	117.1	20.2	5.1	53.1	12.5	62 %						

0															
	Ablation parameters														
	<i>Total ablation duration (s)</i>	<i>Avg duration per ablation lesion (s)</i>	<i>Total energy (J)</i>	<i>Avg power (W)</i>	<i>Avg temperature (°C)</i>	<i>Max temperature (°C)</i>	<i>Impedance drop (Ω)</i>	<i>Impedance Max (Ω)</i>	<i>Impedance Min (Ω)</i>	<i>Impedance drop (%)</i>	<i>Avg contact (g)</i>	<i>Max contact (g)</i>	<i>Min contact (g)</i>	<i>Force-Time Integral (gs)</i>	<i>Lesion Size Index</i>
Patient 1	3060	30.3	141,779	43 ± 13.5	26.3 ± 2.6	28.3 ± 3.3	6.1 ± 3.0	101.5 ± 10.6	95.3 ± 9.9	5.9 ± 2.8	NA	NA	NA	NA	NA
Patient 2	5616	34	188,333	32.1 ± 8.8	32.3 ± 1.3	33.3 ± 1.6	9.7 ± 4.3	110.9 ± 8.4	101.2 ± 7.3	8.6 ± 3.6	9.1 ± 5.9	28.7 ± 16.6	2.3 ± 3.3	63.8 ± 69.7	4.2 ± 1.1
Patient 3	1998	42.5	68,535	33.6 ± 4.1	33.8 ± 1.1	34.7 ± 1.4	10 ± 4.3	118.3 ± 6.0	108.2 ± 5.5	8.4 ± 3.5	11.9 ± 6.0	33.1 ± 16.4	2.5 ± 3.5	103.8 ± 119.3	4.9 ± 1.0
Patient 4	3391	18.8	115,798	32.4 ± 9.4	33.3 ± 1.0	34.4 ± 1.5	8.7 ± 3.8	92.7 ± 5.0	84 ± 4.8	9.3 ± 3.9	9.6 ± 7.0	22.8 ± 14.6	2.4 ± 3.4	52.4 ± 57.6	4.5 ± 0.87
Patient 5	2114	23	84,793	39.1 ± 7.8	31.9 ± 1.4	33 ± 1.8	9.5 ± 4.7	102.9 ± 7.4	93.4 ± 6.0	9.1 ± 4.1	11.9 ± 7.7	30.5 ± 16.8	3.2 ± 4.7	107.6 ± 113.9	5.4 ± 1.0
Patient 6	5238	24.3	174,547	31.9 ± 8.5	33.3 ± 1.5	34.8 ± 2.1	14.2 ± 8.6	100.4 ± 17.1	86.1 ± 11.4	13.5 ± 6.2	14.1 ± 9.11	36 ± 21.7	4.6 ± 6.6	120.6 ± 127.7	5.2 ± 1.2
Patient 7	2859	42.6	99,368	33.2 ± 9.0	31.2 ± 1.1	32.3 ± 1.5	9.8 ± 4.6	112.4 ± 7.3	102.6 ± 6.8	8.6 ± 3.8	13.0 ± 4.4	36.7 ± 21.6	3.3 ± 4.4	88.8 ± 115.5	4.8 ± 1.2
Patient 8	2870	35.9	109,853	37.1 ± 7.0	34.3 ± 2.1	35.5 ± 2.5	6.6 ± 3.6	90.8 ± 6.7	84.2 ± 6.1	7.2 ± 3.8	14.8 ± 7.9	35.7 ± 21.2	4.4 ± 5.0	93.5 ± 99.7	5.9 ± 1.1
Patient 9	2615	38.4	104,370	38.4 ± 8.0	33.4 ± 3.2	34.4 ± 3.5	7.9 ± 4.9	107.5 ± 11.7	99.7 ± 10.3	7.2 ± 4.1	14.6 ± 10.3	39.7 ± 26.8	3.6 ± 5.2	99.9 ± 123.0	5.5 ± 1.2
Patient 10	3385	30.5	117,437	32.5 ± 9.8	35.4 ± 5.3	36.8 ± 5.8	10.9 ± 6.2	101.7 ± 15.9	90.8 ± 14.0	10.4 ± 5.5	16.5 ± 10.2	38.9 ± 20.6	5.1 ± 6.1	134.2 ± 148.3	5.5 ± 1.1

Table 4. Acute Procedural Outcomes and Follow-up.

		Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7	Patient 8	Patient 9	Patient 10
Acute procedures	Success	No VT induction performed post ablation due to risk of haemodynamic compromise in the context of moderate-severe AS	No	Non-inducible with PES up to 4 ES in the baseline state and with burst pacing down to RV ERP 250ms and with up to 40mcg/min of isoprenaline	Non-inducible with PES up to 4 ES in the baseline state and with burst pacing down to RV ERP 250ms and with up to 40mcg/min of isoprenaline	Non-inducible with PES up to 4 ES in the baseline state and with burst pacing down to RV ERP 250ms and with up to 40mcg/min of isoprenaline	Non-inducible with PES up to 4 ES. At the end of the final ablation no VT was inducible with 3 induction (RV x2, epicardial x 1) in the baseline state and with burst pacing down to RV ERP 250ms and with up to 40mcg/min of isoprenaline at peak HR and in recovery. Only VF was inducible that was reverted to SR (from AF also) with an external shock of 200J.	Non-inducible with PES up to 4 ES in the baseline state and with burst pacing down to RV ERP 250ms and with up to 40mcg/min of isoprenaline. Only VF was inducible requiring cardioversion 200J to SR.	Non-inducible with PES up to 4 ES in the baseline state and with burst pacing down to RV ERP 250ms and with up to 40mcg/min of isoprenaline. Only VF was inducible requiring cardioversion 200J to SR.	Non-inducible with PES up to 4 ES in the baseline state and with burst pacing down to RV ERP 250ms and with up to 40mcg/min of isoprenaline. Only VF was inducible requiring cardioversion 200J to SR.	Non-inducible with PES up to 4 ES in the baseline state and with burst pacing down to RV ERP 250ms and with up to 40mcg/min of isoprenaline. Only VF was inducible requiring cardioversion 200J to SR.

			targeted)								
	Failure	No	No	No	No	No	No	No	No	No	No
	Procedural complications	Retroperitoneal haematoma (conservatively managed)	None	None	None	None	None	None	None	None	None
Follow-up	Duration (days)	370	410	360	7	306	78	68	285	8	64
	VT recurrence	No	No	No	Died of non-procedural related complications 7-days post ablation	No	No (including NIPS post ablation - non-inducible VT)	No	No	No	No
	Hospital readmission	No	No	No	Yes	No	No	No	No	No	No
	Change in AAD therapy	Amiodarone ceased	None	Amiodarone ceased, sotalol commenced	None	None	Amiodarone ceased	Amiodarone ceased	Amiodarone ceased	Amiodarone ceased	Amiodarone ceased

Figure 2. An example of simultaneous pacing during substrate-based catheter ablation to loss-of-capture (non-excitability). A 74-year-old with severe ischaemic cardiomyopathy and episodes of sustained VT underwent a catheter ablation procedure. Non-clinical VT1 was induced with PES with a RBBB V1 (transitioning to a rS in V3)/superior axis morphology (Panel A). High-density mapping identified a large basal-mid inferior scar which was targeted with substrate-based ablation using bipolar pacing (guided by CF) at an output of 10mA at 9ms pulse width until loss-of-capture (Panels B-E).

Figure 3. A 74-year-old with ischaemic cardiomyopathy (LVEF = 35%) presented with recurrent ICD shocks associated with a RBBB/superior axis clinical VT morphology. VT induction was not performed in the context of risk of haemodynamic compromise given the patients history of severe aortic stenosis. A substrate-based catheter ablation was performed and scar homogenisation of large basal inferior substrate (Panel A). In addition to elimination of all late and fractionated potentials, high-output pacing was performed (10mA at 9ms pulse width) during ablation to achieve scar non-excitability as an additional ablation endpoint (Panel B).

Figure 4. A 74-year-old with ischaemic cardiomyopathy (LVEF = 37%) presented with electrical VT storm with a clinical VT demonstrating a RBBB (transition in V4)/superior axis morphology. 5 VT morphologies were induced during mapping and catheter ablation (VT1-5, Panel A) with VT3 matching the clinical VT (Panel B). EAM showed extensive basal inferior and antero-lateral low voltage (Panels C and D). Substrate-based ablation was performed to abnormal late potentials with this scar utilising simultaneous high-output pacing (10mA at 9ms pulse width) to achieve scar non-excitability. Impedance recordings during pace and ablation demonstrate ~10ohm reduction at 40W power (without any pacing interference, Panel E). Examples of ablation with initial pace-capture to loss-of-capture were demonstrated, including pacing delay prior to loss-of-capture (Panel F). Both high-density mapping and ablation sites were guided by real-time ICE, which could also indicate underlying lesion formation during ablation (Panel G).

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