Influence of baseline inducibility and activation mapping on ablation outcomes in patients with structural heart disease and ventricular tachycardia

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Abstract

Introduction:Stand-alone substrate ablation without baseline ventricular tachycardia (VT) induction and activation mapping has become a standard VT ablation strategy. We sought to evaluate the influence of baseline VT inducibility and activation mapping on ablation outcomes in patients with structural heart disease (SHD) undergoing VT ablation. Methods:This is a single center, observational and retrospective study including consecutive patients with SHD and documented VT undergoing ablation. Baseline VT induction was attempted before ablation in all patients and VT activation mapping performed when possible. Ablation was guided by activation mapping for mappable VTs plus substrate ablation for all patients. Ablation outcomes and complications were evaluated. Results: 160 patients were included (203 VT ablation procedures) and were classified in 3 groups according to baseline VT inducibility: group 1 (non inducible, n=18), group 2 (1 VT morphology induced, n=53), and group 3 (>1VT morphology induced, n=89). VT activation mapping was possible in 35%. After a median follow-up of 38.5 months, baseline inducibility of >1VT morphology was associated with a significant incidence of VT recurrence (42% for group 3 vs. 15.1% for group 2 and 5.6% for group 1, Log-rank p<0.0001) and activation mapping with a lower rate of VT recurrence (24% vs. 36.3%, Log-rank p=0.035). Independent predictors of VT recurrences and mortality were baseline inducibility of >1VT morphology (HR 12.05 IC 95% 1.60-90.79, p=0.016) and LVEF<30% (HR 2.43 IC 95% 1.45-4.07, p=0.001), respectively. Complications occurred in 11.2% (5.6% hemodynamic decompensation). Conclusions:Baseline VT inducibility and activation mapping may add significant prognostic information during VT ablation procedur

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Running title: **Ventricular tachycardia ablation in structural heart disease**

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Conflict of interest

Dr. O. Cano has received consultant fees from Biosense Webster. All other authors have nothing to disclose.

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Conclusions : Baseline VT inducibility and activation mapping may add significant prognostic information during VT ablation procedures.

Key words: ventricular tachycardia; ablation; activation mapping; substrate mapping

Introduction

Ventricular tachycardia (VT) ablation strategies in patients with structural heart disease (SHD) have significantly evolved during the last years. Substrate ablation with complete elimination of the potentially arrhythmogenic substrate has become a standard treatment in this setting and has been related to better acute success rates and reduction of VT recurrences during follow-up[1-8]. This strategy is usually preferred for patients with hemodynamyic instability during induced VT, advanced heart failure with poor hemodynamic condition or non-inducible patients at the time of the procedure. However, stand-alone substrate ablation has also been advocated as a first ablation strategy even in the absence of these previous conditions thus obviating the evaluation of baseline VT inducibility and the potential utility of VT activation mapping[9-13]. We sought to evaluate the role of baseline VT inducibility and the influence of activation mapping on outcomes in patients with SHD undergoing VT ablation.

Methods

This is a single-center, observational retrospective study including consecutive patients with SHD and documented sustained monomorphic VT (SMVT) referred to our center for VT ablation. All patients undergoing VT ablation in our center are prospectively included in a dedicated database. Baseline clinical, imaging and analitics data as well as procedural and follow-up details are included in this database. Patients were included in the final data analysis only if a baseline programmed ventricular stimulation (PVS) protocol aiming for VT induction was performed after substrate mapping and before ablation. All patients gave informed consent and the study was carried out following the recommendations of the ettical committee of the Hospital Universitari i Politècnic La Fe.

Workflow

The procedures were performed either under general anesthesia or under concious sedation with propofol and fentanyl. Endocardial LV mapping was preferentially performed by a transseptal access although the retroaortic approach was also used when considered necessary. For those cases in which an epicardial involvement was suspected, an epicardial access was obtained using the technique described by Sosa et al.[14]

A complete substrate mapping of the chamber/s of interest was initially obtained during the baseline patient's rhythm and using either a linear (Smart-Touch, Biosense Webster) or a multielectrode catheter (PentaRay, Biosense Webster). Low voltage areas as well as late potentials were registered and tagged. Scar was defined as areas with bipolar voltage < 0.5 mV. Upon completion of the substrate mapping, a standardized PVS protocol was used to induce VT in all patients included in the study. This protocol consisted in a 8 beats basal train at three different cycle lengths (600, 500 and 400 ms) followed by up to 4 extraestimmuli with a minimun coupling interval of 200 ms or until ventricular refractoriness from the right ventricular apex, right ventricular outflow tract, or different LV sites. If a stable and hemodynamically tolerated SMVT was induced, activation mapping of the VT was performed and entrainment maneuvers were used whenever possible to clearly define the VT mechanism and circuit. In case of hemodynamic inestability VT was paced terminated or cardioverted. In this last scenario, the 12-lead ECG QRS morphology of the induced tachycardia was used as a reference during pacemapping. When the initially induced VT was mapped and/or terminated (spontaneously, pace-terminated, shocked or during radiofrequency ablation), PVS was performed again aiming for induction of additional VTs.

Once the areas of interest were identified, a contact force ablation catheter (Smart Touch, Biosense Webster) was used for radiofrequency (RF) delivery. In patients with a hemodynamically stable VT, RF was applied during tachycardia to evaluate the response to RF ablation. In case of VT interruption during RF application, additional lessions were administered to eliminate the substrate and then a new PVS protocol was used to test VT inducibility. In the case of non-inducibility and presence of abnormal signals (late potentials) additional lessions were applied until all the abnormal signals were abolished. Meanwhile, in all patients with hemodynamically unstable VT a substrate ablation strategy ($\pm$pacemapping as an adjunctive strategy whenever possible) was performed targeting all the sites with abnormal signals registered during the initial substrate mapping. Patients with baseline non-inducibility underwent substrate ablation exclusively.

Objectives

The primary objective of the study was to evaluate the influence of baseline VT inducibility and VT activation mapping on patient's outcome in terms of VT recurrence during follow-up.

Secondary objectives included the evaluation of baseline VT inducibility and VT activation mapping influence on the developement of hemodynamic inestability during or after the ablation procedure and on mortality/heart transplantation during follow-up.

Definitions and variables

The clinical tachycardia was defined as the tachycardia previously registered in a 12-lead ECG or, when 12-lead ECG was not available, the tachycardia registered in the ICD log in terms of cycle length and/or intracardiac electrogram morphology. Abnormal electrograms included late potentials registered during the patient's baseline rhythm defined as a high frequency low amplitud signals occurring in the terminal part

of the QRS complex (last 40 ms) or beyond QRS complex termination and diastolic or presystolic potentias registered during VT.

Complete activation mapping was considered when the critical isthmus and or exit of the tachycardia circuit could be clearly defined by entrainment maneuvers. Acute success was defined as non-inducibility of any type of sustained ventricular arrhythmias at the end of the procedure. Survival free of VT recurrence was defined as the absence of sustained VT, symptomatic or asymptomatic, either registered by intracardiac electrograms from an ICD or from a surface 12 lead ECG.

We also recorded the incidence of new-onset of hemodynamic inestability during the procedure or during the first 24-48 h after ablation and the possible relationship with VT induction and the use of activation mapping during the procedure. Hemodynamic inestability during the procedure was defined as persistent hypotension despite vasopressors and requiring mechanical support or procedure discontinuation. During the first 24-48h after the procedure any hemodynamic worsenig, the need of vasoactive drugs or ventricular assist device implantation was also cosidered as procedure-related hemodynamic decompensation.

Follow-up

All patients included had a clinical follow-up after the procedure until hospital discharge. Then, patients were followed in the arrhythmia clinic every 6 months and whenever considered necessary. All patients with a previous ICD were included in a remote monitoring program.

Statistical Analysis

Data are presented as mean ± standard deviation or median and interquartile range (IQR) for continuous variables. Discrete variables are presented as percentage and were compared using Chi-square test while continuous variables were compared using either the t- Student test or analysis of varianze (ANOVA) as appropriate. VT recurrence free survival was calculated using the Kaplan–Meier method with differences between groups compared with the log-rank test. Time-to-event was defined as time from procedure to occurrence of outcome event. Death from any cause within the follow-up period was considered for mortality analysis and was censored at date of death for VT recurrence endpoint. To identify predictors of VT recurrence and death univariate and multivariate Cox proportional hazards models were used. For multivariate analysis, variables traditionally associated with VT recurrences and with a P value [?] 0.15 in the univariate analysis were included in the model. HR and 95% confidence intervals (CI) from the Cox model were reported. Two-tailed P values < 0.05 were considered statistically significant. All analyses were performed by using SPSS (IBM SPSS Statistics, Version 22.0, Armonk, New York, USA).

Results

A total of 203 VT ablation procedures were performed in 163 patients with SHD in our institution from March 2011 to September 2019. In 3 patients PVS was not performed before ablation due to baseline hemodynamic inestability and were not included in the study. The remaining 160 patients were finally included in the analysis and were distributed according to the results of baseline PVS (figure 1): group 1 (not inducible patients) n=18, group 2 (1 VT morphology induced) n=53, and group 3 (>1 VT morphology induced), n=89. In 4 patients the procedure was performed under mechanical hemodynamic support (ECMO in 3 patients and Levithronix in 1 patient). Baseline characteristics of the patients are described in table 1. Table 2 shows the principal findings during the VT ablation procedure.

Effect of baseline VT inducibility on acute success and VT recurrences

PVS was performed at the end of the procedure in 129 patients (80.6%). In the remaining 31 patients PVS was not performed at the discretion of the operator because of perceived risk of hemodynamic inestabilization or previous baseline non-inducibility. Overall acute success rate of the procedure in patients with baseline VT inducibility (groups 2 and 3) and considering non-inducibility of any sustained VT was 66% (76% for group 2 and 60% for group 3, p=0.068). The clinical VT was still inducible in 6.2% of patients (4.5% and

7.7% for groups 2 and 3, respectively) and any non-clinical VT/pleomorphic/polymorphic VT or VF was present in 17.8% of patients at the end of the procedure (4.5% and 26.9% for groups 2 and 3, respectively).

Overall survival free of VT recurrence after a single procedure was 71,1% with a median follow-up of 38.5 months (IQR 16.8-63.2). There were significant differences in survival free of VT recurrence among groups depending on baseline VT inducibility: 94.4% for group 1; 84.9% for group 2 and 58% for group 3, Log-rank p<0.0001 for overall comparison, Log-rank p=0.001 for comparison between group 2 and 3 (figure 2A). Repeat procedures were performed in 31 patients (mean 1.25+-0.58 procedures per patient) with an overall survival free of VT recurrence that increased to 85.6% after the last procedure with a median follow-up of 29.5 months (IQR 38.9-54.4). Group 1 survival free of VT recurrence after the last procedure remained stable at 94.4% but increased for patients in group 2 (94.3%) and group 3 (78.7%).

Effect of activation mapping on acute success and VT recurrences

Complete activation mapping was performed in 50 patients (35.2%, 50/142 inducible patients) and was significantly more frequently among patients with only 1 VT morphology induced compared with patients with >1 VT morphology induced (49.1% vs. 27%, p=0.011). In the remaining 110 patients substrate guide ablation +- pacemapping was used as ablation strategy. Acute success was comparable between patients with complete activation mapping and patients without activation mapping (81.2% vs 70.7%, respectively, p=0.22).

However, survival free of VT recurrence was significantly higher when activation mapping had been performed during the procedure (76% vs 63%, Log-rank p=0.028)(figure 2B). Of note, in patients with only 1 VT induced (group 2) and complete activation mapping (26 group 2 patients) only 4 VT recurrences occurred during follow-up in comparison with 44 VT recurrences in 115 patients with either >1VT induced or impossibility of activation mapping (84.6% survival free of VT recurrence vs. 63.8%, respectively, Log-rank p=0.002)(Supplementary material).

Predictors of VT recurrence during follow-up

Inducibility of >1 VT morphology, the presence of a large endo/epicardial scar (>30%), LVEF < 30% and impossibility of performing activation mapping during VT ablation were associated with VT recurrence in univariate analysis (table 3). A Cox proportional model was used for multivariate analysis and variables with a p value [?]0.15 in univariate analysis were included in the model. Baseline induction of >1 VT morphology was the only independent predictor of VT recurrence in multivariate analysis (HR 12.05 IC 95% 1.60-90.79, p=0.016) (table 3).

Mortality

Overall mortality was 35.6% (50%, 30.2% and 36% for group 1, 2 and 3, respectively) without significant differences between groups (Log-rank p=0.398)(figure 4B). Causes of death were classified as arrhythmic/sudden death in 5 cases (8.8%), advanced heart failure in 27 cases (47.4%), non-cardiac deaths in 17 cases (29.8%) and unknown cause in 8 cases (14%). VT recurrence was not associated with mortality/cardiac transplantation (mortality/transplant 51% for patients with VT recurrence vs. 38.9% for patients without VT recurrence, Log-rank p=0.253). However, patients with baseline 1 VT morphology (group 2) and activation mapping during the procedure had significantly lower mortality during follow-up (19.2% vs 38.8%, Log-rank p=0.039)(figure 4A). In multivariate analysis the only independent predictor of mortality was baseline LVEF<30% (HR 2.43 CI 95% 1.45-4.07, p<0.001)(table 4).

Procedure related complications

Overall complications rate was 11.3% without significant differences among groups (11.1% for group 1, 17% for group 2 and 7.9% for group 3, p=0.251)(table 2). Complications included: 4 cardiac tamponades requiring pericardiocentesis, 3 arterial pseudoaneurysms (2 requiring surgical correction) and 1 femoral hematoma not requiring further interventions. Finally 1 patient died after an acute coronary oclussion (proximal LAD) during the procedure which was inmediately stented but resulting in refractary cardiogenic shock.

Only 9 patients (5.6%) had an acute hemodynamic decompensation during or after the VT ablation procedure. Baseline mean PAINSDD score was 10.7+-6.7 (<10: 48.8%, 10-16: 31.2% and [?]17: 20%). There was no relationship between acute hemodynamic decompensation and activation mapping. In patients with inducible VT (groups 2 and 3), only 1/50 patients (2%) with activation mapping developed acute hemodynamic decompensation in comparison with 6/92 patients with VT inducibility but no activation mapping, p=0.421. The incidence of acute hemodynamic decompensation depending on the PAINSDD score was as follows: 6.4% for PAISDD < 10.2% for PAINSDD 10-16, and 9.4% for PAINSDD [?]17, p=0.337. None of group 1 patients had acute hemodynamic decompensation in comparison with 7.5% of group 2 patients and 5.6% of group 3 patients, p=0.486. Of these 9 patients, 2 died due to refractary cardiogenic shock (one of them after a ventricular assist device was implanted during the ablation procedure) and 1 underwent cardiac transplantation while in the rest 6 patients the hemodynamic decompensation was managed with inotropic drugs and could be stabilized and recovered without further complications.

Discussion

Principal findings

The main finding of our study is that baseline VT inducibility is independently associated with VT recurrences during follow-up in patients with SHD undergoing VT ablation. Thus, patients with no baseline VT inducibility or only 1 VT morphology induced had a significantly higher survival free of VT recurrences when compared with patients with more than 1 VT morphology induced. In the same manner, complete activation mapping during the VT ablation procedure allowing delinitation of the VT circuit was associated with lower VT recurrences. Of note, although no overall relationship between baseline VT inducibility and mortality could be stablished, the combination of inducibility of a single VT morphology and VT activation mapping during the ablation procedure was associated with lower mortality.

Utility of baseline inducibility and activation mapping during VT ablation

VT in the setting of SHD is usually associated with the presence of scar and the principal eletrophysiological mechanism involved in the genesis and manteinance of the tachycardias is re-entry. Traditional VT ablation strategies involved baseline induction of the tachycardia and activation mapping when possible trying to depict the tachycardia circuit in order to guide the ablation strategy. During the last years substrate mapping and ablation without baseline VT induction or activation mapping has proved to be an effective and safe strategy[9-13]. The rationale for this new approach is that complete elimination of the abnormal substrate/signals is associated with better acute success in terms of non-inducibility of any VT (either clinical or not) at the end of the procedure when compared with ablation of the clinical VT only, and has clearly been related with a significant reduction of VT recurrences during follow-up. On the other hand, concerns regarding the developement of hemodynamic decompensation during or after VT ablation procedures have also favored the avoidance of baseline VT induction and activation mapping[15-16]. However, one of the downsides of stand-alone substrate ablation is that it may lead to an extensive ablation that may not be always necessary and could be avoided by a more physiologic approach identifying and ablating only the critical parts of the substrate that are responsible of VT generation and maintenance. Moreover, although re-entry is the principal mechanism of scar-related VT, other less frequent VT mechanisms can be present in patients with SHD and would not be identified unless VT induction is attempted thus hindering the procedure success probabilities[17-18].

Current existing literature in this regard is controversial[11-13]. Two previous meta-analyses have evaluated the influence of VT ablation strategy on outcome. Briceno et al. included 396 patients from 6 different studies (5 observational cohort studies and 1 randomized prospective study) comparing complete substrate ablation vs. standard ablation of stable VTs[12]. Substrate ablation was found to be associated with a decrease in the composite end point of ventricular arrhythmias recurrence/all-cause mortality when compared with the standard approach (RR 0.57, 95% CI 0.40–0.81). Kumar et al. included 403 patients from 6 studies and found no differences in the VT recurrence rate between substrate based VT ablation vs. standard VT ablation using activation mapping/entrainment with a mean follow-up of 18 months (0.72, 95% confidence

interval [CI] 0.44-1.18, p=0.2)[13].

Our study shows that baseline VT inducibility adds significantly important prognostic information in patients with SHD undergoing VT ablation. Following this strategy, patients with baseline inducibility of >1VT morphology are clearly identified at a very high risk of VT recurrences (42% over median follow-up of 38.5 months) while patients with no baseline inducibility or with only 1 VT morphology induced have very low VT recurrences rates (6.6% and 15.1% after the first procedure). It is remarkable that PVS at the end of the procedure did not have this prognostic value in our series. Group 2 patients with acute success at the end of the procedure (determined by non inducibility with PVS) had significantly higher VT recurrence free survival when compared with group 3 patients with acute success (85.4% vs. 63.6%, Log-rank p=0.005)(Supplementary data).

Moreover, activation mapping during VT seems to add additional value and was also associated with lower VT recurrence rates in our study although did not reach statistical significance in multivariate analysis. The utility of VT activation mapping has been recently described[19-20]. Hadjis et. al.[19]showed that VT recurrence was significantly lower among those patients with complete delineation of the diastolic pathway (12%) during VT when compared with partial or no recording of the diastolic pathway (50% and 45%, respectively, p=0.02).

Of interest, hemodynamic decompensation during or after the procedure occurred only in 9 patients (5.6%) in our series. This is a low percentage considering the presence of baseline VT inducibility in up to 89% of the patients and complete VT activation mapping in 35%. Santangeli et al. reported 11% incidence of acute hemodynamic decompensation among 193 patients with scar-related VT undergoing VT ablation[15]. Unfortunately, no information about the incidence of acute hemodynamic decompensation depending on the ablation strategy has been yet published.

Limitations

This is a single center observational study with the inherent limmitations to this study design and using the specific strategy described so the results can not be extrapolated to other VT ablation protocols. Further randomized studies comparing substrate ablation vs. baseline VT induction + activation mapping when possible + critical isthmus substrate ablation should be encouraged to definitively elucidate the role of baseline VT inducibility and activation mapping in this setting. A significant percentage of patients was on active treatment with antiarrhythmic drugs at the time of the procedure and this could have influenced the results of baseline PVS.

Conclusions

Baseline VT inducibility of >1 VT morphology during VT ablation in patients with SHD is independently associated with a significant increase in VT recurrence during follow-up. Baseline VT inducibility and activation maping may add significant prognostic information in patients with SHD undergoing VT ablation.

Founding sources: none

Conflict of interest

Dr. O. Cano has received consultant fees from Biosense Webster and Boston Scientific. All other authors have nothing to disclose.

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Figure Legends.

Figure 1: Flowchart of patients included in the study.

Figure 2: A. Cumulative survival free of VT recurrence stratified by baseline inducibility over 66 months; B. Cumulative survival free of VT recurrence stratified by activation mapping during VT ablation procedure over 66 months.

Figure 3: A. Cumulative survival free of death/cardiac transplantation stratified by baseline inducibility + VT activation mapping over 66 months; B. Cumulative survival free of death/cardiac transplantation stratified by VT recurrence over 66 months.

Figure 4. Principal outcomes per study group (%).

Figure 1.

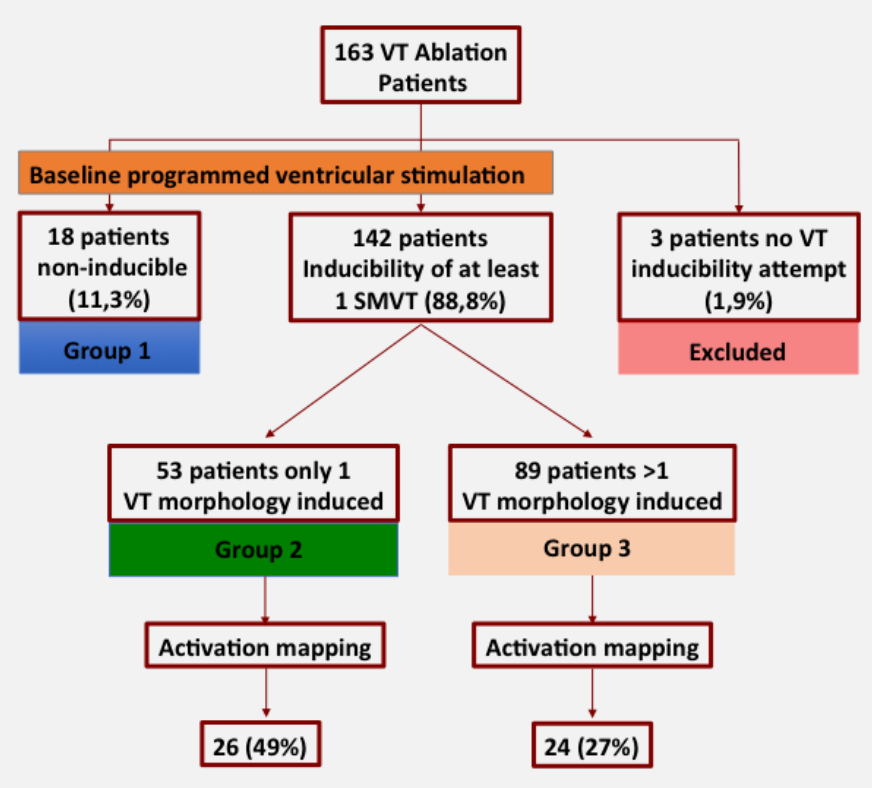

Figure 2.

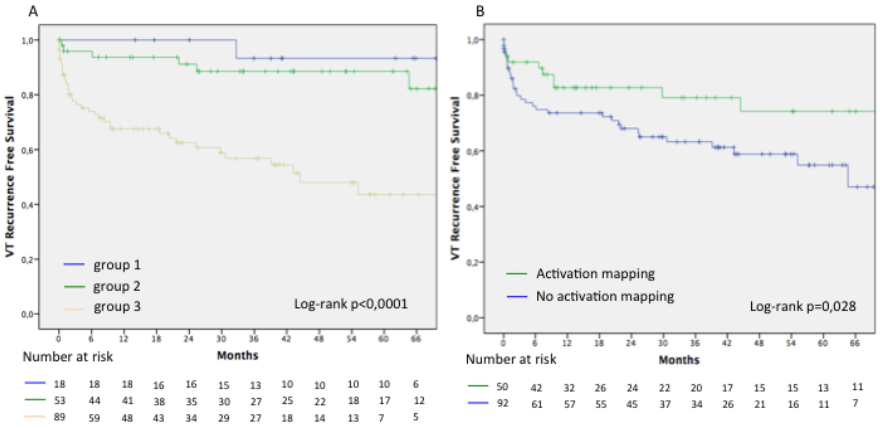

Figure 3.

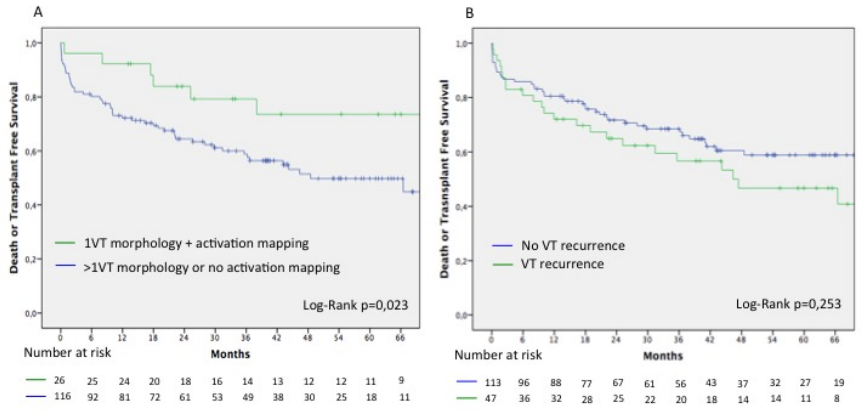

Figure 4.

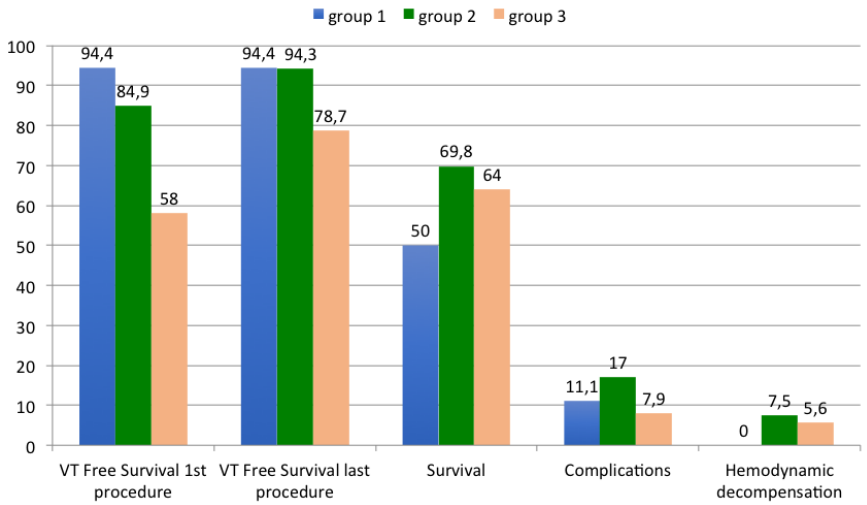

Table 1. Baseline characteristics of the patients

	Overall (n=160)	**Group 1 (n=18)**	**Group 2 (n=53)**	**Group 3 (n=89)**	***P* value**
Age (mean ± SD)	63±13	69±11	61±14	62±13	0.080
Male (n, %)	144 (90)	14 (78)	48 (91)	82 (92)	0.178
Hypertension	97 (61)	13 (72)	32 (60)	52 (58)	0.550
Diabetes	44 (28)	4 (22)	14 (26)	26 (29)	0.813
Hyperlipidemia	87 (54)	10 (56)	27 (51)	50 (56)	0.828
BMI	27.2±4,3	26.9±4,2	26.9±3,8	27.4±4,5	0.779
NYHA class - I - II - III - IV	35 (22) 98 (61) 24 (15) 3 (2)	2 (11) 11 (61) 5 (28) -	14 (26) 32 (60) 4 (8) 3 (6)	19 (21) 55 (62) 15 (17) -	0.063
Chronic renal disease	44 (28)	6 (33)	15 (28)	23 (26)	0.800
Prior myocardial infarction	80 (50)	7 (39)	26 (49)	47 (53)	0.552
Prior revascularization	62 (39)	2 (11)	23 (43)	37 (42)	0.037
Prior stroke	13 (8)	1 (6)	1 (2)	11 (12)	0.080
LVEF	35±15	41±16	40±17	31±12	<0.0001
Betablocker	122 (76)	12 (67)	40 (76)	70 (79)	0.545
ACE-I or ARB	116 (73)	13 (72)	36 (68)	69 (78)	0.733
Sotalol	5 (3)	-	2 (4)	3 (3)	0.715
Calcium cannel blocker	17 (11)	3 (17)	5 (9)	9 (10)	0.672
Amiodarone	65 (41)	6 (33)	23 (43)	36 (40)	0.753
Anticoagulation	70 (44)	8 (44)	19 (36)	43 (48)	0.363
Underlying heart disease - ischaemic - non-ischaemic cardiomyopathy	93 (58) 67 (42)	9 (50) 9 (50)	30 (56) 23 (44)	54 (61) 35 (39)	0.117
Arrhythmic storm (n, %)	39 (24)	4 (22)	11 (21)	24 (27)	0.689
PAINSDD score (mean ± SD)	11±7	11±6	10±7	11±7	0.410

BMI: body mass index; NYHA: New York Heart Association functional class; LVEF: left ventricular ejection fraction; ACE-I: Angiotensin-converting-enzyme inhibitors; ARB: Angiotensin-receptor blockers.

Table 2. Procedural data.

	Overall (n=160)	**Group 1 (n=18)**	**Group 2 (n=53)**	**Group 3 (n=89)**	***P* value**
Number of procedures (n, %) - 1 - 2 - 3 - 4	129 (80) 25 (16) 3 (2) 3 (2)	17 (94) 1 (6) - -	46 (87) 6 (11) - 1 (2)	66 (74) 18 (20) 3 (4) 2 (2)	0.327

	Overall (n=160)	**Group 1 (n=18)**	**Group 2 (n=53)**	**Group 3 (n=89)**	***P* value**
Procedure time (mean ± SD)	196±56	137±39	188±52	212±53	<0.0001
Fluoroscopy time (mean ± SD)	6.7±6.3	6.4±7.8	6.5±5.5	6.8±6.6	0.959
Radiation dose (cGy*cm^2) (mean ± SD)	219±337	237±518	208±305	221±320	0.962
LV access (n, %) - transseptal only - retroaortic only - transseptal + retroaortic	121 (76) 22 (14) 5 (3)	12 (67) 2 (11) 1 (6)	37 (70) 11 (21) -	72 (81) 9 (10) 4 (5)	0.148
RV mapping (n, %)	49 (31)	2 (11)	22 (42)	25 (28)	0.040
Multielectrode mapping catheter (n, %)	107 (67)	9 (50)	33 (62)	65 (73)	0.114
Clinical VT cycle length (mean ± SD)	372±92	318±52	381±103	369±87	0.393
Epicardial access (n, %)	46 (29)	3 (17)	14 (26)	29 (33)	0.402
Endocardial scar (%)(mean ± SD)	15.8±13.1	8.2±5	12.3±11.8	18.8±13.7	0.008
Epicardial scar (%)(mean ± SD)	17.7±25.3	-	24.3±29.4	15.1±23.6	0.335
Radiofrequency time (min)(mean ± SD)	21.9±13.2	13.8±6.4	19.1±10.1	23.7±14.5	0.215
PVS at the end of procedure (n, %)	129 (81)	7 (39)	44 (83)	78 (88)	<0.0001

	Overall (n=160)	**Group 1 (n=18)**	**Group 2 (n=53)**	**Group 3 (n=89)**	***P* value**
Major complications - Vascular - Tamponade - Stroke - Acute hemodynamic decompensation - Coronary oclussion	4 (2.5) 4 (2.5) - 9 (5.6) 1 (0.6)	1 (5.6) 1 (5.6) - - -	2 (3.8) 3 (5.7) - 4 (7.5) -	1 (1.1) - - 5 (5.6) 1 (1.1)	0.329

LV: left ventricle; RV: right ventricle; VT: ventricular tachycardia; PVS: programmed ventricular stimulation

Table 3. Univariate and multivariate analysis for VT recurrence.

Variables	**Univariate Model**	**Univariate Model**	**Multivariate Model**	**Multivariate Mo**
	HR (95% CI)	**P value**	**HR (95% CI)**	**P value**
Inducibility > 1VT morphology	10.75 (1.47-78.53)	0.019	12.05 (1.60-90.79)	0.016
Acute success	0.65 (0.36-1.16)	0.150	0.62 (0.34-1.14)	0.125
Lage Endo/Epi scar (> 30%)	2.28 (1.20-4.33)	0.011	1.71 (0.88-3.30)	0.110
LVEF < 30%	1.99 (1.11-3.57)	0.020	1.43 (0.78-2.62)	0.243
Activation mapping	0.47 (0.23-0.96)	0.039	0.63 (0.31-1.30)	0.216

Table 4. Univariate and multivariate analysis for death or tranplant.

Variables	**Univariate Model**	**Univariate Model**	**Multivariate Model**	**Multivari**
	HR (95% CI)	**P value**	**HR (95% CI)**	**P value**
1VT morphology + activation mapping	2.61 (1.12-6.06)	0.026	2.08 (0.88-4.93)	0.095
LVEF < 30%	2.69 (1.63-4.21)	<0.0001	2.43 (1.45-4.07)	0.001
VT recurrence	0.74 (0.45-1.23)	0.255	1.13(0.68-1.87)	0.623
Lage Endo/Epi scar (> 30%)	1.20 (0.66-2.17)	0.544	0.95 (0.52-1.74)	0.889