Ventricular tachycardia ablation in structural heart disease: impact of ablation strategy and non-inducibility as an end-point on long term outcome.

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Abstract

Background: We sought to investigate the long term outcomes after catheter ablation (CA) of ventricular tachycardia (VT) in the context of structural heart disease in a large multicenter cohort. The impact of different ablation strategies (substrate ablation versus activation guided versus combined) and non-inducibility as an end-point was evaluated.

Methods: Data was pooled from prospective registries at 5 centres over a 5 year period. Success was defined as survival free from recurrent ventricular arrhythmias (VA). Multivariate analysis of factors predicting survival free from VA was by Cox regression.

Results: Five hundred sixty-six patients underwent CA for VT. Patients were 64 ± 15 years, 72 % were male. Left ventricular ejection fraction was 35 ± 15 % and 66 % had ischaemic heart disease. At 2.3 (IQR 1.0 - 4.2) years, success was achieved in 44 % after a single procedure, rising to 60% allowing for repeat procedures. Mortality at final follow up was 22 %. Multivariate analysis showed that higher left ventricular ejection fraction (HR 0.989, 95 % CI 0.981 - 0.998, $p = 0.014$), younger age (HR 1.012, 95 % CI 1.002 - 1.012, $p = 0.020$), ischaemic heart disease (HR 0.587, 95 % CI 0.440 - 0.783, $p < 0.001$), and non-inducibility of VA (HR 0.700, 95 % CI 0.552 - 0.888, $p < 0.003$) predicted long term survival free from VA (all $p < 0.05$). There was no impact of the approach to ablation.

Conclusion: CA eliminates VT in a large proportion of patients long term. Ablation strategy did not impact outcome and hence substrate ablation is a reasonable initial strategy. Non-inducibility of VA predicted survival free from VA and may be worth pursuing as a procedural end-point.

Introduction

Ventricular arrhythmias (VA) cause significant morbidity and mortality in patients with structural heart disease (SHD). Implantable cardioverter-defibrillators (ICD) prevent sudden cardiac death but do not prevent recurrent VA. Management of patients with recurrent VA and ICD shocks remains a challenge. Shocks are unpleasant and these patients are prone to VT storm, have increased heart failure hospitalisations and higher mortality (1-5). Antiarrhythmic drugs (AAD) reduce VA burden but can have significant adverse effects and are not always effective (6, 7).

Catheter ablation (CA) of ventricular tachycardias (VT) is being utilised increasingly in the context of SHD (8, 9). Increasingly, approaches to CA are based partly or wholly on substrate modification, whereby surviving myocardial fibres within areas of scar are ablated to interrupt critical isthmi for re-entrant VT (9-11). CA reduces the burden of VT and ICD shocks (12-14). Recent data suggests that CA is superior to AADs in terms of preventing recurrent VA, ICD therapies, and possibly reducing mortality (15, 16).

There are limited data investigating the long-term outcome after CA of VA in SHD and the available published data originates mostly from a small number of world leading centres (11, 14, 17, 18). Furthermore, there remains uncertainty as to the safest and most effective approach as well as the usefulness of procedural end-points such as non-inducibility of VA. The present study investigated the impact of VT ablation on long term outcome in a large cohort of patients with SHD of mixed aetiologies in a multi-centre registry. The impact of ablation strategy and non-inducibility of VA as a procedural end-point was assessed in terms of (i) procedural safety, (ii) long tern efficacy, and (iii) long term mortality.

Methods

Study design and patient sample

A multicenter registry was compiled from a collaborative group of UK tertiary centres experienced in VT ablation. Independent prospective registries were held for consecutive patients undergoing catheter ablation of VT, including baseline demographics, procedural data, complications and follow-up. All consecutive patients were included over a 5 year period: 01/01/2010 - 31/12/2014.

Of patients undergoing catheter ablation of VT the sole inclusion criteria was structural heart disease which was defined as significantly impaired left ventricular function (ejection fraction < 45 %) or other confirmed structural abnormality. Patients were included regardless of the aetiology. Patients with channelopathies were not included.

The peri-procedural management, procedural techniques, and follow-up varied between centres, although there were certain commonalities as described below.

Ablation procedure

All procedures were performed either under conscious sedation or under general anaesthesia. Systemic intravenous anticoagulation using heparin was administered when mapping the left ventricle endocardialy with a target ACT of 300 - 350 seconds. Access to the arterial and venous system was routinely from the right groin. The route to access the endocardial left ventricle was at the operator's discretion and involved either the transseptal route, retrograde access through the arterial system, or both. Epicardial access was obtained where necessary using a Seldinger

approach. Where it was thought necessary, epicardial access was sought at the index procedure and this was not deferred to a second or staged procedure.

Electroanatomic mapping systems were used with irrigated catheters to deliver radiofrequency energy in all cases. Multipolar mapping catheters, steerable or robotic sheaths, and contact-force sensing ablation catheters were used at the discretion of the operator when they were available. Power settings varied from 30-50 Watts with a temperature limit of 42 - 48 °C.

Ablation strategy

The approach to ablation of VT was at the discretion of the treating physician and included both substrate based modification performed in sinus rhythm or activation mapping of VT, or both. Substrate modification involved voltage mapping in sinus rhythm with identification of sites of late activation as demonstrated by split, fractionated or isolated late potentials which were targeted for ablation (19, 20). Ablation was delivered focally to abolish these signals, often in clusters or lines, but there was no attempt to create lines of block or to isolate areas of tissue.

Activation mapping was performed for either spontaneous or induced VT. In cases where activation mapping was performed, the aim was to identify and ablate the diastolic pathway, or failing this to ablate the exit site. Pace mapping was utilized in many patients as an adjunct to activation mapping to help to localize the VT exit site (21).

Programmed ventricular stimulation and non-inducibility of VA as a procedural end-point

All 5 centres participating in this study routinely perform programmed ventricular stimulation at the end of VT ablation procedures. This was not performed in all cases, for example where there was no realistic prospect of having achieved non-inducibility, or where patients were unwell and unlikely to tolerate further VT/cardioversion well. Otherwise programmed ventricular stimulation was performed with the rationale that it might (a) reveal a mappable VT that could be targeted, (b) prompt the operator to map and ablate epicardialy if they had not already done so, or (c) provide prognostically useful information. Programmed ventricular stimulation was routinely performed from two different locations with two different cycle lengths and three successive extrastimuli at decreasing intervals until reaching ventricular refractoriness.

Follow-up and endpoints

A majority of patients were followed up in the ICD clinic. Patients without implanted devices were followed up in conventional outpatient clinics. Patients who had not been seen recently were contacted for follow up data. Where patients could not be contacted, follow up was taken from the last point of contact.

It was not thought possible to eradicate all VA over long term follow up including non-sustained episodes. Furthermore, much of the study period was before the landmark MADIT-RIT trial, and hence use of ATP was initially more liberal(22). We therefore included a combined end-point of death or recurrent VA, with recurrent VA (including both VT and VF) defined as (i) receiving an appropriate ICD shock for VA, (ii) VA causing a hospital admission, or (iii) VA requiring a change in anti-arrhythmic drug treatment or requiring catheter ablation. Episodes of non-sustained VT not meeting these criteria, including those treated with ATP, were not counted as failure.

Statistical analysis

Continuous variables are reported as mean ± standard deviation, or median (range or interquartile range where stated) if not normally distributed. Continuous data were compared by Student's t-test if normally distributed or Man-Whitney U test if not normally distributed. Categorical data were compared by chi-squared test. Kaplan-Meier curves were used to analyse survival free from VA. Groups were compared using the log-rank test.

Multivariate analysis of factors predicting long term mortality and a composite of recurrent VT or death was by Cox regression and included the following factors: Age, gender, left ventricular ejection fraction, ischaemic heart disease, dilated cardiomyopathy, use of contact force sensing catheters, substrate guided ablation only as an ablation strategy, activation mapping only as an ablation strategy, or an ablation strategy incorporating both activation and substrate guided ablation, and demonstration of non-inducibility of VA. These were all included as categorical covariates, with the exception of age and left ventricular ejection fraction which were included as continuous covariate. Variables were then removed stepwise from the model when the p-value exceeded 0.10, and variables with $P < 0.05$ in the final model were considered to be significant predictors of recurrent VA and death. A similar analysis of factors predicting 30 day mortality was performed using binary logistic regression. Analysis was performed using SPSS 16 (SPSS,Inc., Chicago, IL).

Results

Study population

Baseline characteristics of the patients are summarized in Table 1. Five hundred sixty-six patients underwent CA for VA. Patients were aged 64 ± 15 years and the majority (72 %) were male. The aetiology of structural heart disease was varied and included ischemic heart disease in 66 %, dilated cardiomyopathy in 14 % and other cause in 21 % (Table 1). Mean left ventricular ejection fraction (LVEF) was 35 ± 15 %. An implantable cardioverter-defibrillator (ICD) or a cardiac resynchronisation device with an ICD function (CRT-D) had been implanted prior to the first catheter ablation in 78 % of the patients. More than three quarters of the study population were on pharmacological treatment with beta-blockers and ACE-inhibitors/angiotensin-receptor blockers at baseline and 43 % were already taking amiodarone.

Procedure characteristics

566 patients underwent a total of 761 catheter ablation procedures for VT (Table 2). Of these, 74 % of patients had one procedure only, 19 % underwent a second procedure, 5 % underwent a third, and less than 2 % of patients underwent 4 or more procedures. In 333/566 patients (59 %), the procedure was performed on an urgent basis on patients who had been admitted to hospital with VA, whereas in 233/566 (41 %) the procedure was performed on an elective basis. Procedure duration was 189 ± 68 minutes and fluoroscopy time was 25 ± 20 minutes. 379/566 patients (67 %) had access to the endocardial left ventricle via a transseptal approach whereas 273/566 (48 %) had a retrograde, transaortic approach. Epicardial access was obtained in

52/566 patients (9 %). A contact force catheter was used in a third of the patients (33 %).

Ablation strategy and non-inducibility as an end-point

The approach to CA was guided solely activation mapping in 146/566 subjects (26 %, Table 2), solely substrate ablation in 163/566 patients (29 %), and a combination of these two approaches in 257/566 patients (45 %). Programmed ventricular stimulation to induce VA was performed at the end of the procedure in 398/566 patients (70 %). Non-inducibility of VA was demonstrated in 322/566 patients (57 %). Non-inducibility of VA was achieved in 56 of 146 patients (39 %) where ablation was guided by activation mapping alone, compared to 71/163 patients (44 %) where ablation was substrate guided, and 195 of 257 patients (76 %) where a combination of both approaches was utilized (combined approach compared to both activation only and substrate only approaches both P < 0.001; activation only versus substrate only P = 0.358).

Success following VT ablation

The single procedure success rate using the composite endpoint of death or recurrent VA was 61 % at 1 year (Figure 1A & 1B) and 44 % at a final follow up of 2.3 years (IQR 1.0 - 4.2 years). Allowing for repeated procedures the success rate increased to 78 % at 1 year and 60 % at final follow. A total of 127 patients (22.4 %) died during follow up (Figure 1C). Success at final follow up was achieved in 71/163 (44 %) who had substrate guided ablation only (HR on univariate analysis 0.99, 95 % CI 0.78 - 1.27, P = 0.957) compared to 62/146 (43 %) of those who had activation guided ablation only (HR 0.97, CI 0.76 - 1.25, P = 0.814) and 115/257 (45 %) of those who had a combined approach (HR 1.03, CI 0.83 - 1.28, P = 0.798). In patients

in whom non-inducibility of VA was achieved at the end of the procedure long term success was achieved in 161/322 (50 %) patients (HR on univariate analysis 0.70, CI 0.56 - 0.87, *P* = 0.001).

Predictors of recurrent VT and death

Figure 2A shows a multivariate analysis of the factors predicting the primary end point of recurrent VA or death; notably there was no impact of the approach to ablation although the impact of non-inducibility of VA was significant (for Kaplan-Meier analyses see Figures 1A and 1B). After stepwise removal of factors with a *P* > 0.10, those relevant factors remaining were increasing age (HR 1.012 for each year beyond the mean, 95 % CI 1.002 - 1.012, *P* = 0.020), higher LVEF (HR 0.989 for each percentage point increase in ejection fraction beyond the mean, 95 % CI 0.981 - 0.998, *P* = 0.014), ischemic heart disease (HR 0.587, 95 % CI 0.440 - 0.783, *P* < 0.001), and non-inducibility of VA (HR 0.700, 95 % CI 0.552 - 0.888, *P* = 0.003) at the end of the procedure. Notably, there was no impact of the ablation strategy (in terms of substrate guided ablation, activation guided ablation or both) on outcome. There was a trend towards reduction in the primary end point with the use of contact force sensing catheters (HR 0.815, 95% CI 0.630 - 1.055, *P* = 0.121).

Predictors of death following VT ablation

Figure 2B shows a multivariate analysis of the factors predicting death over long term follow up after VT ablation. Notably there was no impact of the approach to ablation, although the impact on non-inducibility of VA was significant (see also Kaplan Meier analyses in Figure 1C). After stepwise removal of factors with a *P* > 0.10 those relevant factors remaining were age (HR 1.051 for each year beyond the mean of the cohort, 95 % CI 1.032 - 1.071, *P* < 0.001), LVEF (HR 0.972 for each percentage point

increase in ejection fraction beyond the mean, 95 % CI 0.956 - 0.988, *P* < 0.001), the presence of dilated cardiomyopathy (HR 1.932, 95 % CI 1.180 - 3.165, *P* = 0.009), non-inducibility of VA (HR 0.538, 95 % CI 0.369 - 0.784, *P* < 0.001) and the use contact force sensing catheters (HR 0.551, 95 % CI 0.360 - 0.843, *P* = 0.004).

Procedural complications and mortality

Major complications occurred in 12.0 % of patients (Table 2). A significant proportion of these were made up of haematoma at the access site (1.8 %) and tamponade requiring drainage in 3.5 %. Both atrioventricular block and TIA/stroke were infrequent at 0.5 % each. The mortality rate was low at 24 hours (0.5 %) although this increased to 2.7 % when including all deaths up to 30 days. Notably, all the deaths occurred in patients who underwent the CA procedure on an urgent basis having been admitted to hospital for VA (333 of 566 patients, 59 %). In the 233/566 patients (41 %) who had their procedure performed electively there were no deaths within 30 days. If considered separately this would give a 30 day mortality of 0 % when VT ablation was performed electively compared to 4.5 % (15/333) when performed as an emergency (*P* < 0.001).

Predictors of 30 day mortality

Figure 2C shows a multivariate analysis of the factors predicting 30 day mortality after VT ablation. After stepwise removal of factors with a *P* > 0.10 those remaining were higher age (HR 1.078, 95 % CI 1.013 - 1.1347, *P* = 0.012) and lower LVEF (HR 0.904, 95 % CI 0.839 - 0.974, *P* = 0.008), ischaemic heart disease (HR 0.11, 95% CI 0.03 - 0.51, *P* = 0.005), and dilated cardiomyopathy (HR 0.18, 95% CI 0.03 - 1.31, *P* = 0.091), and non-inducibility of the VT (HR 0.134, 95 % CI 0.028-0.638, *P* = 0.022). There was no impact of the approach to ablation in the final model.

Discussion

This multicentre registry of VT ablation in patients with SHD is one of the largest and has one of the longest follow up periods reported to date. Survival free from VA was similar to that reported by others in the short term, but attrition continued over time and 44 % remained alive and free from VA after a single procedure or 60% if allowing for repeat procedures at a median of 2.3 years. The major complication rate including events up to 30 days was higher than reported by most at 12 %, and although mortality within 24 hours was low at 0.5 % this increased to 2.7 % at 30 days; notably all deaths occurred in the 59 % of patients who had been admitted to hospital with VA and having procedures as an emergency. The approach to ablation in terms of being guided by substrate mapping, activation mapping or a combined approach had no effect on long term success, long term mortality, or 30 day mortality. Non-inducibility of VA at the end of the procedure was associated with a higher long term success rate, lower long term mortality, and lower 30 day mortality.

Outcome after VT ablation

Data on the efficacy of CA in the context of SHD derives from prospective and retrospective analysis demonstrating variable outcome with arrhythmia-free survival ranging from 30 - 71 % (8, 11, 14, 17, 23). Two randomised trials comparing catheter ablation to AADs at the time of ICD implantation for VT in the context of ischaemic heart disease found survival free from VA in 47 % and 79 %, both at 1.9 years (11, 14). The recent VANISH trial was the largest trial yet in VT ablation, randomising 132 patients with ischemic heart disease to ablation; they reported a VA free survival of 41 % at 2.3 years (15). The results achieved in this study are comparable to these smaller randomised studies with survival free from VA after a single ablation procedure of 44 % at 2.3 years, rising to 60 % if allowing for repeat procedures.

Whilst it is reassuring that similar results can be achieved outside of the controlled conditions and selected patients in a randomised trial, there is still certainly scope to improve outcomes with new techniques and technologies. Furthermore, 22 % of patients died during follow-up, highlighting the need for ongoing treatment of the underlying heart disease, management of heart failure and selection for other therapies such as transplant or ventricular assist devices.

Impact of non-inducibility of VA

Non-inducibility of VA at the end of the procedure has been shown to predict freedom from VA and survival in a small number of studies, albeit mostly in the context of ischaemic heart disease (9, 15, 24-27). In one single centre registry of 160 patients with ischemic heart disease, non-inducibility predicted freedom from VA and survival (25). Similarly, in the multicentre Thermocool Ventricular Tachycardia Ablation Trial which included 231 post-myocardial infarction patients, non-inducibility was highly predictive of freedom from VT recurrence (9). These studies were included in a recent meta-analysis of 736 patients undergoing VT ablation which found a favourable effect of non-inducibility(27). Similarly, inducibility of VA during an electrophysiological study in ICD patients from the Multicenter Automatic Defibrillator Implantation Trial (MADIT) II was associated with a greater likelihood of ICD therapies for VA (28).

The current study reports outcomes in almost as many patients as the recent meta-analysis over a longer period of follow up and confirms a protective effect of non-inducibility in a mixed cohort of patients with ischaemic and non-ischaemic aetiologies. Non-inducibility of VA was found to be one of the strongest predictors of 30 day mortality, long term mortality and freedom from VA. It is recognized that this

association may also reflect the extent of underlying cardiac pathology which may convey some of the prognostic advantage with non-inducibility. However, the size of this cohort has allowed a meaningful multivariate analysis to control for other factors so far as is possible. Non-inducibility of VA was found to independently predict freedom from VA and also all-cause mortality.

Taken together with the findings from these other studies, our data suggests that non-inducibility of VA is desirable and is reasonable to pursue as a procedural end-point. Nevertheless, Randomised controlled trials are needed to confirm whether pursuing this as an outcome really improves outcomes significantly. Furthermore, non-inducibility can be difficult to achieve in the context of advanced heart disease and it remains to be seen whether aggressive approaches utilizing haemodynamic support are warranted(29).

Impact of approach to ablation

Although activation mapping offers the chance to visualize and target a re-entry-circuit, the VT needs to be sustained, consistent and haemodynamically tolerated. Ablation may only eliminate one of many potential VTs. Maintaining VT in these frail patients may also be dangerous. Several studies have utilized a substrate-based VT ablation technique targeting channels within scar in sinus rhythm, often with good effect and with very low complication rates (11, 14, 19, 20, 30-33). Although substrate based ablation is intended as a 'gentler' approach, since ablation can be performed in sinus rhythm without the need to sustain VT, there is concern that this may lead to more extensive ablation in patients with already poor ventricular function and also may miss critical areas. There are currently little data comparing the safety and

efficacy of these techniques and no consensus as to the ideal approach to VT ablation.

A meta-analysis of 6 studies including 403 patients reported no difference in outcomes with either approach(34). A randomised trial comparing these 2 approaches in 118 patients suggested better outcomes with an aggressive substrate modification approach (31). This study is the largest yet to compare the safety and efficacy of these ablation strategies comprising more patients than the sum of these previous studies and testing this across multiple centres and aetiologies. There was no difference in the procedural safety comparing activation and substrate mapping. There was no difference in long term survival free from VA between patients having solely substrate based ablation versus ablation guided by activation mapping or a combined approach which was confirmed on multivariate analysis.

The decision to adopt one or other approach will have been guided by operator preference and specifics of the case. It is probable that many cases started with one approach before 'crossing over' to a combined approach when the patient was still found to be inducible, potentially disadvantaging the combined approach group. Nevertheless, this does suggest that substrate ablation as an initial standalone strategy is not inferior to ablation guided by activation mapping or a combined approach, accepting that cross over may be required to achieve non-inducibility.

These data would support substrate based ablation as an initial approach followed by activating mapping if VT remains inducible. Equally though, we have found no evidence of a detrimental impact of an approach utilizing activation mapping in appropriately selected patients.

Other factors predicting outcome

A higher LVEF and younger age predicted 30 day survival, long term survival and survival free from VA. The association between poor LV function and a worse outcome has been demonstrated previously (14, 17, 18, 35), and the link with age is not surprising. Ischemic heart disease predicted 30 day survival, long term survival free from VA, but not long term survival, perhaps suggesting that VA may be more straight forward to eliminate with this aetiology but that death may still occur in a significant proportion due to disease progression. Scarring in non-ischemic heart disease may be less extensive but is often patchy and located epicardialy or mid-myocardialy (36-38). This limits the chances of successful ablation endocardialy (39). Furthermore, there may also be a higher incidence of seemingly focal VT in non-ischemic heart disease (40). Studies comparing outcome of VT ablation in patients with IHD and non-ischemic aetiologies favour the outcome in IHD (41-43).

There was a trend towards improved long term survival free from VA when contact force sensing catheters were used, which reached significance when looking at crude survival. Taken together this suggests the use of contact force sensing catheters may improve outcome. This is plausible since achieving lesions that are more than superficial requires good contact.

Notably all deaths within 30 days occurred in patients admitted to hospital with VA having emergency procedures, giving a mortality of 4.5 % in this patient group. This cohort is likely to be sicker than those undergoing elective VT ablation and may be under represented in randomised trials. Another large multicentre registry reported a similar mortality (5 %) (44). Centres performing VT ablation should be aware of this, should anticipate problems and manage patients in a high dependency setting.

However, patients undergoing elective VT ablation had a 0 % mortality. Taken together with the recent VANISH trial, these data raise the question as to whether VT should be ablated early prior to developing VT storm (15).

Limitations

Registry data can be incomplete and may be biased. Nevertheless the data were collected prospectively and the complication rates and success rates are very comparable to other prospective studies and trials. Although cases were categorised based on the ablation approach it is likely that many 'crossed over' to the combined approach group particularly if VT were inducible on programmed electrical stimulation. The association between non-inducibility of VA and improved outcomes may relate partly to the degree of underlying heart disease and it is recognised that multivariate analysis may not fully account for confounding factors. Randomised trials are needed to investigate the impact of different ablation approaches and to determine whether striving for non-inducibility as an end-point improves outcome.

Conclusions

Achieving non-inducibility of VA as a procedural end-point seems much more important than how it was achieved in terms of the ablation strategy. This therefore supports a strategy of substrate ablation followed by programmed electrical stimulation, proceeding to activation mapping only where needed to achieve non-inducibility. The 30 day mortality following VT ablation for electrical storm is appreciable and highlights the need for aggressive care in this sub-group. Early ablation of VA in an elective setting is safer and should be considered. The long term mortality is significant in these sick patients and they are likely to benefit from ongoing treatment of their underlying heart disease and heart failure.

Table 1. Patient demographics

Number of patients [N]	566
Age [years]	64 ± 15
Male/Female gender [%]	72 / 28
Underlying heart disease	
Ischaemic heart disease	373 (66 %)
Prior stenting	162 (29 %)
Prior CABG	132 (23 %)
Dilated cardiomyopathy	77 (14 %)
Sarcoid-associated cardiomyopathy	12 (2 %)
Arrhythmogenic ventricular cardiomyopathy	29 (5 %)
Hypertrophic cardiomyopathy	11 (2 %)
Other	64 (11 %)
Left ventricular ejection fraction (LVEF)	35 ± 15 %
ICD device	
ICD	308 (54 %)
CRT-D	136 (24 %)
No defibrilator	122 (22 %)
Hypertension	185 (33 %)
Type 2 diabetes	80 (14 %)
eGFR	68 ± 26
Beta-blocker	474 (84 %)
ACE-I or ARB	429 (76 %)
Sotalol	59 (10 %)
Calcium channel blocker	21 (4 %)
Flecainide	9 (2 %)
Amiodarone	244 (43 %)
Anticoagulation	183 (32 %)

Table 2: Procedural data

Procedure number	1 (IQR 1-3)
1 procedure	420 (74 %)
2 procedures	110 (19 %)
3 procedures	27 (5 %)
4 procedures	6 (1 %)
5 procedures	2 (0.4 %)
6 procedures	1 (0.1 %)
Procedure time	189 ± 68 min
Fluoroscopy time	25 ± 20 min
Radiation dose	2507 ± 3885 cGycm^2
Access	
Transseptal puncture	379 (67 %)
Retrograde	273 (48 %)
Epicardial	52 (9 %)
Contact force sensing catheters	189 (33%)
Activation guided ablation only	146 (26 %)
Substrate guided ablation only	163 (29 %)
Activation and substrate guided ablation	257 (45 %)
VT stimulation study at end of procedure	398 (70 %)
VT non-inducibility demonstrated	322 (57 %)
Major Complications	
Haematoma	10 (1.8%)
Tamponade	20 (3.5%)
Stroke or TIA	3 (0.5%)
Complete heart block	3 (0.5%)
Other major	20 (3.5%)
Death within 24 hours	5 (0.8%)
Death within 30 days	15 (2.7%)
Any major complication upto 30 days	68 (12.0%)

Figure legends

Figure 1A:Survival free from ventricular arrhythmia by ablation strategy.

Legend: Kaplan-Meier curve shows survival free from VA after a single procedure. The number at risk is shown at the bottom with lables showing the success rate at 1 year and at final follow up. The impact of ablation strategy is shown by the different lines with groups compared using the Log Rank test.

Figure 1B:Survival free from ventricular arrhythmias and the impact of non-inducibility.

Legend: Kaplan-Meier curve shows survival free from VA following a single ablation procedure. The number at risk is shown at the bottom with lables showing the success rate at 1 year and at final follow up. The impact of non-inducibility is shown by the different lines with groups compared using the Log Rank test.

Figure 1C: Survival following ablation for VT and the impact of non-inducibility.

Legend: Kaplan-Meier curve shows survival following VT ablation. The number at risk is shown at the bottom. The text beneath the curve states the mortality at 24 hours, at 30 days, and at final follow up. The impact of non-inducibility is shown by the different lines with groups compared using the Log Rank test.

Figure 2A: Multivariate analysis of factors predicting failure following a single VT ablation procedure.

Legend: Multivariate analysis of factors predicting the combined end-point of recurrent VA or death. The figure shows hazard ratio and 95% confidence intervals.

Figure 2B: Multivariate analysis of factors predicting death during long term follow up.

Legend: The figure shows hazard ratio and 95% confidence intervals.

Figure 2C: Multivariate analysis of factors predicting 30 day mortality after VT ablation.

Legend: The figure shows hazard ratio and 95% confidence intervals.

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